EMERGENCY MEDICINE SECRETS

SIXTH EDITION

QUESTIONS YOU WILL BE ASKED
TOP 100 SECRETS ■ KEY POINTS ■ WEBSITES

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EMERGENCY MEDICINE SECRETS, SIXTH EDITION ISBN: 978-0-323-35516-2

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Library of Congress Cataloging-in-Publication Data

Names: Markovchick, Vincent J., editor. | Pons, Peter T., editor. | Bakes, Katherine M., editor. | Buchanan, Jennie A., editor.
Title: Emergency medicine secrets / [edited by] Vincent J. Markovchick, Peter T. Pons, Katherine M. Bakes, Jennie A. Buchanan.
Other titles: Secrets series.
Description: Sixth edition. | Philadelphia, PA: Elsevier, [2016] | Series: Secrets | Includes bibliographical references and index.
Identifiers: LCCN 2015038975 | ISBN 9780323355162 (paperback)
Subjects: | MESH: Emergencies–Examination Questions. | Emergency Medicine–Examination Questions.
Classification: LCC RC86.9 | NLM WB 18.2 | DDC 616.02/5076–dc23
LC record available at http://lccn.loc.gov/2015038975

Executive Content Strategist: Jim Merritt
Content Development Specialist: Lisa Barnes
Publishing Services Manager: Catherine Albright-Jackson
Senior Project Manager: Doug Turner
Book Designer: Ryan Cook

Printed in Canada

Last digit is the print number: 9 8 7 6 5 4 3 2 1

To my wife, Leslie, and daughters, Nicole, Tasha, and Nadia, the four greatest ladies in my world. I thank them for their lifelong support of all my endeavors, including the editing of EM Secrets. *I also thank my coeditors, Peter Pons, Katherine Bakes, and Jennie Buchanan, and all of the chapter authors for their contributions to this book. And I thank the publisher for the privilege of editing this book through six editions. Finally, I want to acknowledge all of the wonderful residents, nurses, medical students, and all members of the EM family with whom I've had the great honor and privilege to work over the past many years. It is their knowledge, enthusiasm, and intellectual curiosity that 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

For my mentors, Vince and Peter; I am eternally grateful. And for my parents, Ursula and Phil, my constant support; I love you forever.

KMB

To Myron H. Buchanan (22 October 1935–9 April 2015), the best Dad a girl could have ever asked for. I love and miss you every day, Mr. B. Thank you for making this path possible; I could not have done it without you.

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PREFACE

This book is for all students and practitioners of emergency medicine, both novice and experienced. Because emergency medicine continues to mature as a specialty and our daily practice of it continues to evolve, we have reorganized some of the chapters and added appropriate content to reflect these changes. With difficulty we have also selected the Top 100 Secrets from more than 300 submitted by the chapter authors and editors. We hope that this book continues to be a concise, valuable, and enjoyable method of imparting information and knowledge. Knowing some of the most important questions about a particular presentation or problem is the first step to obtaining the answers needed at the patient's bedside. However, medicine being both an art and a science is nothing if not humbling, and knowledge alone does not treat all that ails. Listen to your patients and make them feel heard. Treat them all with the care and empathy you would wish for any member of your family. Getting to the correct diagnosis can be and is invigorating, but positively impacting a life confirms our calling.

ACKNOWLEDGMENT

The editors would like to acknowledge and express our heartfelt thanks to Ms. Carol Lucas for her role in the preparation of this and previous editions of *Emergency Medicine Secrets.* Her meticulous attention to detail and penchant for hard work while always maintaining an upbeat attitude and good humor have been invaluable to the timely completion of this manuscript. We were very fortunate to have had her assistance in this endeavor.

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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. Always consider the most serious possible cause of the patient's presenting signs and symptoms.
2. Treat ventricular fibrillation (VF) with immediate defibrillation if the arrest is witnessed; treat with cardiopulmonary resuscitation (CPR) and then defibrillation if the arrest is unwitnessed.
3. If the arrest is result of pulseless electrical activity (PEA), remember its common reversible causes (i.e., hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, hypothermia, massive pulmonary embolism, drug toxicity, electrolyte disturbances, acidosis, and myocardial infarction) and treat them appropriately.
4. Preoxygenation with 100% fraction of inspired oxygen (FiO_2) for 5 minutes or 8 vital capacity breaths, as well as passive apneic oxygenation, are critical steps to prevent oxygen desaturation during rapid-sequence intubation (RSI).
5. Never paralyze a patient unless you are certain that his or her airway can be ventilated with a bag-valve mask or an extraglottic rescue device.
6. Serum lactate is a commonly used marker to assess the extent of systemic hypoperfusion and the response to resuscitation.
7. The primary resuscitation goals in patients suffering from shock are to maximize oxygenation, establish adequate ventilation, improve hemodynamic distribution, and treat the underlying cause.
8. Atypical presentations of serious disease are more common in elderly patients.
9. Aggressive symptomatic treatment, such as pain control and comfort care, is always appropriate in the hospice patient the ED.
10. Patients who receive multiple high-dose imaging examinations (especially computed tomography [CT]) are at highest risk of long-term radiation consequences.
11. When evaluating results of a research paper, the smaller the number needed to treat, the more effective the intervention or treatment.
12. Of the readily available methods, rectal temperatures are the most accurate representation of core body temperature.
13. A normal electrocardiogram (ECG) on initial presentation does not exclude acute coronary syndrome (ACS).
14. Twenty-five percent of patients ultimately diagnosed with ACS do not have a primary complaint of chest pain.
15. Consider mesenteric ischemia when abdominal pain is out of proportion to physical findings.
16. In a patient who has nausea and vomiting, consider causes other than gastrointestinal (GI) disorders.
17. In the treatment of anaphylaxis, hypotension is the indication for intravenous (IV) epinephrine.
18. Administer IV epinephrine as a drip, not as a bolus, in the non–cardiac arrest situation.

19. When an anterior packing fails to control epistaxis, a posterior bleed originating from sphenopalatine artery should be suspected, and a posterior pack should be placed.
20. Every ED should have an interdisciplinary evidence-based guideline for the management of acute stroke.
21. Pulse oximetry measures oxygenation, not ventilation.
22. Tidal volume and respiratory rate affect the patient's ventilation and partial pressure of carbon dioxide ($PaCO_2$).
23. Oxygenation and ventilation problems in patients using mechanical ventilators can be managed by removing them from the ventilator and following the *DOPE* (**d**isplacement, **o**bstruction, **p**neumothorax, **e**quipment) mnemonic.
24. Continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) will reduces the need for need for endotracheal intubation in both the ED and hospital areas.
25. A D-dimer assay is only useful to exclude thromboembolic disease in patients with a low pretest probability.
26. Determination of pretest probability for venous thromboembolism (VTE) is critical in knowing when to initiate a diagnostic workup and how to interpret test results.
27. Cardinal symptoms of congestive heart failure (CHF) include fatigue, exertional dyspnea, paroxysmal exertional dyspnea, and orthopnea.
28. Nitrates are first-line pharmacotherapy for the treatment of CHF, and diuretics should be reserved for those patients with fluid overload.
29. An unstable patient with any tachydysrhythmia, regardless of the mechanism, requires electrocardioversion.
30. When trying to decide whether the rhythm your patient is in is ventricular tachycardia (VT) or supraventricular tachycardia (SVT) with aberrancy, assume VT and treat accordingly.
31. Calcium channel blockers should not be used to treat wide-complex tachycardias.
32. Consider ruptured abdominal aortic aneurysm in any patient with abdominal pain, pulsatile mass, and hypotension.
33. Consider HIV/AIDS in patients at risk and who have an illness or infection, particularly those with opportunistic infections or extreme presentations of common diseases.
34. 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.
35. Epigastric pain may be caused by myocardial ischemia, so an ECG should be obtained in adult patients with epigastric discomfort, visceral-type pain, or cardiac risk factors.
36. Consider mesenteric ischemia in any patient with atrial fibrillation and abdominal pain.
37. An external pacemaker can be used if a permanent pacemaker malfunctions.
38. Indications for emergency dialysis include acute pulmonary edema, life-threatening hyperkalemia, and life-threatening intoxication or overdose with agents normally excreted by the kidneys.
39. The ECG changes seen as potassium rises are a tall, peaked T wave; loss of the P wave; and widening of the QRS complex.
40. In the emergent treatment of hyperkalemia, glucose and insulin supplemented by an inhaled β agonist is the most effective method to drive potassium into the cell and

acutely lower serum potassium. In addition, IV sodium bicarbonate and calcium chloride are indicated in patients with metabolic acidosis and a wide-complex cardiac rhythm.

41. Seizures, coma, and acute neurologic findings in a previously normal patient are the only indications to give hypertonic saline in patients with profound hyponatremia.
42. Anion gap metabolic acidoses are common and life threatening; the four major causes are lactic acid, ketones, renal failure, and toxic ingestions.
43. 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.
44. It is unnecessary to gradually empty the bladder when treating an episode of acute urinary retention.
45. Ketamine provides sedation, analgesia, and amnesia while protecting the cardiovascular status and airway reflexes, making it an ideal agent for procedural sedation in children.
46. In patients with severe sepsis, initiate early goal-directed therapy in order to reduce the mortality rate by 25%.
47. Simple abscesses are treated with incision and drainage and do not require antibiotics.
48. Necrotizing infections are a surgical emergency that require emergent operative debridement, IV fluid resuscitation, and broad-spectrum IV antibiotics.
49. Adult botulism presents as nonspecific anticholinergic symptoms followed by symmetric cranial nerve palsies and descending paralysis.
50. Antibiotics are not recommended in children with bloody diarrhea with concern for toxin release and development of hemolytic uremic syndrome (HUS).
51. Traditional triage rules do not apply to lightning victims; remember reverse triage, and concentrate on victims who appear to be in cardiopulmonary arrest.
52. Ultrasound should be the first imaging study in children with suspected appendicitis. CT should only be used if ultrasound is equivocal and there is high clinical suspicion for appendicitis.
53. Bilious emesis in a neonate is a surgical emergency until proven otherwise, requiring an upper GI series or surgical consultation.
54. The highest impact intervention for a newborn or pediatric resuscitation is appropriate respiratory support.
55. A suicide attempt should be considered in patients with illogical explanations for serious accidents.
56. Consider testicular torsion in any male with lower abdominal pain.
57. The antidote for acetaminophen overdose is *N*-acetyl cysteine. It is most effective when administered within 10 hours, regardless of whether it is given orally or intravenously.
58. Obtain a carbon monoxide (CO) level, and treat any patient who has inhaled smoke in an enclosed space with nonrebreather, high-flow, mask oxygen.
59. The classic triad of opioid poisoning is central nervous system (CNS) depression, respiratory depression, and miosis.
60. Because specific amounts of drugs ingested are often difficult to determine in small children, prolonged observation is often required to rule out a potentially toxic ingestion.

61. Neither pregnancy nor tubal ligation excludes a diagnosis of pelvic inflammatory disease.
62. Victims of sexual assault 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.
63. A pregnant woman with hypertension and seizures should be treated with IV magnesium sulfate and consideration of emergent delivery of the fetus.
64. With few exceptions, procedures performed in the ED can be done with fewer complications and greater success using ultrasound guidance.
65. All trauma patients should be completely undressed and examined, front and back.
66. Use clinical decision rules such as the National Emergency X-Radiography Utilization Study (NEXUS) or the Canadian C-spine rule to avoid overuse of cervical spine radiographs.
67. The use of steroids in blunt spinal cord trauma is not standard of care.
68. In patients with traumatic brain injury, maintain cerebral perfusion and oxygenation, and avoid hypotension.
69. Preservation of vision in a chemical burn is directly related to the length of time from the exposure to the initiation of irrigation; do not wait for the patient to arrive at the ED to begin eye irrigation.
70. Diplopia on upward gaze is the hallmark of a blow-out fracture of the orbital floor.
71. In patients with blunt or penetrating neck trauma, perform endotracheal intubation early, before airway distortion occurs.
72. In a patient with chest trauma, immediate threats to life that must be identified and treated in the primary survey include airway obstruction, tension pneumothorax, open pneumothorax, flail chest, massive hemothorax, and cardiac tamponade.
73. A contaminated wound is one with a high degree of bacterial inoculum at the time of injury, and is not synonymous with a dirty wound.
74. A single negative focused assessment with sonography for trauma (FAST) does not reliably exclude significant intraperitoneal injury.
75. In patients with pelvic fractures, mechanical stabilization of the pelvis, reversal of shock, and correction of coagulopathy are critical early steps to avoid exsanguination.
76. Children manifest shock later than adults with the same percentage of blood loss, yet decompensate more quickly once this critical volume is lost.
77. Because of the laxity of children's ligamentous structures, cervical fractures, rib fractures, and aortic injuries are less common, but cervical ligamentous injuries and pulmonary contusions are more common.
78. Scaphoid fractures can escape radiographic detection in the acute setting. Patients with tenderness to palpation of the anatomic snuffbox should be treated with a thumb spica splint and repeat evaluation in 1 to 2 weeks under the assumption that they may have a fracture.
79. Any laceration over the dorsal metacarpophalangeal (MCP) joint is suspicious for a fight bite. Fight bites require meticulous exploration and wound care in the operating

room. If the wound penetrates the extensor hood, thorough joint washout and IV antibiotics are required.

80. Consider nonaccidental trauma (child abuse) in children when the injury pattern is incompatible with the developmental age, such as long bone fractures in a nonambulatory child.
81. Use a tourniquet on extremities and digits to control hemorrhage; visually examine and properly repair the wound.
82. Disasters and multiple casualty incidents (MCIs) occur whenever the needs of an incident exceed the resources available to respond to it.
83. Communication is the most important component to ensure an efficient and effective disaster response.
84. The "ideal" terrorist weapon is cheap, easy to manufacture, easy to disseminate, and will produce large numbers of casualties (e.g., an improvised or conventional explosive device).
85. A chemical agent attack will generally be recognized by having a large number of casualties with similar symptoms over a short time in a relatively small geographic area.
86. Percutaneous coronary intervention (PCI) is preferable to thrombolytics for emergency management of ST-segment elevation myocardial infarction (STEMI) unless there is an anticipated delay of more than 90 to 120 minutes to balloon inflation.
87. Documenting adherence to evidence-based guidelines is helpful in defending against a malpractice claim.
88. 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.
89. Patients who have persistent hemodynamic instability or peritonitis after abdominal trauma require emergent laparotomy.
90. Never restrain a patient in the prone position; restrain the patient on his or her side to minimize risks of aspiration and sudden death.
91. In the event of a biologic agent attack, all symptomatic patients should be considered to be infectious until a definitive diagnosis of a nontransmissible agent is confirmed.
92. Consider domestic violence in women with depression, suicidal ideations, chronic pain, psychosomatic complaints, or multiple ED visits.
93. Ingestion of a disc or button battery mandates an emergent GI or surgery consultation for endoscopic or surgical removal.
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. Consider toxic shock syndrome in any patient with a rapidly progressive shock syndrome and diffuse erythematous rash, and ensure there is no removable infected source of endotoxin production.
96. A CT scan for appendicitis is negative only if the entire appendix has been visualized and is normal.
97. Consider cyanide poisoning when the patient has inhaled smoke from burning furniture fabric (e.g., wool, silk, or polyurethanes).

98. Obtain a CT scan of the head on any patient taking warfarin (Coumadin) with even a minor head trauma.
99. Emergency medical services (EMS) has proven benefit for treatment of cardiac arrest, respiratory distress, and traumatic injury.
100. Spinal epidural abscess should be suspected as the cause of back pain in immunocompromised patients and IV drug users who have localized spinal tenderness and fever.

DECISION MAKING IN EMERGENCY MEDICINE

Vincent J. Markovchick, MD, FAAEM

CHAPTER 1

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. Emergency medicine physicians must know something about all aspects of medical care, with an emphasis on identifying and treating acute life threats.

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 and limited resources also impede the use of conventional methodology in the ED.

4. **How do I identify the patient with a life-threatening condition?**
   Three components are necessary to quickly identify the patient with a life-threatening condition:
   1. A chief complaint and a brief, focused history relevant to the chief complaint
   2. A complete and accurate set of vital signs in the field and in the ED that are accurately taken and critically interpreted
   3. An opportunity to perform a rapid, focused physical examination that includes visualization, auscultation, palpation, and observation

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 patients with life-threatening conditions. 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 may 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

Abstract

The initial approach to a patient in the ED is different than taught for other patients in the office or hospital setting. This chapter discusses this approach with an emphasis on quickly identifying those patients who have life-threatening or serious conditions that need to be addressed emergently. It also discusses the unique approach to formulating a differential diagnosis for an ED patient.

Keywords:

differential diagnosis, decision making, EM decision making, vital sign interpretation

membrane thermometer was used or if the patient was hyperventilating or mouth breathing when the oral temperature was taken. When either fever or hypothermia is suspected, measure a rectal temperature.

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 is found in the field with acute onset of left lower quadrant abdominal pain. She is cool, clammy and diaphoretic, with a pulse of 116 beats per minute and blood pressure of 78 palpable. She receives 1500 mL of intravenous (IV) fluid en route to the ED. She 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 when the vital signs, although in the normal range, are inconsistent with the patient's chief complaint and overall clinical appearance. For example, a 20-year old man with severe asthma who presents with hours of dyspnea and poor air movement may have a "normal" respiratory rate of 14 breaths per minute. For this patient one would expect a respiratory rate of 20 to 30 breaths per minute, and thus a respiratory rate of 14 is abnormal, indicating fatigue and impending 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?

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 to this patient in the ED?

The differential diagnosis formulated in the ED must begin with the most serious condition possible to explain the patient's presenting symptoms and be continued from there. An example is a 60-year-old man who exhibits nausea, vomiting, and epigastric pain. Instead of assuming the condition is caused by a gastrointestinal disorder, an acute myocardial infarction (MI) must first be considered and appropriate steps must be taken to stabilize the patient (i.e., start an IV line, initiate oxygen [O_2], and place a cardiac monitor). Then rule out an MI by completing an adequate history and physical examination, an electrocardiogram (ECG), and appropriate laboratory studies.

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 condition. This approach may overlook the most serious, albeit sometimes a very uncommon, problem. Therefore the practice of emergency medicine involves some degree of healthy paranoia to consider the most serious conditions compatible with the patient's presenting symptoms. Through a logical process of elimination, first rule in or out the life threats before gravitating to the more likely diagnoses.

15. Is a diagnosis always possible or necessary in the ED?

No. Patients should be informed of goals in the ED. Sometimes, the most important thing is to know that they don't have a life-threatening condition. It can often take days, weeks, or months for a final diagnosis to be made. It is unreasonable to expect that every patient 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, anxiety-provoking work environment for you.

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

Admit to the patient and document in the medical record the inability to make a diagnosis. It is the role of the ED physician to rule out and stabilize serious or life-threatening conditions, not to always arrive at a definitive diagnosis. For example, a patient who comes to the ED with acute abdominal pain; who has had an appropriate history, physical examination, and diagnostic studies; 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 of labeling the patient with a benign diagnosis such as gastroenteritis or gastritis that is not supported by the medical record. More important, 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 returning 1 or 2 days later with something more serious, such as a ruptured appendix.

17. What is the most important question to ask a patient who comes 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 a 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 occasion 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?

The medical condition is the first obvious factor to consider. Beyond this, ask yourself the following questions: Is there a medical need that can be fulfilled only by hospitalization, or can the patient be safely observed in the outpatient setting? For example, does the patient need oxygen therapy or cardiac monitoring? Can the patient who has sustained head trauma adhere to head trauma precautions at home, or does he or she require in-patient care because of homelessness or living alone? 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 needed follow-up care. Failure to do so constitutes patient abandonment. Specific verbal and written 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 complications 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?

1. Why did the patient come to the ED?
2. Have I made the patient feel better?

Generally, most patients come 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 that 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 a heart attack. When indicated, 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 the patient's signs and symptoms.
3. Always inquire about a patient's social situation before ED discharge.
4. Remember to focus on alleviating the patient's somatic or psychological pain.

QUESTIONS

1. When formulating a differential diagnosis on an ED patient, one should ask what is:
   a. The most likely diagnosis
   b. The most serious diagnosis
   c. The most uncommon diagnosis
   d. The most benign diagnosis
   The correct answer is *b.*
2. What is the top priority in the initial encounter with a patient in the ED?
   a. Introduce yourself.
   b. Order appropriate diagnostic studies.
   c. Identify and stabilize the life threat.
   d. Obtain a past medical history.
   The correct answer is *c.*
3. What is the most often inaccurate vital sign obtained by EMTs in the prehospital setting?
   a. Blood pressure
   b. Pulse
   c. Respiratory rate
   d. Temperature
   The correct answer is *c.*

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. What is CAB, and why is it recommended?

In 2010 the American Heart Association recommended changing the basic life-support sequence from ABC to chest compressions, airway, and breathing (CAB) for everyone except neonates. This change was recommended to prioritize chest compressions, which are often delayed while a rescuer is trying to open the airway and provide rescue breaths.

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

1. 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.
2. Obtain a defibrillator.
3. Initiate CPR immediately with emphasis on high-quality chest compressions (i.e., compressions should be at least 100 per minute, have adequate depth [at least 2 inches in adults], and allow for complete chest recoil between compressions).
4. Lone rescuers should perform 30 chest compressions before opening the airway and delivering two breaths. When two or more rescuers are present, CPR should similarly begin with chest compressions, but the delay in providing oxygenation and ventilation will be less. Interruptions in chest compressions should be minimized.
5. When managing the airway, 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.
6. Assist breathing by performing mouth-to-mouth, mouth-to-mask, or bag-valve-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 face mask 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.
7. Check the patient's rhythm every 2 minutes if the patient is pulseless, and perform defibrillation if necessary.

4. 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.

5. What is passive oxygen insufflation?

Passive oxygen insufflation is accomplished by placing an oropharyngeal airway, a nonrebreather face mask with high-flow oxygen, and a nasal cannula with high-flow oxygen on the patient. Preliminary

Abstract

Cardiac arrest is a leading cause of mortality in the United States. Factors associated with survival, usually defined as survival to hospital discharge, include activating emergency medical services (EMS), early provision of cardiopulmonary resuscitation (CPR), and early electrical defibrillation if cardiac arrest is the result of pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF). Patients with an initial cardiac rhythm of asystole or pulseless electrical activity (PEA) have uniformly worse prognoses than those with VF or VT. Understanding and mastering principles of resuscitation, including the ABCs, are critical when providing care to patients who experience cardiac arrest.

Keywords:

cardiac arrest, resuscitation, treatment, management

data suggest this approach may be superior when compared with a traditional active ventilatory approach using a bag-valve in conjunction with other cardiocerebral resuscitation strategies.

6. What is capnography, and how should it be used during resuscitation?

Capnography is the monitoring of the partial pressure of carbon dioxide in exhaled gases. Continuous quantitative waveform capnography is recommended for patients experiencing cardiac arrest who are intubated throughout resuscitation. It is used to confirm endotracheal tube placement, for monitoring the quality of CPR, and detection of return of spontaneous circulation (ROSC).

7. 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. This approaches prevents hyperventilation, which may be deleterious in the cardiac-arrest setting.

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

- Monitored cardiac arrest: When a patient in a monitored setting experiences sudden pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF), immediate electrical defibrillation supersedes performance of CPR.
- 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.

9. 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 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.

10. 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).

11. What is coronary perfusion pressure (CPP)?

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

12. What is the association between CPR, CPP, and ROSC?

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

13. 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.

14. Discuss the role of pharmacologic therapy during CPR.

The immediate goal of pharmacologic therapy is to improve CPPs and thus myocardial blood flow, which is associated 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. Atropine is no longer recommended for routine use in the management of cardiac arrest.

KEY POINTS: STANDARD ADULT DOSAGES OF CARDIAC ARREST MEDICATIONS

1. Epinephrine: 1 mg intravenous (IV)/intraosseous (IO) push (every 3 to 5 minutes)
2. Vasopressin: 40 U IV/IO push (can replace first or second dose of epinephrine)
3. Amiodarone: 300 mg IV/IO push; 150 mg IV/IO push as second dose, if needed

15. 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 before defibrillation, and several communities have adopted this in the prehospital setting where most arrests are unwitnessed.

16. What are the indications for open-chest cardiac massage?

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

17. 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 caused by 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, and myocardial infarction.

18. 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 hydroxycobalamin, which combines with cyanide to form cyanocobalamin (vitamin B_{12}). Sodium nitrite and sodium thiosulfate are considered second-line therapy for cyanide toxicity.

- Tricyclic antidepressants act as type Ia antidysrhythmic agents and cause cardiac conduction slowing, ventricular dysrhythmias, hypotension, and seizures. Vigorous serum alkalinization with sodium bicarbonate 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.

19. How should VF be treated?

Rapid identification and treatment of VF 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 to 200 J biphasic). The antidysrhythmic agent of choice is amiodarone, which enhances the rate of successful defibrillation and reduces the likelihood of recurrent VF after successful conversion. Administration of epinephrine or vasopressin before defibrillation may improve defibrillation success, although defibrillation should not be delayed while waiting for medication; in addition, CPR before defibrillation (see Question 13) may also improve defibrillation success in the setting of prolonged VF.

20. 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 the speed at 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 and 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.

21. Should you administer one shock at a time or a sequence of shocks (also referred to as *stacked shocking*)?

No study has shown survival benefit with stacked shocks. If one shock fails to eliminate VF, the incremental benefit of another shock is low, and resumption of CPR is likely to confer greater value than another immediate shock.

22. What is the optimal placement of electrode pads used for defibrillation?

For ease of placement, the anterolateral pad position is recommended. However, there is no efficacy difference between this position and the three others (anteroposterior, anterior–left infrascapular, or anterior–right infrascapular).

23. What if VF persists after initial treatment?

a. Continue CPR.
b. Perform endotracheal intubation and ensure adequate oxygenation and ventilation.
c. Administer epinephrine (1 mg IV/IO push) or vasopressin (40 U IV/IO push) to augment aortic diastolic blood pressure and to improve myocardial perfusion.
d. Administer amiodarone (300 mg IV/IO push). Amiodarone may be repeated (at 150 mg IV/IO push) after 3 to 5 minutes.
e. Consider administering magnesium sulfate (1 to 2 g IV push).

24. 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.

25. 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/IO push) or vasopressin (40 U IV/IO push).

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; treat with CPR and then defibrillation if the arrest is unwitnessed.
2. If the arrest is caused by 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 the result of asystole, remember to exclude fine VF.

26. 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 and is not recommended.

27. 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 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.

28. 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 interferes 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 in terms of rapid access to the patient's central circulation.

29. When may prehospital resuscitation efforts be terminated?

According to the most recent American Heart Association 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 basic life support (BLS) and ACLS, including successful endotracheal intubation, achievement of IV access and administration of appropriate medications, and determination of a persistent asystolic or agonal rhythm, as well as when no reversible cause for the arrest is identified.

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

This remains controversial. Epinephrine has been evaluated in human trials in approximately 9000 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 to hospital discharge or survival with good neurologic outcomes. It remains unknown whether a high-dose epinephrine approach in conjunction with high-quality postresuscitation care, including use of targeted temperature management, will increase survival with good neurologic function. Vasopressin acts directly on V_1-receptors and, unlike epinephrine, is more effective in an acidemic environment. Vasopressin has been compared with epinephrine in at least three human trials totaling approximately 1500 patients and demonstrated no significant difference in survival. A recent metaanalysis of 10 randomized controlled trials including 6120 patients showed no benefit of vasopressin over epinephrine alone in unselected cardiac arrests.

31. 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.

32. 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 carbon dioxide 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.

33. 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.

34. What should I do after ROSC?

Once ROSC is achieved, the vulnerable and highly tenuous postresuscitation period begins. This period is marked by a profound systemic inflammatory response syndrome resulting from whole-body ischemia and reperfusion. Patients commonly experience hemodynamic instability, 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, although its efficacy remains unknown. In addition, early aggressive percutaneous coronary intervention and targeted temperature management should be performed to improve survival and neurologic recovery, respectively.

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

5% to 7%.

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QUESTIONS

1. Cardiac arrest resulting from VF is:
   a. Best treated with immediate CPR followed by intravenous epinephrine
   b. Best treated with immediate defibrillation when the arrest is witnessed
   c. Associated with the highest mortality rate
   d. Best treated with magnesium sulfate
   The correct answer is *b.*
2. ROSC after cardiac arrest is:
   a. A rare event
   b. Most common when PEA is the initial rhythm
   c. Directly related to degree of CPP during CPR
   d. Inversely associated with the cumulative dose of epinephrine administered
   The correct answer is *c.*
3. Asystole:
   a. Is associated with the lowest mortality rate
   b. May actually be fine VF
   c. Is treated with amiodarone and atropine
   d. Results from too aggressive CPR
   The correct answer is *b.*

AIRWAY MANAGEMENT

W. Gannon Sungar, DO, and Richard D. Zane, MD

CHAPTER 3

1. Which ED patients need airway assessment?

Emergency physicians are masters of the airway, and every patient in the ED should have their airway assessed.

2. What are the different mechanisms of respiratory failure?

Respiration consists of oxygenation and ventilation, and patients can experience respiratory failure by four main mechanisms:

1. Loss of airway protective reflexes: Often result of nonrespiratory cause (e.g., trauma, toxicologic) causing collapse of the airway anatomy and loss of airway patency
2. Hypoxemic: Failure of oxygenation manifested by cyanosis and/or low readings on pulse oximetry
3. Hypercapnic: Failure of ventilation that leads to elevated partial pressure of carbon dioxide (pCO_2) levels, resulting in acidosis and altered mental status
4. Mixed: Failure of both oxygenation and ventilation

3. How do I assess a patient's respiratory status?

If a patient is able to speak, he or she has an intact airway. Lacking this, signs of airway collapse include sonorous respirations, pooling of secretions, and inability to swallow. Assessment of oxygenation and ventilation is achieved by looking at the patient's skin color, work of breathing, respiratory rate, and mental status.

4. Does a lack of a gag reflex mean my patient can't protect his or her airway?

No, the lack of a gag reflex is an unreliable marker for airway collapse, because up to 25% of the population lacks a gag reflex at baseline. Also, the presence of a gag reflex does not imply ability to protect the airway.

5. What is a definitive airway?

A *definitive airway* is defined as a cuffed endotracheal (ET) tube through the vocal cords, with the cuff below the level of the vocal cords.

6. What is the most common cause of airway obstruction?

The tongue is the most common cause of airway obstruction, because it blocks 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.

7. How can I initially assist a patient in respiratory failure?

Patients with airway collapse may benefit from an airway maneuver or airway stenting, including:

- Head tilt–chin lift: Lift the chin cephalad and anterior, creating slight extension of the head.
- Jaw thrust: Lift anteriorly at the bilateral mandibular angles, moving the tongue off of the posterior oropharynx.
- Artificial airway: A nasopharyngeal (NP) or oropharyngeal (OP) airway can be placed in the naris or mouth, respectively, to stent the tongue off of the posterior oropharynx and maintain upper airway patency.
- Bag-valve mask (BVM): After using one or more of the airway maneuvers above to establish airway patency, a bag-valve mask can be used to support oxygenation and ventilation.

8. How do I predict patients who will be difficult to assist with a BVM?

Risk factors for difficult mask ventilation address challenges with achieving a good mask seal; for example, the presence of a beard, obstruction or obesity, edentulousness, age older than 55 years, facial trauma, and stiff lungs (chronic obstructive pulmonary disease [COPD], asthma, chest trauma).

Abstract

Airway management skills are critical to the practice of emergency medicine. This chapter discusses in detail the indications for and techniques of airway management.

Keywords:

airway management, intubation, rapid-sequence intubation (RSI), laryngeal-mask airway (LMA), King airway, rescue airway devices

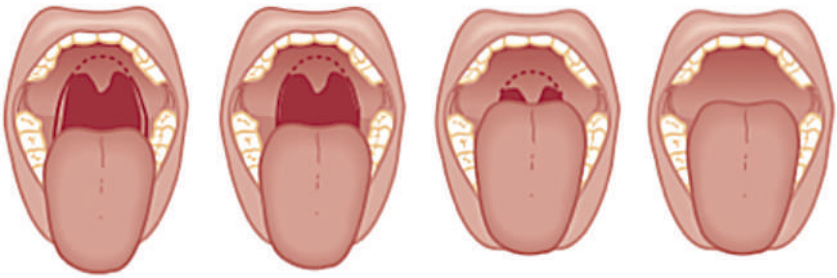

Figure 3-1. Mallampati score. *(From Brown CA, Walls RM: Airway. In Marx JA, Hockberger RS, editors:* Emergency medicine concepts and clinical practice*, ed 8, Philadelphia, 2014, Saunders, p 5.)*

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

RSI is a method of facilitating ET intubation by inducing short-term paralysis after induction of unconsciousness. Because all emergency patients are at risk for aspiration, the airway must be secured as quickly as possible, ideally after a period of preoxygenation followed by induction of unconsciousness, paralysis, and then intubation. It is best if there is no positive pressure ventilation before tube placement.

10. How do I assess for a difficult intubation?

The LEMON mnemonic is a helpful reminder of factors associated with a difficult intubation:

- **L:** Look externally to assess for facial trauma, blood in the airway, loose or false teeth, cervical collar, and so on.
- **E:** Evaluate using the 3-3-2 rule to predict difficult airway anatomy.
  - A patient who cannot open his or her mouth to fit three fingers between their central incisors may have limited mouth opening necessary for direct laryngoscopy.
  - A hyomental distance (hyoid to tip of chin) fewer than three finger breadths predicts a more difficult anterior larynx.
  - Fewer than 2 finger breadths from hyoid to the thyroid cartilage predicts a short neck and a cephalad larynx.
- **M:** The Mallampati score (Fig. 3-1) is a measure of baseline airway patency and is a good predictor of ease of laryngoscopy.
  - Class I: Complete visualization of the uvula and the tonsillar pillars
  - Class II: Visualization of the entire uvula
  - Class III: Visualization of only the base of the uvula
  - Class IV: Limited to visualization of the hard palate only
- **O:** Obstruction. Evaluate for visualized foreign bodies and stridor.
- **N:** Neck mobility: Decreased neck mobility due to kyphosis, c-collar, or other condition may limit manipulation techniques that can aid in direct laryngoscopy.

11. What basic equipment is necessary for ET intubation?

- Laryngoscope, direct and video (see Question 12). The following are two common direct laryngoscope blades:
  - The Macintosh (curved) blade is placed anterior to the epiglottis into the vallecula and acts to lever the epiglottis off of the cords using the median glossoepiglottic fold.
  - The Miller (straight) blade is used to lift the underside of the epiglottis anteriorly, revealing the cords underneath. Miller blades are more commonly used in pediatric intubations where the epiglottis tends to be more floppy.
- Suction: A Yankauer suction catheter should be available to help remove saliva, blood, or emesis from the airway enhancing the view of the cords.

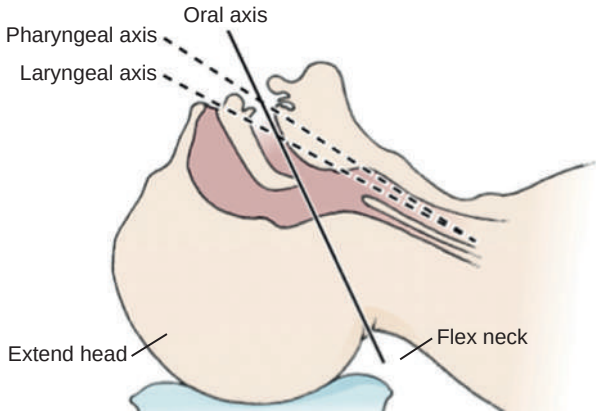

Figure 3-2. Sniffing position. *(From Reardon RF, McGill JW, Clinton JE: Tracheal intubation. In Roberts JR, Hedges JR, editors:* Clinical procedures in emergency medicine*, ed 6, Philadelphia, 2014, Saunders, p 69.)*

- ET tube: Adult males generally accept a 7.5- to 9-mm ET tube, whereas women are generally intubated with a 7- to 8-mm tube.
- Gum elastic bougie: A bougie is a semirigid introducer with a flexed tip that is commonly used for difficult intubations with incomplete visualization of the larynx. Lacking direct visualization, a bougie can be passed under the epiglottis and tracheal insertion can be confirmed as the flexed tip of the bougie bounces along the tracheal rings. An ET tube is then inserted into the trachea over the bougie.

12. What is a video laryngoscope, and what types are there?

A video laryngoscope is a laryngoscope blade with a camera embedded at its tip that displays images on a screen most commonly placed at the patient's bedside. There are several blade shapes, from a traditional Macintosh blade to more angulated blades. Video laryngoscopy has become the technique of choice for RSI, because it has been shown to provide superior views of the glottic opening and decreased time to intubation in difficult airways. Video laryngoscopy should be used for virtually all emergent intubations, and direct laryngoscopy should be abandoned as an antiquated technique.

13. What are the steps (seven Ps) to RSI?

1. Preparation: As with all procedures, preparation is the key to success. With every RSI attempt, equipment should be checked, medications should be planned ahead of time, and a back-up plan should be in place should the attempt fail.
2. Positioning: The sniffing position (Fig. 3-2) with the neck flexed relative to the torso and the head extended optimally aligns the oral, pharyngeal, and laryngeal axes for direct laryngoscopy.
3. Preoxygenation: Adequate preoxygenation maximizes the time available to perform the intubation before desaturation occurs by replacing alveolar nitrogen with oxygen (nitrogen washout). Proper preoxygenation can be achieved with 3 to 5 minutes of normal ventilation with a nonrebreather mask or with eight vital-capacity breaths at 100% fraction of inspired oxygen (FiO_2).
4. Pretreatment: Laryngoscopy is a strong stimulus that can activate both the sympathetic and parasympathetic nervous systems. Children younger than 10 years old should be given atropine to prevent bradycardia. Patients with head injuries may benefit from lidocaine and/or fentanyl to decrease the transient increase in intracranial pressure during intubation, although this has never been shown to improve outcomes.
5. Paralyze (with induction): Induction medication should be immediately followed by administration of the paralytic (Table 3-1).
6. Pass the tube: The tube should be visualized passing through the cords.
7. Postintubation management: Immediately after intubation and confirmation of tube placement, the patient should be sedated, most commonly with propofol or a combination of an opioid and benzodiazepine.

Table 3-1. Common Medications for Rapid Sequence Intubation

	Pretreatment			*Induction*				*Paralytic*	
	ATROPINE	**LIDOCAINE**	**FENTANYL**	**ETOMIDATE**	**KETAMINE**	**MIDAZOLAM**	**PROPOFOL**	**SUCCINYLCHOLINE**	**ROCURONIUM**
Class	Anticholinergic	Amino amide	Opioid analgesic	Imidazole derivative	PCP derivative	Benzodiazepine	GABA agonist	Depolarizing agent	Nondepolarizing agent
Dosage	0.02 mg/kg	1.5 mg/kg	3 μg/kg	0.3 mg/kg	1-2 mg/kg	0.1-0.2 mg/kg	1.5-3 mg/kg	1.5 mg/kg	1 mg/kg
Administration	3-5 min before intubation			Immediately before paralytic				Onset: 45-60 sec Duration: 5-9 min	Onset: 45-60 sec Duration: 5-9 min
Effect/benefit	Blunts bradycardic response to increased vagal tone from laryngoscopy Decreases bronchorrhea	Blunts elevation in ICP associated with laryngoscopy Decreased bronchospasm	Decreases sympathetic response to intubation (ICP, tachycardia, hypertension)	Hemodynamically neutral Decreases ICP	Bronchodilator (good for RAD) Preserves respiratory drive (awake intubation) Nystagmus Elevated ICP/IOP	Anticonvulsant effect Decreases ICP	Decreases ICP Decreased airway resistance Rapid on/off	Fasciculations Hyperkalemia Increased ICP, IOP	Nondepolarizing so no fasciculations or risk of hyperkalemia May increase HR, BP, CO
Notes	Recommended in all children <10 yr Max dose 1 mg	Indicated in patients with suspected head trauma	Use in patients at risk for decompensation with sympathetic surge (aortic dissection, ICH)	Proposed adrenal suppression, but not clinically relevant with single dose used for RSI	Adverse effects: Bronchorrhea Bronchospasm Tachycardia Hypertension Emergence phenomenon	Adverse effects: Negative inotrope Hypotension (mild)	Adverse effects: Hypotension Pain on infusion Anaphylaxis with soy/egg allergy	Risk of life-threatening hyperkalemia in burns, crush injury, neuromuscular disease, and acidosis	Prolonged duration may delay neurologic examination in trauma patients

BP, Blood pressure; *CO*, cardiac output; *GABA*, γ-aminobutyric acid; *HR*, heart rate; *ICH*, intracranial hemorrhage; *ICP*, intracranial pressure, *IOP*, intraocular pressure; *PCP*, phencyclidine; *RAD*, reactive airway disease; *RSI*, rapid-sequence intubation.

14. What is passive apneic oxygenation?

Passive apneic oxygenation is a technique for preventing hypoxia during RSI, in which a nasal cannula with high-flow oxygen is placed on the patient during the preoxygenation phase of RSI and left in place throughout the intubation attempt. Studies have shown that despite apnea during paralysis, passive oxygenation can greatly extend the time it takes for a patient to become critically hypoxic.

15. What medications are used for RSI?

Table 3-1 describes the pretreatment, induction, and paralytic medications commonly used for RSI.

16. What are the contraindications to using succinylcholine?

Because succinylcholine causes muscle depolarization and release of intracellular potassium, potentially life-threatening hyperkalemia can occur in certain high-risk populations. Succinylcholine is contraindicated in patients at risk for baseline hyperkalemia, including end-stage renal disease and severe acidosis; in patients with major burns or crush injuries in the past 3 to 5 days (not acutely); and in patients with any condition causing upregulation of acetylcholine receptors at the neuromuscular junction, including neuromuscular disease, stroke, and spinal injury.

17. How deep do I advance an ET tube?

A traditional rule is that an ET tube should be placed at a depth equal to three times the tube size in centimeters (e.g., an 8-mm tube should be placed 24 cm at the teeth). Because of airway anatomy, deeper tube placement will usually end up in the right mainstem bronchus.

18. How do I confirm ET tube placement?

ET tube placement is best confirmed by direct visualization of the tube passing through the vocal cords. Signs of correct tracheal tube placement include fog in the tube, bilateral breath sounds with bagging, absence of breath sounds over the epigastrium, and color change on colorimetric capnometry. Continuous waveform end-tidal carbon dioxide (CO_2) (capnography) is the most reliable method for confirming and monitoring correct ET tube placement. If continuous wave form end-tidal CO_2 is not available, a colorimetric end-tidal CO_2 detector should be used.

19. What are the contraindications to RSI?

RSI is contraindicated in any patient for whom securing of the airway is predicted to be difficult. Anticipation of a difficult airway based on anatomic features (foreign body, allergic reaction, airway infections, malignancies) or traumatic anatomic distortion (massive facial trauma, facial burns) is a relative contraindication to RSI. Difficult airway must be considered in the context of the environment, skill of the operator, and available equipment.

20. What are the steps to awake fiberoptic intubation?

Awake fiberoptic intubation is an excellent option for patients with some respiratory effort in whom RSI is contraindicated, most commonly as a result of airway obstruction. Patients can be placed in the upright position, the oropharynx is anesthetized with nebulized or topical anesthetic spray, and the patient is given moderate sedation, often with ketamine, maintaining the respiratory drive. A flexible fiberoptic scope threaded through an ET tube is then maneuvered into the oropharynx via a nasal or oral approach and passed through the cords under direct visualization, at which point the ET tube is advanced.

21. What is delayed sequence intubation (DSI)?

DSI can be used in patients with hypoxia refractory to traditional preoxygenation techniques. DSI is the use of ketamine for procedural sedation, with the procedure being preoxygenation. Patients are given 1 to 2 mg/kg of ketamine and then receive bag-valve-mask ventilation or are placed on a noninvasive ventilator until their oxygenation can be maximized, at which point standard RSI is preformed.

22. What is an extraglottic airway?

An extraglottic airway is a device that is placed without direct visualization above or posterior to the larynx, typically blocking off the esophagus, allowing ventilation and oxygenation. These are good rescue devices for patients who are difficult to ventilate with a bag-valve mask or as a temporizing method after failed ET intubation.

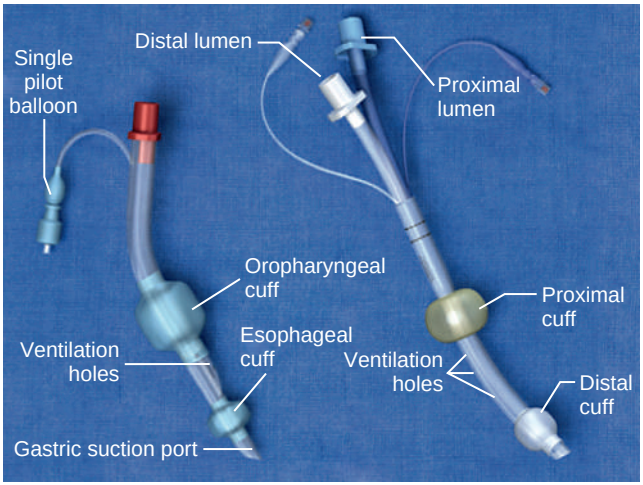

Figure 3-3. King LT *(left)* and Combitube *(right).* *(From Reardon RF, Mason PE, Clinton JE: Basic airway management and decision making. In Roberts JR, Hedges JR, editors:* Clinical procedures in emergency medicine, *ed 6, Philadelphia, 2014, Saunders, p 57.)*

23. What is a laryngeal mask airway (LMA)?

An LMA is an oval mask with a cuffed rim that is inserted into the oropharynx and is intended to create a seal directly over the larynx, allowing ventilation and oxygenation. An intubating LMA is an LMA with a rigid channel through which an ET tube can be directed through the cords. LMAs are considered the rescue device of choice in a cannot-intubate/cannot-ventilate scenario.

24. What is a King airway? What is a Combitube?

Both the Combitube and the King airway (Fig. 3-3) are other types of extraglottic devices that are commonly used by prehospital providers.

- King airway: The King LT is a single-lumen tube with a small distal balloon and a larger proximal balloon that is inserted blindly into the mouth with the intention of placing the tip in the esophagus. The two balloons are then inflated, blocking the esophagus and oropharynx and isolating the extraglottic space. Ventilation is achieved through a side port between the two balloons.
- Combitube: The Combitube is a dual-lumen tube that is blindly inserted into the mouth. If the tip is placed in the esophagus (95% of the time), both pharyngeal and esophageal balloons can be inflated, and using the longer lumen, the patient can be ventilated through a side port similar to the King LT. If the tip of the tube is placed in the trachea, the patient can be ventilated through the distal tip using the shorter lumen.

25. What are the indications for a surgical airway?

Cricothyrotomy is the surgical airway of choice in the ED for patients with a failed airway. A failed airway is failure to intubate, ventilate, and oxygenate by other means. A cricothyrotomy is performed by making a vertical incision over the cricothyroid membrane, palpating down to and making a horizontal incision through the membrane, and inserting an ET tube.

26. What factors make pediatric airway interventions more difficult?

Direct laryngoscopy can be more challenging in the pediatric patient because of anatomic differences, including a relatively large occiput causing neck flexion, a more superior and anterior larynx, a relatively larger tongue, and an epiglottis that is shorter and more difficult to manipulate. Additionally, pediatric patients have higher relative oxygen consumption and lower residual capacity and therefore become hypoxic much more quickly than adult patients.

Table 3-2. Laryngoscope Blade Sizes for Pediatric Intubations

AGE	LARYNGOSCOPE BLADE SIZE
Premature infant	0
Full-term infant	1
Older children	2
Adults	3-4

27. How do I know what equipment size to use for pediatric airway interventions?

Laryngoscope sizes can be seen in Table 3-2, and uncuffed ET tube size can be estimated using a Broselow tape or with the following formula (subtract 0.5 for cuffed tubes):

$$\text{ET tube size} = (\text{Age}/4) + 4$$

28. What is the surgical airway option for pediatric patients?

Because of anatomic differences in children, including a smaller or absent cricothyroid membrane and an immature larynx, surgical cricothyrotomy is contraindicated in children younger than 8 years old. The surgical airway of choice in pediatric patients is transtracheal jet ventilation, in which a large-bore needle is inserted through the cricothyroid membrane and high-flow oxygen is delivered.

KEY POINTS: AIRWAY MANAGEMENT

1. Approach every airway with a clear plan in place for initial intervention, as well as a complete set of back-up plans should your initial attempt be unsuccessful.
2. Preoxygenation with 100% FiO_2 for 5 minutes or 8 vital-capacity breaths and passive apneic oxygenation are critical steps to prevent oxygen desaturation during RSI.
3. Never paralyze a patient unless you are certain that they can be ventilated using a bag-valve mask or an extraglottic rescue device.
4. Succinylcholine is contraindicated in patients at risk for baseline hyperkalemia or an exaggerated potassium shift, including end-stage renal disease, severe acidosis, neuromuscular disease, and delayed presentations of burn or crush injuries.
5. Video laryngoscopy provides a better view of the vocal cords, has been shown to decrease time to intubation in difficult airways, and should be the first choice for almost all RSI attempts in the ED.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Barry Simon for his previous contributions to this chapter.

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QUESTIONS

1. In which of the following cases would the use of succinylcholine for RSI be considered safe?
   a. A 66-year-old female brought from a nursing home with fever, tachycardia, altered mental status, and an electrocardiogram (ECG) showing diffuse peaked T waves
   b. A 34-year-old male trauma patient with bilateral comminuted femur fractures, postoperative day 4, who failed extubation in the surgical intensive care unit and requires reintubation.
   c. A 52-year-old man brought in by paramedics after being pulled from a fire with full thickness burns to his chest, abdomen, and back and soot in his oropharynx
   d. A 59-year-old female with a history of right-sided hemiparesis from a prior ischemic stroke exhibiting symptoms of respiratory failure likely caused by aspiration pneumonia

   The correct answer is *c.*
2. A 6-year-old male is brought in to the ED after being ejected from a motor vehicle. He has a score of 3 on the Glasgow Coma Scale (GCS), and the decision is made to intubate. What size laryngoscope blade should be used; what size ET tube should be used; and at what depth should it be placed?
   a. Miller 1 laryngoscope, 2.5 cuffed ET tube inserted to 8 cm at the teeth
   b. Miller 2 laryngoscope, 5 cuffed ET tube inserted to 15 cm at the teeth
   c. Miller 2 laryngoscope, 5 cuffed ET tube inserted to 20 cm at the teeth
   d. Miller 4 laryngoscope, 5.5 cuffed ET tube inserted to 20 cm at the teeth

   The correct answer is *b.*
3. Which medication order and dosages would be appropriate for RSI of a 28-year-old male (70 kg) who is unresponsive with a score of 3 on the GCS after being hit in the head with a baseball bat?
   a. Succinylcholine 100 mg, etomidate 20 mg, fentanyl 200 mg
   b. Ketamine 140 mg, succinylcholine 100 mg
   c. Atropine 0.5 mg, etomidate 20 mg, succinylcholine 100 mg
   d. Fentanyl 200 μg, etomidate 20 mg, succinylcholine 100 mg

   The correct answer is *d.*

CHAPTER 4

SHOCK

Jason S. Haukoos, MD, MSc

1. Define *shock*.

Shock is 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.

1. Hypovolemic: Examples include trauma, gastrointestinal bleeding, ruptured ectopic pregnancy, ruptured abdominal aortic aneurysm, and diabetic ketoacidosis.
2. Cardiogenic: Examples include acute myocardial infarction, cardiomyopathy, and valvular dysfunction.
3. Distributive: Examples include sepsis, anaphylaxis, and spinal cord injury.
4. Obstructive: Examples include pulmonary embolism (PE), cardiac tamponade, and tension pneumothorax.
5. Toxic/metabolic: Examples of sources include carbon monoxide, cyanide, β-blocker, calcium channel blocker, adrenal insufficiency, and thyroid storm.

5. How do I 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 whether shock is present and its underlying cause. Examples of system-based symptoms and signs include the following:

- Central nervous system: Altered mentation
- Cardiovascular: Decreased cardiac output (CO), tachycardia, hypotension, and weak rapid pulses
- Pulmonary: Tachypnea and hyperpnea
- Renal: Decreased urine output
- Skin: Delayed capillary refill; skin is 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 experiencing shock should have a Foley catheter in place 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 more than 1 mL/kg/h, a reduced urine output ranges from 0.5 to 1 mL/kg/h, and a severely reduced urine output is less than 0.5 mL/kg/h. 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 may progress to

Abstract

Shock is a clinical syndrome characterized by widespread inadequate oxygenation and supply of nutrients to tissues and organs, resulting in cellular dysfunction. Although shock is somewhat rare, patients who develop shock have a relatively high mortality that is dependent on time to identification and aggressiveness of treatment. General treatment of shock centers on maximizing oxygenation and ventilation, maintaining adequate tissue perfusion, and treating the underlying cause. Specific treatment depends on the reason for shock, which may be grouped into five distinct categories: hypovolemic, distributive, cardiogenic, obstructive, and toxic/metabolic.

Keywords:

shock, hypovolemic shock, cardiogenic shock, distributive shock, obstructive shock, toxic/metabolic shock, etiology, diagnosis, treatment

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 is experiencing shock?

Management of patients in shock begins with airway, breathing, and circulation (ABCs). Because of poor delivery and uptake of oxygen, all patients should receive either 15 L of oxygen by nonrebreather mask or intubation. Additionally, all patients should have large-bore intravenous access and a cardiac monitor.

9. Define oxygen delivery (DO_2).

$$DO_2 = CaO_2 \times CO$$

CaO_2, Arterial oxygen concentration.

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

Hgb, Hemoglobin;
SaO_2, arterial oxygen saturation;
PaO_2, arterial partial pressure of oxygen.

DO_2 is the product of CO and CaO_2. CaO_2 is defined by the Hgb level, the SaO_2, and the PaO_2. Maximizing CO, Hgb, SaO_2, and the PaO_2 will maximize DO_2.

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

Vital signs are crucial. Heart rate, respiratory rate, blood pressure, and pulse oximetry should be monitored closely in patients experiencing, or with suspected, 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 CO and often do not reflect 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 thought to be experiencing shock should not be allowed to stand as a method of assessing changes in vital signs.

13. Are there other signs that are helpful in assessing an acutely ill patient?

Yes. Besides vital signs and 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 concentration, central venous pressure (CVP), and central venous oxygen saturation ($ScvO_2$) or mixed venous oxygen saturation (SvO_2).

14. How should I use and interpret serum lactate concentration?

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 before 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 greater than 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 measurements of 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. CVP 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 intercostal 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 experiencing shock?

The guiding principal for using 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 is depressed. A supranormal CVP thus allows for improved cardiac filling.

18. What is venous oxygen saturation, and what is the difference between $ScvO_2$ and SvO_2?

Venous oxygen saturation provides a measure of tissue oxygenation (i.e., the balance between DO_2 and oxygen 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 $ScvO_2$ or SvO_2 during resuscitation?

An $ScvO_2$ less than 65% suggests decreased oxygen supply or increased demand. In response, attempt to improve DO_2 by increasing SaO_2 and/or PaO_2 via oxygen supplementation, Hgb concentration via transfusion, and/or CO via inotropic support.

20. What is early goal-directed therapy?

Goal-directed therapy refers to the practice of resuscitating patients to defined physiologic end points (e.g., mean arterial pressure, CVP, urine output, serum lactate concentration, CO, Hgb level, and SvO_2), indicating that systemic tissue perfusion and vital organ function have been restored. In the ED, goal-directed therapy has been rigorously studied in patients with sepsis; however, it is being evaluated in patients with postcardiac arrest and trauma. 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 end points and survival.

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

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

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

A patient in the Trendelenburg position is 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 intraabdominal 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 conditions:

- Temperature higher than 38° or 36°C
- Heart rate faster than 90 beats per minute
- Respiratory rate greater than 20 breaths per minute or partial pressure of carbon dioxide ($PaCO_2$) less than 32 mm Hg.
- Serum white blood cell count greater than 12,000 mm^3 or less than 4000 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, or overdose.

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

See Chapter 49.

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 cause and administer specific treatment (e.g., thrombolysis or percutaneous coronary intervention in the setting of acute coronary syndrome). Consider intraaortic 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 CO. 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 with dobutamine and that tachyphylaxis may result from its use if used for a prolonged period of time.

28. How do I treat shock resulting from anaphylaxis?

See Chapter 20.

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 caused by PE?

Massive PE causes shock by reducing the cross-sectional area of the pulmonary outflow tract, thus increasing right-sided heart pressures and reducing blood flow to the left side of the heart, all of which result 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 resulting from 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. Patients experiencing neurogenic shock 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 CO.

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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QUESTIONS

1. Shock is characterized by:
   a. Adequate nutrient delivery to tissues
   b. Inadequate ventilation resulting in cellular dysfunction
   c. Adequate oxygenation resulting in free radical formation
   d. Inadequate oxygenation resulting in hypoxia
   The correct answer is *d.*
2. Appropriate therapies for cardiac tamponade include all of the following except:
   a. Pericardiocentesis
   b. Aggressive intravenous fluid administration
   c. Supplemental oxygen
   d. Blood transfusion
   The correct answer is *d.*
3. What mechanism of shock is present in patients with PE, and what are the best therapeutic approaches?
   a. Cardiogenic; dobutamine
   b. Obstructive; intravenous fluids and thrombolytics
   c. Obstructive; intravenous fluids and pericardiotomy
   d. Obstructive; intravenous fluids and norepinephrine
   The correct answer is *b.*

EMERGENCY ULTRASOUND

Molly E.W. Thiessen, MD

CHAPTER 5

1. What is ED ultrasound all about?

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

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, intraabdominal 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. The most recent American College of Emergency Physicians (ACEP) ultrasound guidelines add echocardiography and soft-tissue/musculoskeletal, urinary tract, thoracic, ocular, and deep venous thrombosis (DVT) ultrasound to these core emergency ultrasound modalities, and their use in the ED can assist in rapid diagnosis of a variety of conditions. Additionally, ultrasound for procedural guidance has been shown to improve the safety of emergency procedures.

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 a sonographic Murphy sign in right upper quadrant pain; aortic dilation in suspected AAA; and pericardial fluid in patients with possible pericardial tamponade are used to make real-time patient care decisions in the ED. In contrast, an ultrasound performed by a radiologist 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 *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 pouch (Fig. 5-1), the splenorenal recess, the 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).

Abstract

This chapter describes the current use of clinical ultrasound in the emergency department.

Keywords:

emergency ultrasound, emergency sonography, echocardiography, focused assessment with sonography for trauma (FAST), extended focused assessment with sonography for trauma (EFAST)

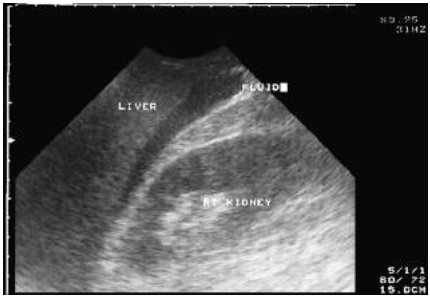

Figure 5-1. View of the Morison pouch showing intraperitoneal fluid. *RT,* Right.

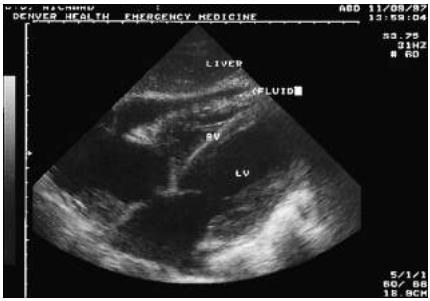

Figure 5-2. Subxiphoid cardiac view shows a pericardial effusion. *LV,* Left ventricle; *RV,* right ventricle.

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, 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.

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

Consider patient scenarios based on the following vital signs and ultrasound findings:

- Stable vital signs, negative ultrasound
- Stable vital signs, positive ultrasound
- Unstable vital signs, negative ultrasound
- Unstable vital signs, positive ultrasound

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, other sources of hypotension should be considered, and if intraabdominal injury is not excluded, a bedside diagnostic peritoneal lavage can be performed. If the vital signs are unstable and the ultrasound is positive for free fluid, the patient should directly undergo a laparotomy.

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

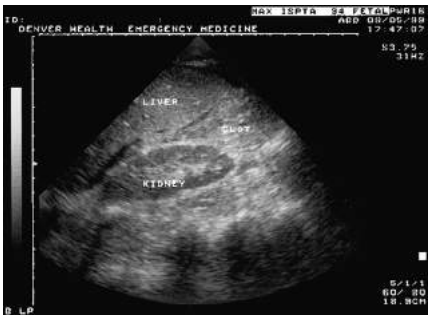

Figure 5-3. Clotted blood in the Morison pouch.

9. Can I determine 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 the Morison pouch is shown in Figure 5-3. This image was initially 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 a fluid-filled stomach or bowel.

11. What is extended focused assessment with sonography for trauma (EFAST)?

EFAST incorporates use of thoracic ultrasound into a standard FAST examination to detect pneumothorax. In most cases, a high-frequency linear transducer is applied to the anterior chest wall in the midclavicular line at the level of the second intercostal space, and the clinician is looking for the absence of normal lung "sliding" and lack of comet-tail artifact, indicating pneumothorax. Additional views are often obtained in the midaxillary line, and alternative probes may be used. EFAST has been found to be more sensitive than chest radiography for the detection of occult pneumothoraces in patients experiencing trauma (48.8% versus 20.9%), although both have a very high specificity (99.6% for EFAST and 98.7% for chest radiography).

KEY POINTS: ELEMENTS OF THE EFAST EXAMINATION

1. Hepatorenal recess or Morison pouch image (see Fig. 5-1) to search for hemoperitoneum, with additional oblique view of the right chest to search for hemothorax
2. Splenorenal recess image to search for hemoperitoneum, with additional oblique view of the left chest to search for hemothorax
3. Retrovesicular recess image (pouch of Douglas in females) to search for hemoperitoneum
4. Images of both pericolic gutters to search for hemoperitoneum
5. Subxiphoid or left parasternal cardiac image to search for pericardial effusion
6. Bilateral anterior chest wall images to evaluate for pneumothorax

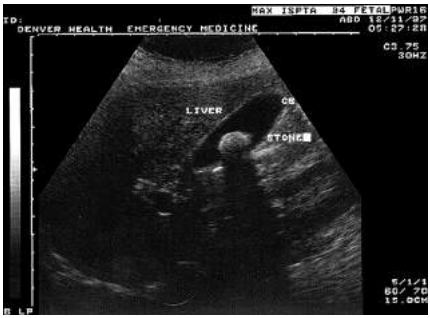

Figure 5-4. Long-axis view of the gallbladder shows a gallstone. The gallstone is represented by an echogenic proximal surface and distal attenuation shadow. *GB,* Gall bladder.

12. What are the sonographic appearances of the gallbladder and related structures?

The gallbladder is cystic, so the sonographic appearance is a pear-shaped 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 it can be thicker immediately after eating or if in an edematous state, 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, 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 collects in layers within the gallbladder and appears sonographically as mildly echogenic material without any shadowing.

13. What findings are suggestive of acute cholecystitis?

The primary findings of the emergency gallbladder ultrasound are the presence of gallstones and a sonographic Murphy 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.

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

Ultrasonography is the imaging study of choice for evaluating abdominal pain or bleeding in patients in the first or second trimester of pregnancy. 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.

15. 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' gestational age by transvaginal ultrasound (6 weeks or more using transabdominal ultrasound). The discriminatory zone is the level of β-HCG at which one would expect to see evidence of an IUP by ultrasound. Although this depends on the institution where the patient is being seen, it is typically at a β-HCG level of 1000 to 2000 mIU/mL by transvaginal ultrasound and 5000 mIU/mL by transabdominal ultrasound. A gestational sac is seen at approximately 4 to 5 weeks' gestational age, and cardiac activity can be measured as early as 6 weeks' gestational age.

16. 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). In the remaining 20% of patients with nondiagnostic ultrasounds, nearly one fourth have ectopic pregnancies. Patients with a β-HCG level above the discriminatory zone without evidence of an IUP on ultrasound are at particularly high risk of an ectopic pregnancy, even in the absence of a visualized ectopic pregnancy at the time of ED

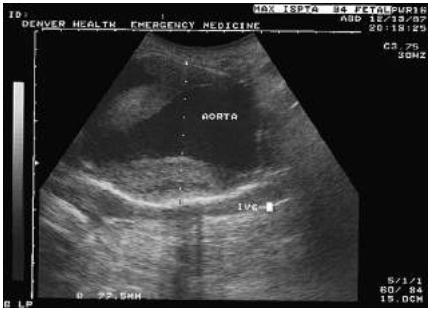

Figure 5-5. Long-axis view of a 7.75-cm diameter abdominal aortic aneurysm. *IVC,* Inferior vena cava.

examination. This increased risk of ectopic pregnancy among patients with nondiagnostic ultrasounds warrants obstetric-gynecologic consultation in the ED.

17. 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. Thus a comprehensive pelvic ultrasound performed by a certified sonographer must be performed in these patients, irrespective of an IUP identified on bedside ultrasound. A pseudosac can be seen in 20% of ectopic pregnancies. It consists of a single-ringed structure in the endometrial cavity, formed in response to the β-HCG produced by the abnormal pregnancy, and is easily mistaken for a true gestational sac, which consists of two concentric rings.

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

Evaluation of the abdominal aorta can be useful in elderly patients who have a pulsatile abdominal mass, nontraumatic abdominal pain, 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.

19. 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 per 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. Patients who have aneurysms that rupture have a mortality of greater than 80%, so ultrasound is an important tool in the detection of AAA.

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

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

- 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.
- It may be used for detection of nontraumatic pericardial effusions (i.e., malignancy, uremic, rheumatologic).
- It may be used for the detection of right ventricular strain as seen in the setting of a pulmonary embolus.
- Another important indication includes the evaluation of patients experiencing cardiac arrest. Contractility can be assessed in patients with cardiac arrest when there is a question of pulseless electrical activity. When there is no evidence of cardiac contractility and other reversible causes

Table 5-1. Emergency Ultrasound Core Application

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

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

of pulseless electrical activity have been ruled out, strong consideration should be given to terminating the resuscitation.

- Emergent echocardiography can be used for assessing undifferentiated hypotension.

21. How can ultrasound be used in the ED to evaluate patients with undifferentiated hypotension?

A combination of ultrasound evaluations can be used to differentiate the reasons for shock in ED patients: a subcostal cardiac view to evaluate for pericardial effusion and tamponade; an inferior vena cava (IVC) view looking for greater than 50% collapse with inspiration, indicating low intravascular volume; a parasternal long-axis cardiac view to estimate overall left ventricular function; an apical four-chamber cardiac view to estimate overall function and evaluate relative chamber size; a view of the hepatorenal recess to evaluate for free intraperitoneal fluid; a view of the pelvis and retrovesical area to evaluate for free intraperitoneal fluid; and finally a few of the abdominal aorta to evaluate for aneurysm. Use of this systematic, goal-directed protocol in patients in the ED who have undifferentiated nontraumatic hypotension allows physicians to narrow their differential diagnosis sooner and gives them a more accurate impression of the final diagnosis (Table 5-2).

22. 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 with suspected renal colic. If hydronephrosis without a known cause is seen on ED ultrasound, further imaging should be pursued.

22. How is lower extremity venous ultrasound performed in the ED to diagnose 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

Table 5-2. Use of Ultrasound for Undifferentiated Shock

	CARDIAC VIEWS	IVC VIEW	ABDOMINAL VIEWS
Hypovolemia caused by abdominal injury	Hyperdynamic	>50% collapse with inspiration	+ Free fluid
Hypovolemia without injury	Hyperdynamic	>50% collapse with inspiration	– Free fluid
Tamponade	+ Pericardial effusion on subcostal cardiac view	No collapse	
AAA	Hyperdynamic (depending on patient's underlying cardiac function)	+/– collapse depending on presence of abdominal free fluid	+ AAA, +/– free fluid
Cardiogenic shock	Globally hypodynamic or regional wall-motion abnormalities on parasternal long-axis and apical four-chamber cardiac views	No collapse	– Free fluid
Pulmonary embolism	Right heart strain on parasternal long-axis and apical four-chamber cardiac views	No collapse	– Free fluid

AAA, Abdominal aortic aneurysm; *IVC*, inferior vena cava.

not touch; the clot echogenicity can vary greatly from echogenic to nonechogenic. 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.

23. How can soft-tissue/musculoskeletal ultrasound be utilized in the ED?

The most common way to use ultrasound for this modality in the ED is for assessment of abscess in the setting of cellulitis. Ultrasound has been shown to be 98% sensitive and 88% specific for suspected abscess (versus needle aspiration). It has been shown to change management in 56% of patients with cellulitis. It is also expanding to include fracture detection, tendon injuries, and joint effusions.

24. What are some future applications for emergency ultrasound?

Uses for emergency ultrasound continue to rapidly expand. This is clearly demonstrated in the 2008 ACEP clinical practice guidelines for ultrasound that expand upon core applications. For instance, one of the fastest-growing applications is to guide invasive procedures. This is not confined to vascular access but is also applicable to other procedures, such as localization and drainage of abscesses, nerve blocks, lumbar puncture, fracture identification and reduction, 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, retinal detachment, and lung pathology. In the pediatric population, it is increasingly being used to detect appendicitis, pyloric stenosis, and intussusception.

25. 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 evolved from a novelty experience to something that is taught across all levels of medical education. Many medical schools are incorporating clinical ultrasound into their basic science curriculum. It is recommended by ACEP to be taught in all residency programs early in training, is a required

element of the recently instated Accreditation Council for Graduate Medical Education (ACGME) milestones, and is tested on emergency medicine specialty boards and the national in-service examinations. There are now more than 90 emergency ultrasound fellowships across the country, and ultrasound 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 or should be used for optimal care of patients in the ED.

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QUESTIONS

1. A 62-year-old male comes to the ED with abdominal pain and hypotension. The patient has had no recent trauma. The most appropriate initial ultrasound evaluation is:
   a. An EFAST examination to look for hemoperitoneum
   b. A goal-directed ultrasound looking at the heart, IVC, and abdomen to look for causes of shock
   c. Ultrasound of the gallbladder to look for cholecystitis
   d. Ultrasound of the abdominal aorta to look for aneurysm
   The correct answer is *b*.
2. A 23-year-old male comes to the ED after a high-mechanism motor vehicle collision. His initial EFAST is normal. He has stable vital signs, no other injuries, and a normal mental status. The most appropriate next step for this patient is:
   a. Observation with serial physical examinations and ultrasound studies
   b. Abdominal CT scan
   c. Bedside diagnostic peritoneal lavage
   d. Immediate laparotomy
   The correct answer is *a*.
3. A 34-year-old female comes to the ED with abdominal pain. Her vital signs are: P105, BP 82/40, R28, T37.0, SaO_2 98% RA. A urine pregnancy test is positive, and β-HCG is 2005 mIU/mL. A bedside transvaginal ultrasound shows an empty uterus. The appropriate next step(s) for this patient include:
   a. Discharge home with a follow-up visit to her primary obstetrician for repeat β-HCG testing in 48 hours
   b. Abdominal CT scan
   c. Consultative ultrasound in the radiology suite
   d. Immediate obstetrics/gynecology consultation for suspected ruptured ectopic pregnancy
   The correct answer is *d*.

GERIATRIC EMERGENCY MEDICINE

Thomas Kelly, MD, FACEP

CHAPTER 6

1. Why dedicate a chapter to geriatric emergency medicine?

Elderly persons are a rapidly growing segment of the population. According to the 2010 U.S. Census, more than 40 million Americans were over the age of 65, which was more people than in any previous census. In addition, between 2000 and 2010, the population of people 65 years and older increased at a faster rate than the total U.S. population. By the year 2030, this number is expected to double. The census data also demonstrated that the population aged 85 and older is growing at a rate almost three times the general population. Elderly patients account for up to 24% of all ED visits and represent 43% of admissions, including 48% admitted to the intensive care unit (ICU). On average, the geriatric patient stays in the ED longer and requires more diagnostic studies than younger patients.

Geriatric patients have unique medical and social characteristics. They often have multiple medical comorbidities, multiple medications, and complex physiologic changes that create great challenges. Diseases often present atypically in elderly patients. The older patient often has reduced functional reserves, which make a careful assessment of the patient's psychosocial environment essential before making decisions about disposition.

2. What is a geriatric ED?

Geriatric EDs are an emerging phenomenon across the United States, designed to provide greater comfort for elders, screen for common morbidities, and allow for selective contact with social workers. It is a specialized, acute care, interdisciplinary model used to identify the distinct needs of the geriatric population. The model used makes use of screening for high-risk conditions with geriatric-specific instruments, along with initiation of treatment, disposition, and follow-up planning in the ED.

3. What important physiologic changes occur with aging?

- Cardiovascular: Decreased cardiac output and compensatory mechanisms with increased systolic blood pressure and systemic vascular resistance
- Pulmonary: Decreased vital capacity and decreased functional reserve
- Musculoskeletal: Loss of muscle strength and mass, impaired mobility, and decreased bone mineralization
- 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

4. 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/h)
- Decreased hemoglobin (11.0 g/dL in women or 11.5 g/dL in men)
- Elevated blood urea nitrogen (28 to 35 mg/dL)

5. 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

Abstract

The geriatric age group is a rapidly growing segment of our population. Elderly patients have physiologic changes, comorbidities and unique medical and social characteristics that create significant challenges for the emergency physician. Falls in geriatric patients are a serious problem and may be the result of environmental or physiologic causes. Consider a fall as the initial presentation of a more serious disease. Atypical presentations of serious disease are more common in elderly patients.

Keywords:

geriatric, elderly, abdominal pain, fall, abuse, dementia, delirium, trauma

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.

6. 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. Falls are the main cause of ED admissions for elderly patients (15% to 30%).

7. 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. Nearly 6% of falls result in fractures. Falls may also be the chief symptom of other pathologies, such as acute myocardial infarction (AMI), sepsis, medication toxicity, acute abdominal pathology, and elder abuse. Use of psychotropic medications, such as benzodiazepines, narcotics, and other sedatives, are associated with an increased risk of falls in the elderly.

8. 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.

9. What about coronary artery disease in the older patient?

Age is a well-known risk factor for coronary artery disease, with 30% of AMIs occurring in patients older than 75 years and more than 60% of patients hospitalized for unstable angina older than 65 years. In the ED, approximately 20% of older patients have dyspnea or chest pain as principal complaints. Coronary disease mortality is also high, with 80% of deaths caused by ischemic heart disease occurring in patients older than 60 years.

10. Should I be concerned about atypical presentations of 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 electrocardiograms (ECGs). 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

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

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

12. Speaking of infections, how do infectious pathologies present in older patients?

Infection is the main complaint of 4% of elderly patients in the ED. The most common conditions are pneumonia (25%), urinary tract infection (22%), and sepsis and bacteremia (18%). Infectious presentation is often atypical in this population. Falls or delirium may be the only clinical manifestations of otherwise serious infections, whereas more classic symptoms such as tachycardia and fever may be absent. Thus acute cholecystitis may present without pain in 5% of cases, without fever in 56%, or without complete blood count abnormalities 41%. Appendicitis presents with classic symptoms in only 20% of geriatric cases, and fever occurs in less than half.

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

Adverse drug-related events and polypharmacy are a significant cause of morbidity in elderly patients and lead to 11% of ED visits for those older than 65 years. They 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 patients who are institutionalized. Adverse reactions to medications are directly proportional to the number of medications being taken. Recent data suggest that three medication classes caused 48% of all ED visits for adverse drug effects in patients older than 65 years: oral anticoagulant or antiplatelet agents (warfarin, aspirin, and clopidogrel), antidiabetic agents (insulin, metformin, glyburide, and glipizide), and agents with a narrow therapeutic index (digoxin and phenytoin).

14. 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

15. Do elderly patients tolerate trauma very well?

No. Elderly patients have distinct characteristics that differ from other trauma victims. Geriatric patients have different mechanisms of injury, with falls making up a large proportion of severe trauma. The number of comorbidities and the use of anticoagulants generally are higher in the geriatric population, and this appears to contribute directly to poorer outcomes. Whereas geriatric patients make up only 18.6% of the trauma population, they account for 28% of trauma deaths in the United States and consume approximately one third of health care dollars spent on trauma.

16. Should I worry if a geriatric victim of trauma 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 patient with hypertension. 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.

17. 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

18. 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 is also 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.

19. 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.

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

Any complaint of abdominal pain in the elderly should be taken seriously. Compared with that of younger patients, mortality rates are 6 to 8 times higher and surgery rates are doubled. Elderly patients have decreased pain perception and are more likely to have normal vital signs in the face of significant intraabdominal pathology. Aging is associated with a decreased response to pyrogens, lower basal body temperature, changes in thermal homeostasis, and a decreased production and conservation of heat. In one study, 30% of older adults who had surgical abdominal pain did not show signs of either a fever or leukocytosis. 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. Computed tomography (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.

21. 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.

22. How do I differentiate between delirium and dementia?

See Table 6-1.

23. What are the four types of elder abuse?

Elder abuse prevalence in the United States is estimated at 11% and occurs as one or more of the following types:

- Physical abuse: Nonaccidental force that results in bodily injury or pain (e.g., hitting, biting, slapping, sexual assault, burns, or unreasonable restraint [physical, chemical])
- Psychological abuse: Threats made with the intent of causing emotional pain or injury
- Exploitation: Caretaker use of the resources of an elder for monetary or personal profit
- 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.

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

24. 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

25. 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

26. 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?
- 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?

KEY POINTS: PRINCIPLES OF GERIATRIC EMERGENCY MEDICINE

1. The geriatric age group (65 years and over) is a rapidly growing segment of the population, and their numbers are expected to double in the next 15 years.
2. Elderly patients have physiologic changes, comorbidities, as well as unique medical and social characteristics that create significant challenges for the emergency physician.
3. Falls in geriatric patients are a serious problem and may be the result of environmental or physiologic causes. Consider a fall as the initial presentation of a more serious disease.
4. Atypical presentations of serious disease are more common in elderly patients.

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QUESTIONS

1. Geriatric patients pose unique challenges for emergency physicians because of:
   a. Polypharmacy and sound physiologic reserves
   b. Comorbidities and atypical presentations of disease
   c. Immunocompetence and skeletal bone mineralization
   d. Increased lean body mass and distinctive social characteristics
   The correct answer is *b.*
2. Which statement about infections in older patients is correct?
   a. Skin and soft-tissue infections are most common, followed by urinary tract infections.
   b. Because they are deconditioned, tachycardia and fever are always present in serious infections.
   c. Falls or delirium may be the only clinical manifestation of a serious infection.
   d. Acute appendicitis presents with classic symptoms in nearly half of geriatric cases.
   The correct answer is *c.*
3. Elderly patients in the ED are more susceptible than most younger patients to all of the following, except:
   a. Hypothermia
   b. Adverse drug reactions
   c. Exploitation
   d. Sexually transmitted diseases
   The correct answer is *d.*

CHAPTER 7

PALLIATIVE CARE AND ADVANCE DIRECTIVES

Cara Bergamo, MD, and Jean Abbott, MD, MH

PALLIATIVE CARE

1. **What is palliative care (PC) medicine?**
   PC is the medical specialty focused on relieving the symptoms and stresses of serious and complex medical illnesses. The goal is to improve quality of life and prevent suffering by addressing the physical, intellectual, emotional, social, and spiritual needs of patients and families. PC's interdisciplinary approach provides comprehensive support for patient and family members and can be used in conjunction with curative or life-sustaining treatments.

2. **What is the difference between PC and hospice?**
   PC is the general term for medical care focusing on comfort and symptom management—quality of living over quantity of life—for patients whose serious illness is not curable. Hospice, in the United States, is an interdisciplinary service covered by major insurances, including Medicare, for patients who are no longer pursuing life-prolonging therapies and whose life expectancy is 6 months or less if their disease takes its normal course. Hospice teams include nurses, physicians, aides, social workers, clergy, and trained volunteers dedicated to addressing medical, spiritual, and psychosocial needs near the end of life.

3. **Why should PC skills be part the scope of emergency medicine?**
   People increasingly live with chronic, complex diseases that require a broad range of support services. This type of disease burden often requires urgent and aggressive symptom control, with or without interventions that extend life. For such patients, the aim in the ED is to discern the patient's medical needs and to use the staff's expertise to reduce suffering and provide medical interventions in line with patient goals.

4. **How does emergency medicine fit in with the specialty of PC and hospice?**
   The American Board of Emergency Medicine was one of 10 boards that sponsored the newly established subspecialty of Hospice and Palliative Care Medicine in 2006. This sponsorship recognizes that emergency physicians need PC skills in urgent or emergent situations for those patients who need medical management but may wish to avoid burdensome or invasive interventions near the end of life. Emergency physicians can do a fellowship in PC after residency or take continuing medical education offerings in the field. Resources and essential tools for emergency medicine physicians are available through the National Center to Advance Palliative Care at http://www.capc.org/ipal/ipal-em.

5. **What are the core PC skills that emergency physicians need to know?**
   PC skills for emergency medicine physicians are listed in Table 7-1. Although PC specialists may be available in some acute care hospitals, these core competencies should be part of initial emergency medicine care. Many treatments can be considered to be palliative interventions if their purpose is to improve quality of life and reduce suffering. ED management of patients focusing on palliative interventions may include fluid resuscitation, administering pain- and symptom-directed medications, starting antibiotics, or even some surgical interventions (e.g., decompressing a bowel obstruction or pinning a hip fracture). Invasive diagnostics and treatment interventions must be assessed for their burdens, benefits, and overall congruence with the patient's goals and values.

6. **Why would a hospice patient call 911? Shouldn't they be calling their hospice provider?**
   Patients and families call emergency medical services (EMS) when they are overwhelmed with symptoms such as pain, fatigue, or trouble breathing. Although hospice organizations commit to

Abstract

Emergency medicine physicians are required to have the skills for managing symptoms and recognizing palliative care (PC) needs in patients with serious and complex illnesses.

Keywords:

resuscitation preferences, end of life, palliative care (PC), hospice, do not resuscitate (DNR), do not attempt resuscitation (DNAR), advance directive

Table 7-1. Core Palliative Skills for Emergency Medicine Physicians

Performing a rapid palliative care assessment
Establishing goals of care
Assessing prognosis and trajectory of illness
Understanding advance directives
Delivering bad news and death disclosure
Symptom management
Management of pain, malignant and nonmalignant
Spiritual, psychologic, and social needs assessment
Recognizing and managing last hours of life

Modified from Desandre PL, Quest TE, Portenoy RK, editors: Palliative aspects of emergency care. New York, 2013, Oxford University Press.

providing 24-hour services 7 days per week, families and patients may panic if they believe a more urgent response is necessary. EDs and EMS providers should not ask patients to stop calling for help, but rather titrate the medical response for the important and growing number of end-of-life and hospice patients.

7. What is an advance directive?

The term "advance directive" refers to one of a number of documents that establish a patient's wishes or designate a surrogate to make medical decisions if the patient cannot speak for himself or herself. Table 7-2 lists the most common forms you may encounter when a patient comes to the ED.

8. How should advance directives be used when they are available to the emergency physician in the ED?

First and foremost, when a patient arrives in the ED, the goal is to attempt stabilization and relief of distress for the patient. One approach to these patients, called the *Rapid Palliative Care Assessment,* can be used as part of the secondary survey.

1. Identify/locate any advance directives.
2. Ameliorate symptoms.
3. Speak with family members or care providers who may be able to help determine the extent of resuscitation a patient desires.
4. Determine decision-making capacity (DMC).

If these measures cannot be undertaken in a timely manner and the patient requires life-saving measures such as cardiopulmonary resuscitation (CPR) or intubation, physicians should always err on the side of life-prolonging measures. The patient, family members, and physicians can have further discussions once the patient is stable and in the hospital as to how they would like to proceed. Undesired treatments can always be withdrawn in a comfortable and controlled manner when the patient's wishes and situation are understood more fully.

9. Why is the patient's capacity for decision-making important?

A patient with decisional capacity has the right to consent or refuse treatments in the ED, even those that could be life sustaining. DMC is more than just "alert and oriented," or even a high score on a Mini-Mental State Examination. It means that the patient can manipulate information by communicating the treatment options he or she faces, the likely consequences of those choices, and an explanation of the reason for his or her choices. If the patient lacks DMC, the emergency physician must weigh any written directives and look for the person who can speak for the patient (the designated agent or surrogate). In a patient without DMC, relevant directives (see Table 7-2), or other clear evidence of wishes, the emergency physician will need to stabilize the patient and resuscitate him or her until these become clear.

10. What happens when patients or family members of patients change their goals of care while in the ED?

Patients and family members may revoke or change their wishes or advance directives at any time, and patient goals often evolve during the course of illness. This can be confusing for the emergency physician and requires listening skills and curiosity to understand reasons for the change. Having a conversation about those reasons can help the patient and loved ones, as well as the emergency

Table 7-2. Advance Directives

ADVANCE DIRECTIVE TYPE	DEFINITION	PROS	CONS	COMMENTS
State DNAR, DNR, or no-CPR directives	State-based forms valid in out-of-hospital setting documenting no CPR in the event of stopping breathing or cardiac arrest	To be honored by EMS and all facilities	Does not instruct on how much to intervene on other predeath care, such as dialysis, transfusions, and intubation for respiratory distress only	Order across settings; only effective in cardiopulmonary arrest
Living will	Person directs withdrawal of life-sustaining treatments when he or she lacks capacity and is in terminal or persistent vegetative state	Must be honored unless an MDPOA is given express authority to override	Only in effect when patient lacks DMC and in terminal condition or PVS as determined by two doctors	Very narrow, inflexible
MDPOA	Agent appointed by patient to make decisions when patient lacks capacity, temporarily or permanently	Has broad range of authority to respond to situation at hand according to patient values	Patient must have shared values with agent; only health care decisions	Standard durable power of attorney cannot make medical decisions; patient can fire agent; the most adaptable way for patient wishes to be expressed

POLST-type form	Legal state-based orders (not directives) signed by patient (or representative) and health care provider to determine treatment wishes near the end of life	Orders are to be fully honored by all providers in all settings in the state where enacted	Intended only for patients with chronic, serious, or advanced illness	New form, broader scope than CPR directives; wishes for wide diversity of treatments can be expressed
Proxy decision-maker for health care	Surrogate when patient lacks decisional capacity but hasn't designated an agent (MDPOA); selected according to state's legal rules	Speaks for patient to provide consent or refusal for interventions	Family and friends may disagree, causing significant stress; proxy cannot withhold artificial hydration or nutrition in most instances	Less freedom than MDPOA appointed by patient for medical decisions
Five Wishes	Privately produced legally accepted document executed by patient to express preferences for medical treatment, predeath and postdeath wishes	Offers opportunity to express wide range of perideath wishes	Procedure-based medical wishes; not very useful as guide in clinical care in most situations	Wishes are intended to be followed but may be overridden by MDPOA or proxies

Modified from Ballentine J: Advance directive summary and comparison, Colorado Advance Directives Consortium, 2012. Available at http://coloradoadvancedirectives.com/; accessed 9-28-15.

CPR, Cardiopulmonary resuscitation; *DMC*, decision-making capacity; *DNAR*, do not attempt resuscitation; *DNR*, do not resuscitate; *EMS*, emergency medical services; *MDPOA*, medical durable power of attorney; *POLST*, Physician Orders for Life-Sustaining Treatment, *PVS*, persistent vegetative state.

physician, either support prior wishes or implement modified goals of care moving forward. The emergency physician needs to reassure loved ones that care focusing on comfort is not abandonment and that the ED is prepared to aggressively manage suffering and to honor the patient's goals.

11. What is appropriate vocabulary if you think the patient would benefit from hospice or PC?

End-of-life care is a sensitive topic for many people. Having conversations with patients and family members about PC should be undertaken with the utmost care. Helpful conversation starters could include:

- How can we best help you today?
- Given the situation in which you find yourself, what are you hoping for? or
- I understand you are at a point where your primary goal is quality and comfort in the time you have remaining. What would help you most?

It is important for emergency physicians not to act first without asking for guidance if there is any time to check with the patient and family. Often, even in urgent situations, there are temporizing treatments that can stabilize the patient and allow time for conversation. Phrases that are not helpful and should be avoided include:

- Do you want us to do everything we can?
- There is nothing more that we can do for you? and
- Do you realize you will die if I don't (intubate [or other procedure]) you?

12. Who do I call if I need help managing a patient who needs hospice or PC?

Some hospitals have inpatient PC teams that will provide ED consultation or support for the admitting physician. An ED consultation could result in admission to a less intensive level of service, saving resources and allowing greater access to the patient for family and friends. Hospice requires you to notify the providing organization (the patient should have that contact information) before a potential admission, because the hospice service may be able to arrange other management options, such as increased home service or admission to an in-patient hospice unit. Another important role for the emergency physician is to identify the need for a PC-oriented discussion with the patient not yet aware of palliative services; the emergency physician can "plant the seed" for the patient, loved ones, and admitting team that this is an important consultation that can occur once the patient is admitted.

13. When is it appropriate to withhold resuscitation in a patient who comes to the ED?

American College of Emergency Physicians policy emphasizes the need to take patient preferences into account when deciding whether to initiate or continue resuscitation. Indications for not attempting resuscitation include direct communication from patient, valid "do not attempt resuscitation" (DNAR) orders, an advance directive, or a request from a patient's legal representative to not perform CPR. Unofficial documentation may be taken into consideration also. Neither hospice nor PC services require patients to decline attempted resuscitation.

14. What are the new advance directive forms called *Physician Orders for Life-Sustaining Treatment (POLSTs)*?

POLST-paradigm forms, which are authorized in at least half of the states since their inception in 1993, are direct physician orders (that include resuscitation preferences) to be honored by EMS providers, physicians, and nurses. These forms differ from standard advance directives in that they are orders signed by providers and patients or their representatives. POLST-paradigm forms are used for seriously ill patients with life-limiting diseases. They communicate preferences for a wider range of end-of-life care and function as legally protected orders to be followed in the whole range of inpatient, EMS, and outpatient settings. In the ED, physicians should confirm the continued validity with the patient, if he or she has decisional capacity, or a surrogate if one is available. If that is not possible, valid POLST documents should be honored while the patient's comfort is ensured.

15. Withdrawing or stopping resuscitation feels worse than not starting. Which is more ethical?

Both withdrawing and withholding unwanted treatments can be morally acceptable. In fact, in the ED, complete information about a patient's prior expressed wishes (verbal or written) is often

unknown. Initiating resuscitative measures to stabilize a patient buys time and errs on the side of survival. If definitive information, such as a POLST-paradigm form requesting no resuscitation or patient values expressed by the patient or his or her agent, become clear, withdrawing unwanted interventions is indicated and morally appropriate.

KEY POINTS

- PC is comprehensive treatment of patients with serious illnesses, often in parallel with life-extending or even curative treatments.
- End-of-life care is an important responsibility and core competency of emergency medicine physicians.
- Withdrawing unwanted emergent interventions is ethically superior to withholding treatments when ED information is insufficient.
- Treatment wishes of a patient or his or her loved ones should be honored when they are known and valid, even if these include refusal of burdensome and unwanted life-saving interventions.
- Aggressive symptom management is always appropriate in the hospice patient.

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QUESTIONS

1. Which of the following interventions is least likely to be a palliative intervention in the ED?
   a. Intravenous opioids for pain relief
   b. Chest tube for shortness of breath
   c. Evacuation of an acute intracranial bleed from a metastatic lung cancer
   d. Foley catheter to relieve urinary retention
   The correct answer is *c.*
2. Which of the following best describes the relationship between hospice and PC?
   a. *Hospice* is a designation for patients who are no longer pursuing life-extending treatments, whereas PC can be used in parallel with other treatments.
   b. *Hospice* and *PC* are synonyms for end-of-life care.
   c. Patients receiving PC can be admitted to the hospital, whereas patients receiving hospice care will have their status revoked if they are admitted.
   d. Both patients receiving hospice care and those receiving PC should not receive CPR attempts.
   The correct answer is *a.*
3. Which written advance directive is a formal order rather than a general expression of patient preferences?
   a. Living will
   b. Medical durable power of attorney
   c. *Five Wishes* document
   d. POLST form
   The correct answer is *d.*

CHAPTER 8

HOW TO CRITICALLY REVIEW EMERGENCY MEDICINE LITERATURE

Debra E. Houry, MD, MPH

1. **Can I skip this chapter if I don't plan to do research?**
   No! Reading 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 observe 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 incidence 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?**
   Blinding is a technique in which patients, physicians, researchers, and anyone else involved in the research study are unaware of whether patients are in the experimental group or the 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

Abstract

Being able to critically appraise emergency medicine literature and understanding the strength of research findings is an important skill to determine what new recommendations to incorporate into clinical practice.

Keywords:

evidence based, research, critical appraisal, sensitivity, specificity, p value

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?

- Trained chart abstractors
- Explicit criteria for case selection and exclusion
- Defined study variables
- Standardized abstraction forms for data collection
- Periodic meetings among researchers to resolve abstraction disputes
- Monitored performance of abstractors
- Blinded chart reviewers
- Measures of interrater agreement

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

A *p* value reflects 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 whether 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, such as the following points:

- 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.

		Disease	
		Present	Absent
Exposure/ test results	Positive	A	B
	Negative	C	D

Figure 8-1. Disease-versus-exposure grid.

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. How should I interpret blog posts or other online reviews of clinical medicine?

Although these reviews are often written by emergency medicine clinicians, these online posts do not usually go through peer review. These posts may have useful information but should be interpreted with caution unless clear citations of rigorous research studies are included.

15. 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 (Fig. 8-1); $A/(A + B) \div C/(C + D)$
- 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; $D/(C + D)$

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QUESTIONS

1. Which of the following research methodologies is best for reporting rare diseases?
   a. Randomized controlled trial
   b. Cohort
   c. Case control
   d. Case series
   The correct answer is *d.*
2. What is the most rigorous research methodology of the choices below?
   a. Randomized controlled trial
   b. Cohort
   c. Case control
   d. Case series
   The correct answer is *a.*
3. Which *p* value is not considered significant?
   a. 0.0001
   b. 0.001
   c. 0.01
   d. 0.10
   The correct answer is *d.*

EVIDENCE-BASED RATIONAL USE OF DIAGNOSTIC IMAGING

CHAPTER 9

Ronald R. Townsend, MD, MA, FACR, and
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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 to 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.

1. Ask an answerable question.
2. Search the literature for current best evidence.
3. Appraise the retrieved evidence.
4. Apply the findings.
5. Evaluate your success with the process.

Crucial to this 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 case may make an examination inappropriate, even if its use is generally supported by evidence.

4. When should I consult a radiologist before 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 before 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., American College of Radiology [ACR] Appropriateness Criteria). These range from opinion papers (not evidence-based) to true attempts at rigorous evidence-based analysis.

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.

Abstract

Use of evidence-based decision making, including correlation of evidence with patient-specific considerations, facilitates optimal care and minimizes risks.

Keywords:

evidence-based imaging, computed tomography (CT), ultrasound, magnetic resonance imaging (MRI), appropriateness criteria, radiation exposure

Table 9-1. Adult Effective Doses for Imaging Procedures

EXAMINATION	AVERAGE EFFECTIVE DOSE (mSv)	RANGE OF VALUES REPORTED (mSv)
PA chest radiograph	0.02	0.007-0.05
Pelvis radiograph	0.6	0.2-1.2
Head CT*	2	0.9-4
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

From Mettler FA, Huda W, Yoshizumi TT, et al: Effective doses in radiology and diagnostic nuclear medicine: a catalog. *Radiology* 248:254–263, 2008.

CT, Computed tomography; *IR*, interventional radiology; *mSv*, milli-Sieverts (1 mSv = 100 mrem); *PA*, posteroanterior.

*CT doses may be below the lower ends of these ranges for some examinations performed with recently available dose-reduction technologies.

7. Is radiation exposure from x-rays and computed tomography (CT) dangerous when used for patients in the ED?

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 (*plain films*) have relatively low associated radiation, so there is less risk. Some examples of average adult effective dose for some imaging procedures are given in Table 9-1. Newer CT scanning technology may facilitate obtaining diagnostic images with significantly 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 are at highest risk. 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 to years. Again, the benefit may substantially 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 often comes 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? Can she be effectively managed with ultrasound, or can she be managed with no additional studies?

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, Is there an evidence-based imaging approach that can address the patient's need without the 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 $5000. 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 it could be tragic if it was not.

11. Should a cervical spine CT be obtained in all patients who are victims of trauma?

No. Many victims of trauma have virtually no likelihood of clinically significant cervical spine injury. An evidence-based approach would be to use one of the published clinical prediction rules to determine who should be imaged. The National Emergency X-Radiography Utilization Study (NEXUS) criteria and Canadian C-spine rule define patients in whom no imaging is necessary (see Chapter 83). There is no clear evidence to favor one of these rules over the other, and there are 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 radiography?

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 and 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 greater than 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 patients with chest pain 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 29).

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 dilation. However, many patients with prior CT documentation of urinary stone disease come 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 often come 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 used 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 hindfoot and midfoot and intraarticular 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.

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.)

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

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 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 more useful in the effort to avoid radiation exposure in pregnant or other high-risk patients, including children. MRI may be useful to diagnose appendicitis in pregnant patients, but evidence regarding its accuracy is

limited. 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 dilation 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 is 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., patient is obese), 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 the best initial evaluation being 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 the cause 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 undergo CT and be managed based on results of the CT whether the radiographs are positive or negative). CT of the abdomen and pelvis with IV (but not oral or rectal) contrast will 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.

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 appendagitis) can be diagnosed with CT. Compression ultrasound can be used for diagnosis of diverticulitis, but it appears to be less accurate than CT.

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 diagnosing testicular torsion and distinguishing torsion from other pathologies. It should be performed emergently to optimize the chance of testicular salvage if torsion is present.

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 posttrauma (see Chapter 84). 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.

28. How about a head CT for trauma patients who are receiving anticoagulants?

Data indicate that trauma patients who are receiving anticoagulants are at greater risk to develop a traumatic brain injury and that 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 such a patient should be very low. These patients may also require closer monitoring and potential repeat head CTs 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 from 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.

30. Should imaging be repeated when a patient is transferred from another institution to my care?

This is usually unnecessary and will contribute to increased costs, excessive radiation exposure, and delays in care. Electronic transmission of images may facilitate image review at the receiving institution, even before the patient arrives. The presence or absence of need for any additional imaging to direct patient management can be determined after that review and examination of the patient. The concept that prior patient evaluation (imaging or otherwise) should be reviewed before embarking on additional diagnostic testing is crucial to avoiding waste in medicine.

WEBSITES

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QUESTIONS

1. Regarding radiation exposure related to imaging of patients in the ED:
   a. The large number of portable chest radiographs obtained in most EDs accounts for the largest part of patient radiation exposure.
   b. Children are relatively resistant to the effects of radiation because of enhanced DNA repair capabilities.
   c. MRI causes the highest radiation dose of all exams.
   d. CT causes most of the radiation exposure.
   The correct answer is *d.*
2. Regarding implementation of evidence-based guidelines for imaging patients in the ED:
   a. It is best to have a single approach to imaging that applies to all patients.
   b. Increased use of ultrasound in many guidelines has resulted in increased patient radiation exposure.
   c. Patient-specific factors including age, gender, and prior evaluation should be considered when applying guidelines.
   d. Most guidelines are developed by organizations with bias and should be ignored.
   The correct answer is *c.*
3. For trauma patients brought by ambulance to the ED:
   a. It is most appropriate to obtain a total-body CT in all patients to avoid missing injuries.
   b. Decisions about imaging should be based on patient specific history, symptoms, and signs.
   c. Patients who are pregnant should never have CT because of risk of radiation exposure to the fetus.
   d. MRI is safer, quicker, and more accessible and should be used instead of CT for initial brain imaging in all patients.
   The correct answer is *b.*

CHAPTER 10

EMTALA, THE JOINT COMMISSION, AND HIPAA

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EMERGENCY MEDICAL TREATMENT AND LABOR ACT

1. What is the Emergency Medical Treatment and Labor Act (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 EMC 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 *EMC*.

An EMC includes 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 described as a fundamental right. For much of the twentieth 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-twentieth 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 money 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 $50,000 per violation (fewer than 100 beds may be fined up to $25,000). However, there

Abstract

The chapter describes the background for the Emergency Medical Treatment and Labor Act (EMTALA), Health Insurance Portability and Accountability Act (HIPAA) and The Joint Commission regulations that impact emergency medicine providers. Key regulatory concepts are emphasized to focus attention on issues that present a compliance risk in emergency medicine settings. The authors also highlight some practical risk management pearls to avoid escalation of potential risk management concerns.

Keywords:

Emergency Medical Treatment and Labor Act (EMTALA), Joint Commission, risk management, Health Insurance Portability and Accountability Act (HIPAA), medical screening examination (MSE), sentinel event, standards, malpractice

are a few provisions that 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.

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 with similar complaints and circumstances.

6. **Does EMTALA apply when a patient in need comes to any part of a hospital's campus, even if it is not an ED?**
Yes. If an individual seeks care 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 EMC or if a prudent layperson would believe that the individual is suffering from an EMC. The term *hospital property* means the entire main hospital campus; this includes the parking lot, sidewalk, driveway, hospital departments, and 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 designated 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. More recently, freestanding EDs have grown in number and are further categorized as hospital outpatient departments (HOPDs) or independent freestanding emergency departments (IFSEDs). HOPDs are owned and operated by medical centers or hospital systems, which means they operate under the same Centers for Medicare and Medicaid Services (CMS) rules and regulations as the hospital or medical center. IFSEDs are not recognized as EDs by Medicare and therefore do not have the same federal regulations.

8. **Is a hospital obligated under EMTALA to medically screen and stabilize any patient seeking care in an ambulance it owns and operates?**
No, as long as the hospital-owned ambulance operates under community-wide emergency medical services (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 describe a proper MSE?**
An MSE is not an isolated event. It is an ongoing process, conducted by qualified medical personnel (QMP), 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 prioritize the order in which individuals will be seen by QMP. The MSE must be appropriate to the individual's presenting signs and symptoms, as well as the capability and capacity of the hospital. The complex reality is that an adequate MSE can range from a quick history and physical to confirm the presence of an upper respiratory tract infection to a complex workup 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 QMP must perform the MSE. A nurse or midlevel 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 QMP must have in their personnel files a job description for this role, qualifications, competencies, and a formal designation to perform an MSE.

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 EMC has been resolved. Once the EMC is resolved, the individual may be discharged home with follow-up care instructions, admitted for ongoing care, or transferred to another facility. Patients in need of psychiatric care 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 capacity to make medical decisions. 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 before 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 deal with patient transfers and the transferring facilities in a simple manner. 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 is OK to transfer a patient?

If a patient is deemed stable (i.e., an EMC 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 patients in unstable conditions. Patients who are unstable 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.
- A patient in an unstable condition 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 patient who has multiple traumatic injuries comes 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 comes 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 a patient who is unstable.

- 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 must 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 EMC.
- The receiving facility has been provided with all medical records related to the patient's emergency condition.
- The transfer is conducted with qualified personnel and proper transportation, including the use of necessary and medically appropriate life-support measures.

16. Can an on-call consultant refuse to see a patient who is unstable?

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 by-laws), 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 patient to a facility with the capability of treating the EMC. 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 on the transfer form the name and address of the consultant who failed to treat the patient.

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 EMC after the initial MSE. A hospital must have written policies and procedures that clearly delineate the responsibilities of on-call physicians to respond, examine, and treat patients with an EMC.

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.

KEY POINTS: REQUIREMENTS WHEN TRANSFERRING A PATIENT WHO IS UNSTABLE

1. Physician certifies that benefits of transfer outweigh the risks.
2. The transfer is coordinated, and risks are minimized before transfer.
3. The receiving facility has accepted the patient and has the capacity and capability to treat him or her.
4. Medical records related to the EMC are copied and sent with the patient (including diagnostic imaging).
5. The transfer is accomplished with qualified personnel and appropriate equipment.

THE JOINT COMMISSION

20. What is The Joint Commission?

The Joint Commission is an independent, not-for-profit organization that evaluates and accredits health care organizations and programs. The origins of The Joint Commission 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 United States.

In 1965, Congress passed the Social Security Amendments that included a provision that empowered JCAH by linking hospital eligibility to participate in the Medicare program with accreditation by JCAH. The CMS deems qualified, private organizations with the authority to evaluate health care organizations' compliance with Medicare regulations in addition to their own established standards. Over the years, the standards changed to represent optimal achievable levels of quality and safety, 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 Joint Commission* on January 1, 2007. The mission of The Joint Commission 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 Joint Commission evaluates and accredits more than 20,500 health care organizations and programs in the United States.

21. What are the standards and performance measurements that The Joint Commission requires?

The Joint Commission 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* includes standards and performance in such areas as emergency management, patient care, medication management, infection prevention, the record of care, leadership, 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 hand-off 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 an important focus of the accreditation process.

22. How is compliance with the standards evaluated and enforced?

The Joint Commission conducts unannounced, on-site surveys that occur 18 to 36 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 backward 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 survey.

To earn and maintain Joint Commission accreditation, a hospital must maintain continuous compliance with Joint Commission 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. 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 Joint Commission 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 a sentinel event is to conduct a timely, credible, and thorough root cause analysis (RCA). An RCA is a process determined by the organization that facilitates the evaluation and identification of the fundamental reason for variation in performance that lead to the occurrence of a sentinel event. The outcome of an RCA should be an action plan designed to implement improvements and reduce risk.

24. How do The Joint Commission 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, procedural sedation, monitoring restraint use, patient rights, staffing, staff competency, ED security, health care literacy, and standardized communication are all areas of focus for The Joint Commission in the ED. 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 has been a leadership standard that focuses on patient flow and overcrowding in the ED. The Joint Commission requires that hospital leadership manage patient flow throughout the hospital to minimize ED overcrowding and minimize delays in care delivery. Effective January 2014, hospitals are required to set goals for managing patients who are boarded in the ED. 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

25. What is Health Insurance Portability and Accountability Act (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 HIPAA of 1996. Congress had 3 years to enact privacy legislation, and when this time passed, HHS developed the Privacy Rule, which was published in late 2000. The idea of the Privacy Rule is to have patients' medical records protected from anyone who does not 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, "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 number, 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 without patient consent to other care providers as necessary to deliver medical or mental health treatment.

There are other circumstances under which it is appropriate to disclose PHI, but a hospital HIPAA authority should be consulted before disclosure. The 12 PHI exceptions allowed or required are:

1. Public health activities
2. Requirement by law
3. Victims of abuse, neglect, or domestic violence
4. Health oversight activities
5. Judicial and administrative proceedings
6. Law enforcement purposes, which are limited to the following six specified conditions:
7. Decedents
8. Organ donation
9. Correctional facilities
10. Serious threat to health or safety
11. Essential government functions
12. Workers' compensation

31. How is the statute enforced, and what are the penalties for a HIPAA violation?

The OCR 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 electronic tracking boards and computer screens
- 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; accessed 1-30-15.
Journey through the History of the Joint Commission: www.jointcommission.org/AboutUs/joint_commission_history.htm
Privacy Rule: www.hhs.gov/ocr/privacy/; accessed 1-30-15.

RISK MANAGEMENT

33. What is risk management?

Risk management is the effort 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).

34. Why are emergency physicians at high risk for malpractice lawsuits?

The primary reasons are the lack of an established physician-patient relationship and lack of communication. 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. Another 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.

35. 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 coming to the ED and requesting care. The EMTALA statute mandates an MSE on all patients coming to the ED.
- 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.

36. Give some examples of patients who place a provider at high risk for a malpractice suit.

- The hostile or belligerent patient. These patients are difficult to deal with and sometimes get less than complete or careless 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 condition.
4. Review nursing records for congruency and document if there is a discrepancy.
5. Communicate clearly with patients, families, and staff.

37. 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

38. What is the most common error emergency physicians make with regard to their malpractice insurance policy?

The most common error is 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).

39. 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 $\times$ 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 presenting condition. 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 judgment. The best insurance is careful and complete patient discharge instructions that include when and where to seek follow-up care 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.

40. 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 review on radiology rereads of radiographs
- Inadequate follow-up review of cardiology rereads of electrocardiograms (ECGs)
- Inadequate follow-up review of delayed clinical laboratory results (e.g., cultures)
- Poor availability of previous medical records
- Inadequate handling of patient complaints (your chance to possibly head off a malpractice suit)
- Inadequate physician and ED staffing patterns (leading to prolonged patient waits and subsequent patient hostility)

41. 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 meet the following conditions:

- Have medical decision-making capacity
- 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.

42. 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).

43. 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 the simple statement, Treat every patient as you would want your mother treated. This, of course, assumes you love your mother.

44. How can writing admission orders for 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.

45. 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 as follows:

- 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.

46. 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.

47. 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 them or if he or she chooses not to follow these guidelines without carefully documenting the reasons for not doing so.

48. 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 because of differences in the average indemnity but was a result of 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.

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QUESTIONS

1. All of the following must be proved for successful malpractice litigation except:
   a. Damages
   b. Duty to treat
   c. Proximate cause
   d. EMTALA violation
   The correct answer is *d.*
2. Which of the following is not covered by EMTALA?
   a. Independent freestanding ED
   b. Hospital ED
   c. Hospital outpatient urgent-care clinic
   d. Freestanding hospital ED
   The correct answer is *a.*
3. All of the following may be authorized to perform an MSE except:
   a. Physician
   b. Emergency medical technician
   c. ED nurse
   d. ED nurse practitioner
   The correct answer is *b.*

CHAPTER 11

EMERGENCY MEDICINE OBSERVATION MEDICINE

Jennifer L. Wiler, MD, MBA

1. What is observation care?

Observation is a family of billing codes, a location, and a service. The family of billing codes describes physician services that the Center for Medicare and Medicare Services (CMS) describes as follows:

> *Observation care is a well-defined set of specific, clinically appropriate services, which include ongoing short-term treatment, assessment, and reassessment before a decision can be made regarding whether patients will require further treatment as hospital inpatients or if they are able to be discharged from the hospital. Observation services are commonly ordered for patients who present to the ED and who then require a significant period of treatment or monitoring in order to make a decision concerning their admission or discharge.*[1]

Hospital observation services require documentation of medical necessity and a physician order for the service to be provided.[2] Observation services can occur in a traditional ED treatment space or bed, or patients can be cohorted. The name of the location is based on the conditions treated or institutional preference (e.g., ED observation unit [EDOU], clinical decision unit [CDU], short-stay unit, chest pain unit, or rapid diagnosis and treatment unit). Although these units are sometimes called *23-hour units,* the average length of stay in an EDOU is 15 hours.[3]

2. What type of patients are appropriate for observation services?

Observation units are appropriate for patients who require therapeutic intensity to prevent an inpatient hospital admission (e.g., acute asthma exacerbation), or for patients for whom there remains a diagnostic uncertainly and more evaluation is needed to determine if inpatient services are warranted.

3. How common are EDOUs?

The number of EDOUs has grown over the last decade.[4] An estimated 19% of U.S. hospitals reported having an EDOUs, with another 12% planning a unit according to a 2003 survey.[5] A subsequent analysis of 2007 National Hospital Ambulatory Medical Care Survey data indicated that the percent of U.S. hospitals with an EDOU had increased to 36%, with more than half administratively managed by the ED.[6] Among academic centers with an emergency medicine residency program, 36% report having an EDOU, with another 45% planning a unit.[7] Internationally, emergency observation services have been reported in several countries and continents, including Canada, Britain, throughout Europe, Australia, India, China, Singapore, and South America.

4. What are some typical diagnoses that are appropriate for ED observation?

ED diagnoses may include chest pain to be evaluated for acute coronary ischemia; asthma/chronic obstructive pulmonary disease (COPD)/reactive airway disease exacerbation; syncope; transient ischemic attack (TIA); deep vein thrombosis; acute onset atrial fibrillation; abdominal pain; psychiatric conditions; acute congestive heart failure; head injury; uncomplicated pyelonephritis; cellulitis/soft-tissue infections; upper gastrointestinal (GI) bleeding; abdominal trauma; toxicology/drug overdose; pneumonia; dehydration/vomiting/diarrhea; social services management; renal colic/kidney stones; extremity pain/injury and intractable back pain; vertigo/ear, nose, and throat (ENT) problems; blood product transfusions; alcohol intoxication; and intractable headache.

5. Can EDOUs provide services to pediatric patients?

Nearly one third of all ED visits are pediatric patients,[8] of which an estimated 4% of patients are admitted to observation units nationally.

Abstract

Emergency medicine observation units are rapidly becoming an integral part of EDs, as well as the safe and cost-effective practice of emergency medicine. This chapter discusses the appropriate use of these units and the billing implications for providers and patients.

Keywords:

emergency medicine observation, observation medicine, observation unit, Medicare Parts A, B, and D

6. What are some common pediatric conditions of patients admitted to an EDOU?
Pediatric conditions include asthma/reactive airway disease, dehydration, gastroenteritis, pneumonia, abdominal pain, seizures, fever, bronchiolitis, croup, poisonings/ingestions and trauma.

7. How does care provided in an EDOU compare with inpatient care for the same conditions?
A number of prospective randomized control studies have shown that patients with chest pain, TIA, syncope, asthma, and atrial fibrillation who were managed in an EDOU had shorter lengths of stay, lower costs, comparable or better clinical outcomes, and improved patient satisfaction compared with similar patients admitted to an inpatient hospital unit.[9,10,11]

8. I have heard that time is an important factor for observation. Why is it important and how is it calculated?
Medicare and some insurance carriers (payers) require that a patient be admitted to observation status for at least 8 hours in order for the provider to be reimbursed for services. Observation time begins when an "admit to observation" order is written and ends with the time of final patient disposition.

9. Does the time of ED services count toward observation time?
No. In most circumstances, the ED group is providing both the emergency medicine and observation care services. Most payers limit payment to only one type of service emergency medicine or observation care.

10. Does observation care have to occur in an observation unit?
No. Observation services can be performed and billed while the patient is in a typical ED bed. However, the best practice endorsed by the American College of Emergency Physicians (ACEP) is a dedicated ED observation area with committed staff and resources.[12]

11. What is the advantage of ED observation medicine to hospitals?
Studies have shown that EDOUs can provide high quality care that is more efficient financially then inpatient care.[13] One study also found that opening a EDOU decreased ambulance diversion and patients who left without being seen.[14] By decreasing the number of short-stay admissions that are placed into hospital beds, EDOUs also help to increase the acuity case mix of hospitals, assuming there is sustained demand for hospital and ED services.

12. What are the different types of observation billing codes?
There are currently three sets of observation current procedural terminology (CPT) codes: (1) admission to and discharge from observation status occurring on the same calendar day (CPT 99234-99236), (2) admission to the EDOU on one calendar day and discharge from observation on the next calendar day (CPT 99218-99220, and discharge code 99217), (3) observation care services provided on dates other than the initial day or discharge date (CPT 99224-99226). More detail is available from ACEP at https://ctxapps.uch.edu/cvpn/aHR0cDovLzEyNy4wLjAuMQ/vpns/portal/homepage.html.

13. What is the difference from a billing and reimbursement perspective between an emergency medicine visit and an observation visit?
Relative value units (RVUs) are used for physician reimbursement. Table 11-1 shows a comparison of 2014 RVUs of ED evaluation and management services with observation care services.

14. What is the difference between observation and inpatient services?
For Medicare patients the difference is one of cost. Outpatient services, including ED and observation services, require more out-of-pocket costs be covered by beneficiaries (covered only by Medicare Part B) versus inpatient admission (covered by Medicare Parts A and B). It is the intensity of service and the anticipated time (i.e., length of stay) that drive the decision about inpatient versus observation status. Per a note from the CMS to beneficiaries

The decision for inpatient hospital admission is a complex medical decision based on your doctor's judgment and your need for medically necessary hospital care. An inpatient admission is generally appropriate when you're expected to need 2 or more midnights of medically necessary hospital care, but your doctor must order such admission and the hospital must formally admit you in order for you to become an inpatient.[15]

Table 11-1. Comparison of Emergency Medicine Evaluation and Management and Observation Care Billing Codes

Emergency Department Codes		*Initial Observation Status Service Codes*		*Subsequent Observation Status*		*Observation Admission and Discharge Codes*	
CPT CODE	**RVUs**	**CPT CODE**	**RVUs**	**CPT CODE**	**RVUs**	**CPT CODE**	**RVUs**
99283	1.73	99218	2.78	99224	1.12	99234	3.79
99284	3.30	99219	3.80	99225	2.03	99235	4.74
99285	4.85	99220	5.20	99226	2.93	99236	6.12
				99217	2.03		

CPT, Current procedural terminology; *RVUs*, relative value units.

15. What is the difference between ED and non-ED (hospital-based) observation services?

From a billing perspective there is no significant difference; the same family of CPT codes are used (see Question 12). However, EDOUs ideally care for patients who have a high likelihood of discharge (approximately 70%) within 24 hours. These patients tend to be those without significant comorbid conditions and those who have a complaint or condition that can follow a straightforward care pathway.

16. Can patients be admitted to observation for only 24 hours?

No. CPT codes (99224-6) describe services that extend longer than 1 calendar day. However, according to CMS.

In the majority of cases, the decision whether to discharge a patient from the hospital following resolution of the reason for the observation care or to admit the patient as an inpatient can be made in less than 48 hours, usually in less than 24 hours. In only rare and exceptional cases do reasonable and necessary outpatient observation services span more than 48 hours.

17. What is required to bill for observation services?

To bill for observation services the patient must require observation to determine whether inpatient hospitalization is required. In addition there must be a medical observation record containing a timed and dated physician's admit-to-observation order. The observation record should describe the patient care delivered while the patient was in the observation unit with physician and nurse progress notes. This record must be supplemental to any ED encounter note.

18. I heard that there is some recent controversy about Medicare patients admitted for observation care. What is the issue?

Because observation status is considered an outpatient status, these charges are not covered by Medicare Part A (which covers hospital charges). Instead, these services are billed under Medicare Part B, which requires beneficiaries to pay 20% of the cost (with no cap on the total expenditures) and also requires that they pay out of pocket for medications received during their stay. Beneficiaries who have Medicare Part D prescription drug coverage may be reimbursed for their medications depending on their type of Medicare coverage.

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QUESTIONS

1. What is the minimum number of hours a patient must be placed in observation status for Medicare to reimburse for the services?
   a. 12
   b. 6
   c. 10
   d. 8
   The correct answer is *d.*
2. Which of the following diagnoses have been shown to be ideal for ED observation care?
   a. Sepsis
   b. End-stage renal disease
   c. Asthma
   d. Compartment syndrome
   The correct answer is *c.*
3. Can a physician bill for both ED and observation services for the same patient during the same encounter?
   a. Yes
   b. No
   c. Depends on how long the decision to admit to observation took
   d. Depends on what the diagnosis for the encounter is
   The correct answer is *b.*

CHAPTER 12

PERFORMANCE EVALUATION AND IMPROVEMENT IN EMERGENCY MEDICINE

Stephen V. Cantrill, MD, FACEP

1. **Why should I care about my performance in my delivery of health care?**
Physicians have the obvious moral obligation to provide high quality care at a reasonable cost, resulting in the best possible outcome for their patients. Unfortunately, there is a paucity of data supporting the routine achievement of this laudable goal, in spite of ever-increasing expenditures on health care. This has led to an increasing interest in performance measurement and reporting by health care institutions, governmental and nongovernmental agencies, medical societies, and certifying boards. This has led to the development of the National Quality Strategy (NQS), which is a guiding document for federal efforts to encourage and promote a high-value and high quality health care system.

2. **What are the broad aims specified in the NQS?**
   - Better care through improved quality
   - Improved health of people and communities
   - More affordable care

3. **What are the priorities outlined by the NQS?**
   - Making care safer by reducing harm caused in the delivery of care
   - Ensuring that each person and family are engaged as partners in their care
   - Promoting effective communication and coordination of care
   - Promoting the most effective prevention and treatment practices for the leading causes of mortality, starting with cardiovascular disease
   - Working with communities to promote wide use of best practices to enable healthy living
   - Making quality care more affordable for individuals, families, employers, and governments by developing and spreading new health care delivery models

4. **How will this impact me and my practice?**
The Center for Medicare and Medicaid Services (CMS) has stated that it will support the NQS through quality measurement and reporting programs, payment incentives, and rule making that will stress effectiveness of care, coordination of care, safety of care, improved patient experience, healthy communities, and improved affordability of care (which CMS refers to as *efficiency*). These efforts will include performance measures targeted for emergency care providers and their institutions with a direct impact on reimbursement.

5. **Aside from CMS, who are the other major players in performance measures and performance measurement?**
There are several. The National Quality Forum (NQF) is a nonprofit, nongovernmental organization created in 1999 by the federal government, along with public and private-sector leaders, to encourage performance improvement and endorse national consensus measures for evaluating and publicly reporting provider and institutional performance. CMS is obligated to promulgate any performance measures approved by NQF. Another important player is the American Medical Association (AMA)'s Physician Consortium for Performance Improvement (PCPI), whose mission it is to align patient-centered care, performance measurement, and quality improvement. PCPI has historically developed more than 250 clinical performance measures covering all specialties. These have been used in national reporting and quality improvement programs. The Agency for Healthcare Research and Quality (AHRQ) is also active in this area. It is an agency within the Department of

Abstract

Ongoing concerns about the cost of health care, the quality of provided care, and the overall health of our population has stimulated interest in evaluating our provision of health care with the goal of reducing harm and improving care coordination and efficiency. Several aspects and implications of these efforts are presented.

Keywords:

performance, quality, measures, Center for Medicare and Medicaid Services (CMS), Physician Quality Reporting System (PQRS), Choosing Wisely

Health and Human Services that is dedicated to research in improving the safety and quality of care. The American Board of Medical Specialties (ABMS) and most specialty boards, including the American Board of Emergency Medicine (ABEM), are also involved in this area from the point of view of maintenance of physician certification (MOC).

6. Please elaborate on the impact on reimbursement.

CMS has two programs that can specifically impact emergency medicine reimbursement. The first, the Physician Quality Reporting System (PQRS), requires reporting of performance measures by physicians. If the reporting requirements are met for the 2014 performance year, the provider will receive a 0.5% pay incentive for his or her Medicare patients in 2015. Failure to meet the reporting requirements will result in a 2% payment penalty for Medicare reimbursement in 2016. PQRS data are reported publicly. The current plan is for the PQRS incentive payment to be discontinued in the 2015 performance, affecting reimbursement in 2017. The second related program is the Value-Based Payment Modifier (VBPM) program. This was mandated under the Patient Protection and Affordable Care Act and specifies that by 2015, CMS should begin calculating physician payments based upon both cost and quality data from an individual's practice. This will initially apply only to physician groups. The current proposal for failure to satisfy VBPM requirements would result in a 2% payment penalty for 2014 performance (applied to 2016 reimbursement) and a 4% payment penalty for 2015 performance (applied to 2017 reimbursement). This could result in up to a 6% payment penalty per physician, or an average estimate of $3336 per emergency physician in 2015 and beyond. The PQRS will be replaced in 2018 by the Merit-Based Incentive Payment System (MIPS), which was established under the Medicare Access and CHIP Reauthorization Act of 2015 (MACRA).

7. What are some examples of current emergency medicine provider performance measures that are in effect now?

- 12-lead ECG performed for nontraumatic chest pain
- Ultrasound for determination of pregnancy location for pregnant patients with abdominal pain
- RhoGAM for Rh-negative pregnant women at risk of fetal blood exposure

8. Are there other performance measures related to emergency medicine?

Yes, there are institutional performance measures in emergency medicine. Some examples include door-to-provider time, left-without-being-seen rate, and median time from ED arrival to ED departure for discharged ED patients. Many of the specifications for these metrics have been developed by the Emergency Department Benchmarking Alliance.

9. Who can specify these performance measures?

Any group can submit measures to NQF, whose approval is required for the performance measure to be put into effect nationally in federal programs. CMS can, however, approve measures on its own if there are no NQF-endorsed measures for a specific topic. NQF also determines, through data analyses by the measure developers, when a specific measure should be retired if there is no longer a provider performance gap in the area covered by the measure. Private payers are free to use any measures they would like.

10. What are some of the problems with performance measure development?

Performance measure creation is a surprisingly difficult process. Several issues have come to light, including lack of good evidence for the measure (e.g., blood cultures in all admitted pneumonia patients), limitations of using only claims-based data when much information is only in the corpus of the patient's record, and assuming that a performance measure for one practice location (e.g., a private office) would be valid in another (e.g., an ED).

11. Are there any significant changes planned by CMS in dealing with performance measures?

Yes, CMS plans to eventually move away from claims-based reporting of measures and allow reporting only via a qualified clinical data registry. This will have significant operational implications for all practitioners of emergency medicine.

12. How is cost of care playing into performance measures?

There are several measures under consideration or development that deal with the efficiency domain. That is, are we, as physicians, ordering ancillary studies that are not indicated? Many of these performance measures address evidenced-based criteria for computed tomography (CT) or

magnetic resonance imaging (MRI). Other important aspects of the efficiency domain are total cost per beneficiary and Medicare spending per beneficiary, which are impacting hospitals' reimbursement.

13. What other developments have there been in the area of addressing rising health care costs?

One development has been the establishment of the *Choosing Wisely* campaign. This is an attempt to address the issues of low-yield testing and therapeutics. It was started in 2012 by the American Board of Internal Medicine Foundation and aims to publicize and engage clinicians and patients to discuss these issues of testing and treatment to determine what is appropriate care for each patient. As of July 2014, 55 specialty societies, including the American College of Emergency Physicians (ACEP), have subscribed to this program.

14. What are the *Choosing Wisely* recommendations from ACEP?

ACEP's 10 recommendations for the *Choosing Wisely* campaign are (www.ChoosingWisely.org/clinician-lists):

1. Avoid CT of the head in ED patients with minor head injury who are at low risk based on validated decision rules. Minor head injury is a common reason for visiting an ED. The majority of minor head injuries do not lead to skull fractures or bleeding in the brain, which would need to be diagnosed by a CT scan.
2. Avoid placing indwelling urinary catheters in the ED for either urine output monitoring in stable patients who can urinate on their own, or for patient or staff convenience. These catheters are used to assist when patients cannot urinate, to monitor how much they urinate, or for patient comfort.
3. Do not delay available palliative and hospice care services in the ED for patients likely to benefit. This is medical care that provides comfort and relief for patients who have chronic or incurable diseases. Early referral from the ED to hospice or palliative care services can benefit patients, resulting in both improved quality and quantity of life.
4. Avoid antibiotics and wound cultures in ED patients with uncomplicated skin and soft-tissue abscesses after successful incision and drainage and with adequate medical follow-up care. Skin and soft-tissue infections are a common reason for visiting an ED. Some infections, called *abscesses,* become walled off under the skin. Opening and draining the abscess is the appropriate treatment; antibiotics offer no benefit.
5. Avoid instituting intravenous (IV) fluids before doing a trial of oral hydration in uncomplicated ED cases of mild to moderate dehydration in children. Many patients who come to the ED with dehydration require fluids. To avoid pain and potential complications, it is preferable to give these fluids by mouth instead of the use of an IV.
6. Avoid CT of the head in asymptomatic adult patients in the ED with syncope, insignificant trauma, and a normal neurologic evaluation. Syncope (passing out or fainting) or near syncope (lightheadedness or almost passing out) is a common reason for visiting an ED, and most of those visits are not serious. Many tests may be ordered to identify the cause of the problem. However, these tests should not be routinely ordered, and the decision to order them should be guided by information obtained from the patient's history or physical examination.
7. Avoid CT pulmonary angiography in ED patients with a low pretest probability of pulmonary embolism and either a negative pulmonary embolism rule-out criteria (PERC) result or a negative D-dimer. Advances in medical technology have increased the ability to diagnose even small blood clots in the lung. Now, the most commonly used test is known as a *CT pulmonary angiogram (CTPA).* However, disadvantages of the CTPA include patient exposure to radiation, the use of dye in the veins that can damage kidneys, and high cost.
8. Avoid lumbar spine imaging in the ED for adults with atraumatic back pain unless the patient has severe or progressive neurologic deficits or is suspected of having a serious underlying condition, such as vertebral infection or cancer with bony metastasis. Low back pain without trauma is a common presenting complaint in the ED. Most of the time, such pain is caused by conditions such as a muscle strain or a bulging disc that cannot be identified on plain film or CT scan.
9. Avoid prescribing antibiotics in the ED for uncomplicated sinusitis. Sinusitis is a common reason for patients to visit the ED. Most patients with acute sinusitis do not require antibiotic treatment,

because 98% of acute sinusitis cases are caused by a viral infection and resolve in 10 to 14 days without treatment.

10. Avoid ordering CT of the abdomen and pelvis in young, otherwise healthy ED patients with known history of ureterolithiasis who have symptoms consistent with uncomplicated kidney stones. Many patients in the ED who are younger than 50 years and who have symptoms of recurrent kidney stones do not need a CT scan unless these symptoms persist or worsen, there is a fever, or there is a history of severe obstruction with previous stones.

15. Have other lists been developed that I should think about?

Yes, a modified Delphi consensus technique was used in surveying 283 emergency medicine providers from six EDs to develop a list of additional situations where specific studies provided little value.

- Do not order CT of the cervical spine after trauma for patients who do not meet the National Emergency X-ray Utilization Study (NEXUS) low-risk criteria or the Canadian C-spine rule.
- Do not order coagulation studies for patients without hemorrhage or suspected coagulopathy (e.g., with anticoagulation therapy, clinical coagulopathy).

16. Are there *Choosing Wisely* recommendations made by other specialties that might apply to emergency medicine?

Yes, all of these can be viewed at the *Choosing Wisely* website. Here are a few of interest:

- Do not order sinus CT or indiscriminately prescribe antibiotics for uncomplicated acute rhinosinusitis. (American Academy of Allergy, Asthma, and Immunology)
- Do not prescribe antibiotics for otitis media in children ages 2 to 12 years with nonsevere symptoms where the observation option is reasonable. (American Academy of Family Physicians)
- Do not order chest radiographs in children with uncomplicated asthma or bronchiolitis. (Society of Hospital Medicine–Pediatric Hospital Medicine)

17. These are recommendations for use of specific studies. Is there an overarching philosophy that I can use to guide me in appropriately ordering diagnostic studies?

Yes, before ordering any study, ask yourself, "How useful will this test be in establishing a diagnosis or assisting in treatment?" Also, try to avoid ordering diagnostic studies for the wrong reasons, such as intellectual curiosity, defensive medicine, unrealistic patient expectations, or peer (consultant) pressure.

18. Should I order tests to "cover" myself?

No, 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 and physical examination.

19. How much can be saved with no compromise in patient care?

In a multicenter study of 20 hospital EDs, both teaching and nonteaching, an ancillary study ordering educational program was developed to address the appropriate use of diagnostic studies. Seventeen tests or groups of tests or studies were targeted. A 12.5% decrease in targeted test charges was shown. No decrease in the perceived quality of care could be shown. The costs of medical testing in the ED can be contained by careful, thoughtful ordering without sacrificing patient care.

KEY POINTS

1. Failure to participate in the CMS, PQRS, and VBPM programs could potentially result in a 4% CMS payment penalty in 2016 and 6% in 2017.
2. The *Choosing Wisely* campaign, in which emergency medicine participates, is an attempt to address the issues of low-yield testing and therapeutics.
3. CMS, in its quality and performance reporting programs, is taking guidance from the NQS.

WEBSITES

CMS Physician Quality Reporting System: www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/PQRS/index.html?redirect=/PQRS/; accessed 3-19-15.
CMS Value-Based Payment Modifier: www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/PhysicianFeedbackProgram/ValueBasedPaymentModifier.html; accessed 3-19-15.
Choosing Wisely: www.choosingwisely.org; accessed 3-19-15.
ACEP, quality related: www.acep.org/quality; accessed 3-19-15.

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QUESTIONS

1. What document outlines the federal government's overarching approach to health care quality?
   a. The NQS (National Quality Strategy)
   b. The PQRS (Physician Quality Reporting System)
   c. The VBPM (Value-Based Payment Modifier System)
   d. The AHRQ (Agency for Healthcare Research and Quality)
   The correct answer is *c*.
2. What is the proposed maximum CMS physician decrease in reimbursement for 2017?
   a. 0.5%
   b. 4%
   c. 6%
   d. 8%
   The correct answer is *c*.
3. Which of the following is not an ACEP entry in the *Choosing Wisely* campaign?
   a. Head CT in minor head trauma
   b. Sinus CT in uncomplicated acute rhinosinusitis
   c. IV fluids in mildly dehydrated children
   d. Use of palliative and hospice services
   The correct answer is *b*.

ALTERED MENTAL STATUS AND COMA

Kenneth C. Jackimczyk, Jr., MD, FACEP

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

Coma is 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. Fewer than 30% of patients have a structural cause for coma.

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

TIPS and vowels; that is, TIPS and AEIOU.

TIPS
Trauma, **t**emperature
Infection (CNS and systemic)
Psychiatric
Space-occupying lesions, **s**troke, **s**ubarachnoid hemorrhage, **s**hock

VOWELS
Alcohol and other drugs
Epilepsy, **e**lectrolytes, **e**ncephalopathy
Insulin (diabetes, diabetic ketoacidosis [DKA])
Oxygen (lack of), **o**piates
Uremia

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 patient who is comatose 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 for a medical alert bracelet, 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

Abstract

Patients who are comatose need to be rapidly assessed and treated in order to optimize clinical outcomes. A systematic approach to the history, physical examination, and diagnostic tests is presented.

Keywords:

coma, altered level of consciousness, Glasgow Coma Scale, decorticate posturing, decerebrate posturing, lumbar puncture (LP), psychogenic coma

Table 13-1. Glasgow Coma Scale

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

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 three sets of progressively more difficult questions.

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

7. What is the Glasgow Coma Scale?

The Glasgow Coma Scale is a simple scoring system used in patients in trauma to describe 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 (Table 13-1). It is not sensitive enough to detect subtle alterations of consciousness in the patient who is not comatose.

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

Vital signs often provide clues to the cause of coma. A core temperature should 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 cause related to the CNS. 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 measure oxygen saturation.

10. What is the Cushing reflex?

The Cushing reflex is 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 patient who is comatose 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 patient who is comatose?

The eyes should be examined for position and reactivity. 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. 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 patient who is comatose and 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 executive who is comatose and smells drunk may have had a sudden subarachnoid hemorrhage and spilled brandy on his or her shirt.
- Make sure that the patient is completely undressed so that occult injuries are not missed.

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

Obtain a rapid blood glucose level, and correct hypoglycemia if it is found. Pulse oximetry should be obtained on all patients to assess for hypoxia. If alcohol intoxication is suspected, determine the alcohol level. 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.

15. Which radiologic studies should be obtained in the patient who is comatose?

Computed tomography (CT) of the cervical spine should be obtained in any patient who is comatose with trauma to the face or head. A chest radiograph may be helpful if hypoxemia, pulmonary infection, or aspiration is suspected.

16. When should I order a CT scan of the head?

A head CT is not indicated in every patient who is comatose. A good history, a 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

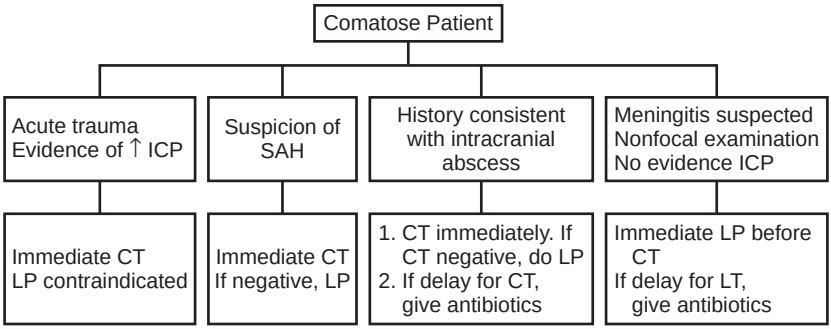

Figure 13-1. Timing and indications for lumbar puncture. *CT,* computed tomography; *ICP,* intracranial pressure; *LP,* lumbar puncture; *SAH,* subarachnoid hemorrhage.

trauma, history of cancer), a non–contrast-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.

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

The indications and timing of LP depend on the following two questions (Fig. 13-1):

1. Is CNS infection suspected?
2. Is there a suspicion of a structural lesion causing increased intracranial pressure?

18. 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, and circulation; and the 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. The goal of physical examination is to differentiate structural from metabolic cause.
2. Focus on vital signs, mental status, and motor examination.
3. Obtain a rapid blood glucose test for every patient who is comatose.

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

Obtain a rapid blood glucose test; if the glucose is low, treat the patient with dextrose 50% in water ($D_{50}W$). It is better to do a rapid blood glucose determination rather than to give glucose empirically. If opioid use is suspected, give 2 mg of naloxone intravenously. Thiamine (100 mg) may be administered to patients who are malnourished or those with a history of alcohol abuse. Antibiotic administration should be considered in all patients who are febrile with coma of unknown etiology. Intubation should be performed in patients who are hypoxic and those who are not protecting their airways. Avoiding hypotension and hypoxia is essential to optimize favorable outcomes.

20. 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 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 mimic 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 test.

- 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.

21. My patient has a history of seizures. Is there any special diagnosis I should consider?

Yes. Consider nonconvulsive status epilepticus. The patient may be having continuous seizures with little or no motor findings. Check carefully for any subtle rhythmic jerking of the eyes, facial musculature, or fingers. An electroencephalograph (EEG) may be required to make the diagnosis of nonconvulsive status.

22. What is locked-in syndrome?

Patients with locked-in syndrome are quadriplegic and cannot speak because damage has occurred to their motor tracts, but they remain completely awake and alert. Some of these patients retain the ability of limited eye movements.

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QUESTIONS

1. A patient who opens his eyes to painful stimuli, localizes to pain, and is verbalizing only incomprehensible sounds has a Glasgow Coma Scale score of
   a. 8
   b. 9
   c. 10
   d. 11
   The correct answer is *b.*
2. Application of a noxious stimulus resulting in hyperextension of the legs with flexion of the arms at the elbows is called
   a. Psychogenic coma
   b. Decorticate posturing
   c. Decerebrate posturing
   d. Locked-in syndrome
   The correct answer is *b.*
3. What physical finding would lead you to suspect a metabolic etiology of coma?
   a. Asymmetric muscle tone
   b. Increased blood pressure and decreased pulse rate
   c. Preserved pupillary reflexes
   d. When the eyelids are forcibly opened, the eyes flutter upward exposing the sclera
   The correct answer is *c.*

CHAPTER 14

FEVER

Lori A. Montagna, MD

1. What temperature constitutes a fever?

The literature describes a fever as a temperature of either 38°C (100.4°F) or 38.3°C (100.9°F). A neonatal fever is a temperature of 38°C. However, patients who are immunocompromised or functionally immunocompromised 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. Clinicians should maintain a high index of suspicion for masked fever in the elderly; neonates; patients with diabetes, chronic alcoholism, HIV/AIDS, or neutropenia; and those who use intravenous drugs or take chronic steroids or immune-modulating drugs. A temperature greater than 41.5°C (106.7°F) constitutes hyperpyrexia, which can be caused by severe infections or, more commonly, central nervous system hemorrhages.

2. Are all methods of measuring temperature equivalent?

Of the minimally invasive methods available, rectal temperatures are the most accurate representation of core body temperature. Oral, axillary, temporal artery, 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 alternative 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, and a fever is caused by elevation of the hypothalamic set point. Inflamed tissue or infecting organisms release pyrogens, which, in turn, activate prostaglandin E_2 (PGE2). PGE2 stimulates the hypothalamus to increase its thermoregulatory set point. The body responds by attempting to retain and generate heat (e.g., by vasoconstricting, increasing muscle tone, shivering, or increasing 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. This failure of thermoregulation occurs when the body absorbs or produces more heat than it releases. Some examples of hyperthermia include heat stroke, thyroid storm, burns, and toxidromes, such as neuroleptic malignant syndrome, serotonin syndrome, and malignant hyperthermia. It is important to distinguish between fever and hyperthermia, because the latter can be rapidly fatal and does not respond to antipyretics. The distinction is often made based on the events immediately preceding the elevated temperature (e.g., heat exposure, medication use, illicit drug exposure). Rapid cooling measures are imperative in cases of severe hyperthermia.

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

This situation is most commonly encountered in pediatrics. Parental palpation overestimates the presence of a fever, but parental accuracy improves when they use palpation in conjunction with a home thermometer. Parents are more likely to be accurate when they report that their baby is afebrile. Most experts feel that palpable fevers should still be taken seriously, and an appropriate assessment of the patient should be made.

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

In general, no. Before the advent of the *Haemophilus influenza* vaccine in the early 1990s and the pneumococcal conjugate vaccine in the early 2000s, there had been specific fever parameters that had been shown to indicate a greater likelihood of serious bacterial illness in children. But since then, the prevalence of these organisms as human pathogens has dramatically decreased, even in

Abstract

This chapter reviews the definition, causes, concerns, treatment, and management of fever.

Keywords:

temperature, fever, hyperthermia, neutropenic fever

nonimmunized children, because of the effect of herd immunity. And with the exception of neonates, it is generally recognized that clinical appearance is more important than the height of the fever. That being said, a temperature greater than 41.5°C (106.7°F) indicates hyperpyrexia, and severe infections, central nervous system abnormalities, and hyperthermia should be considered in patients of all ages.

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

Most physicians use antipyretics to improve patient comfort. Antipyretics should be considered particularly in patients who cannot tolerate the increased metabolic demands of a fever. Acetaminophen and ibuprofen are the most commonly used antipyretics. Ibuprofen has been shown to be more effective. Other nonsteroidal antiinflammatory drugs and aspirin are also options. Aspirin is not usually recommended in children because of the association with Reye syndrome. Complementary methods, such as undressing and cool bathing, generally do not significantly lower body temperature. If the temperature is greater than 41.5°C (106.7°F), rapid cooling measures should be initiated for possible hyperthermia (see Chapter 59).

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 the following 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 (e.g., cocaine, 3,4-methylenedioxymethamphetamine [MDMA, ecstasy], or other methamphetamines)
- Withdrawal syndromes (e.g., delirium tremens or benzodiazepine withdrawal)
- Factitious fever
- Medications

9. Which medications can cause fevers?

Any medication is capable of producing a drug fever; however, antibiotics cause one third of cases (Table 14-1). The fever usually begins 7 to 10 days after initiation of drug therapy. Associated findings include chills (53%), myalgias (25%), eosinophilia (22%), and rash (18%). 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, risk factors for possible immunocompromise, travel history, and past medical history, particularly comorbid illnesses. Be sure to perform a thorough head-to-toe physical examination when the patient is undressed and gowned. Consider sites of occult infection, such as

Table 14-1. Drugs Commonly Associated with Drug Fevers

Antibiotics	Central nervous system acting drugs
Penicillins	Phenytoin
Cephalosporins	Phenobarbital
Isoniazid	Carbamazepine
Nitrofurantoin	Thioridazine
Rifampin	Nonsteroidal antiinflammatory drugs
Sulfonamides	Ibuprofen
Minocycline	Salicylates
Antineoplastic drugs	Other
Bleomycin	Cimetidine
Streptozocin	Iodides
Cardiac drugs	Allopurinol
Procainamide	Prostaglandin E_2
Quinidine	Interferon

the ears, nose, and sinuses; feet; rectum; and pelvic area. Look closely at the skin for evidence of petechiae, purpura, cellulitis, or other concerning rashes.

11. What is the relationship between fever and tachycardia?

The pulse should increase by 8 beats per minute for each 1°C (1.8°F) increase in temperature (Liebermeister rule). 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 (Faget sign). This dissociation occurs with typhoid, malaria, legionnaires' disease, yellow fever, tularemia, brucellosis, and mycoplasma infection. In early septic shock, tachycardia that is out of proportion to the degree of fever is often seen. If this finding is paired with hypotension, the concern for sepsis is even greater. Tachypnea out of proportion to fever is characteristic of pneumonia and gram-negative bacteremia.

12. Do all patients with sepsis have a fever?

No. In fact, remember that systemic inflammatory response syndrome (SIRS) criteria include a 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 patients with infection 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. Consider immediate antibiotics for patients who appear toxic on presentation or who have suspected bacterial meningitis, as well as those in high-risk groups, such as the elderly, neonates, patients with oncologic disease, patients with sickle cell disease, and other patients who are immunocompromised.

14. What is a neutropenic fever?

A neutropenic fever is a single oral temperature of 38.3°C (101°F) or higher or a temperature of 38°C (100.4°F) or higher sustained over 1 hour. An oral temperature is preferred in patients with neutropenia, because a rectal temperature (or examination, for that matter) increases the risk of a local or systemic infection by colonizing gut organisms. *Neutropenia* is defined as an absolute neutrophil count (ANC) of fewer than 500 cells/mm^3 or an ANC that is expected to decrease to fewer than 500 cells/mm^3 within the next 48 hours (see Chapter 44).

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

The classic 1961 definition by Petersdorf et al, describes an FUO as a temperature greater than 38.3°C (100.9°F) documented on several occasions over more than 3 weeks, and an uncertain diagnosis after a 1-week hospitalization. Modern modifications on the definition note that the workup may be done as an outpatient and instead of 1 week of investigation, certain obligatory tests must be performed. These range from laboratory work to chest and abdominal imaging. Most commonly, FUO is found to be an occult infection followed by neoplastic disease or noninfectious inflammatory disease, but in many cases a source for the fever is never found.

16. Is there anything unique about fever in the elderly?

Elderly patients with fever are more likely to have a serious bacterial or viral illness when compared with younger patients. In addition, 20% to 30% of elderly patients with a serious infection may exhibit a blunted or absent febrile response. Some may also show a delayed febrile response. These factors, as well as atypical presentations for infections in the elderly, may delay diagnoses. Unlike the young, a source for an FUO can be found in the majority of cases (87% to 95%). The elderly patient with FUO is more likely to have an occult infection (35% versus 21%), with tuberculosis, abscesses, and endocarditis occurring more commonly in older patients. They are also more likely to have connective tissue disease, such as temporal arteritis and polymyalgia rheumatic, as well as malignancies, compared with their younger counterparts.

17. How long do typical febrile illnesses last?

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

CONTROVERSY

18. Is a fever a friend or foe?

This question has been controversial for centuries. Although fever, *per se,* is self-limited and rarely serious, it is often considered by patients, parents, and doctors to be a sign of severe illness. 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. Fever enhances immunologic processes, including the activity of interleukin (IL)-1, T helper cells and cytolytic T cells, and the synthesis of B-cells and immunoglobulin.

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

A recent Cochrane review has shown that there is some evidence that both alternating and combined antipyretic therapy may be more effective than monotherapy at reducing temperatures in children. There is no clear evidence as to whether dual therapy improves patient comfort, and overall, there is insufficient evidence to know whether dual therapy is beneficial. Many authors in the literature have expressed concern over inappropriate dosing and dosing intervals when either alternating or combined therapy is managed by caretakers.

20. Should antipyretics be given routinely after pediatric immunizations to prevent fevers?

No. A European study of 459 infants showed that although febrile reactions were significantly decreased with prophylactic acetaminophen, antibody responses to several vaccine antigens were also reduced.

21. Should antipyretics be given to prevent febrile seizures?

No. There is no evidence that antipyretics prevent febrile seizures.

KEY POINTS: FEVER

1. Increased temperature may be indicative of either a fever or hyperthermia from another cause.
2. Of the readily available methods, rectal temperatures are the most accurate representation of core body temperature.
3. The degree of temperature elevation is not predictive of serious illness.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. Diane M. Birmbaumer and Sarah M. Battistich for their previous contributions to this chapter.

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QUESTIONS

1. Core body temperature is controlled at which site in the body?
   a. Thyroid gland
   b. Hypothalamus
   c. Pituitary gland
   d. Basal ganglia
   The correct answer is *b*.
2. All of these patients are high risk for a masked fever, except:
   a. 75-year-old male with a history of myocardial infarction
   b. 9-month-old female with history of premature birth at 36 weeks
   c. 46-year-old female with chronic alcoholism
   d. 26-year-old male with HIV
   The correct answer is *b*.
3. All of the following can cause fevers, except:
   a. Benzodiazepine intoxication
   b. Subarachnoid hemorrhage
   c. Rheumatoid arthritis
   d. Hodgkin lymphoma
   The correct answer is *a*.

CHEST PAIN

Lee S. Jacobson, MD, PhD, and Shamai A. Grossman, MD, MS

CHAPTER 15

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

2. What life-threatening causes of acute chest pain must be considered first when evaluating a patient in the ED?

- Myocardial infarction (MI)
- Aortic dissection
- Unstable angina
- Pulmonary embolism (PE)
- Pneumothorax
- Endocarditis
- Pericarditis
- Myocarditis
- Cardiac tamponade
- Mediastinitis/esophageal rupture
- Trauma

3. What are examples of 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
- Symptomatic anemia
- Sickle cell anemia
- Vasoactive drug use
- Anxiety

4. 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, including not only the chest, but also the shoulder, arm, neck, jaw, abdomen, or back.

Abstract

Chest pain is one of the most common chief complaints of patients coming to an ED. It requires a careful systematic approach that includes a careful and complete history, focused physical examination, and indicated diagnostic testing, as well as a broad initial differential diagnosis focusing on the most serious possible causes of the chest pain.

Keywords:

chest pain, differential diagnosis of chest pain, evaluation of chest pain

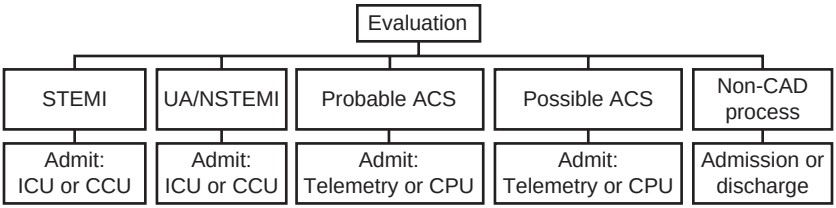

Figure 15-1. Evaluation of chest pain. *ACS,* Acute coronary syndrome; *CAD,* coronary artery disease; *CCU,* coronary care unit; *CPU,* chest pain observation unit; *ICU,* intensive care unit; *NSTEMI,* non–ST-segment elevation myocardial infarction; *STEMI,* ST-segment elevation myocardial infarction, *UA,* unstable angina.

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

All patients with acute chest pain should be approached with the assumption that a life-threatening cause is present. With few exceptions, once patient stability is established, supplemental oxygen, intravenous (IV) access, and cardiac monitoring should be initiated (Fig. 15-1) before any diagnostic studies are started.

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

An accurate history is the most important component of the evaluation. This history can be used to direct a physical examination and further studies.

- 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 15-1).
- Relief of chest pain with nitroglycerin or a gastrointestinal (GI) cocktail 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 29, 31, and 33.

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

Although the knowing classic risk factors for cardiac ischemia is 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 immediate risk of acute coronary syndrome (ACS). Recent American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines suggest that the most important factor in predicting ACS in a patient with chest pain is the history of present illness, rather than cardiac risk factors. However, recent studies suggest that a combination of risk factors, history, electrocardiograph (ECG), and laboratory results may successfully lead to early identification of patients at very low risk.

9. Are there any useful clinical prediction rules for stratifying patients with suspected PE according to their level of risk?

Yes. See Chapter 29.

10. Is radiation of chest pain significant?

Radiating chest pain is suggestive but not diagnostic of cardiac ischemia. Visceral pain (including that caused by cardiac, 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 specifically increases the likelihood of acute MI. Interestingly, although "typical" cardiac chest pain is traditionally taught as radiating to the left arm, recent evidence has suggested that radiation of chest pain to only the right arm may have a higher likelihood of cardiac origin than either radiation to both arms or 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/pleural irritation, pneumothorax, pneumonia, or musculoskeletal chest pain.

Table 15-1. 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 Levine sign, which consists of a patient placing a clenched fist over the sternum to describe the pain, is commonly associated with ischemic heart disease.
- The Kussmaul sign is a paradoxic filling of 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.
- Hypotension is an ominous but nonspecific sign commonly indicative of a more serious pathologic condition. This may be cardiogenic (as may occur in MI) or obstructive (such as in the case of pulmonary embolism, tension pneumothorax, or pericardial tamponade).
- The presence of tachycardia should raise the suspicion of serious abnormality, 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.
- Hypoxia is a sensitive but nonspecific marker of ominous pathologic findings. It can be caused by a variety of cardiopulmonary pathologies and should be treated with supplemental oxygen or positive pressure ventilation as clinically indicated. Hypoxia without a clear cause may require extensive workup.

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. Distant heart sounds may indicate pericardial tamponade. Mediastinal air from an esophageal or bronchial rupture results in a crunching sound called the *Hamman sign*. Decreased breath sounds, localized subcutaneous emphysema, or hyperresonance may indicate a pneumothorax. Localized rales suggest a pulmonary source 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 be related to a deep vein thrombosis (DVT) causing chest pain from a resultant PE.

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

ECG is an excellent rapid-screening assessment that can provide many clues, which are often diagnostic, to the source 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 patients in the ED who are later diagnosed as having had an acute MI.
- ST-elevation MI (STEMI) should be diagnosed by ECG alone (see Chapter 31), and should not require further testing except to rule out STEMI mimics, such as aortic dissection.
- In pericarditis, the initial ECG changes may consist of diffuse ST elevation with depression of the PR segment. Electrical alternans may also be seen in pericarditis with a severe pericardial effusion or tamponade.
- 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 may also result in peaked P waves, 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 rarely and is not pathognomonic.
- Comparison with previous ECGs is critical when possible.

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

The chest radiograph films of patients with chest pain are often normal but may provide a rapid diagnosis of several conditions, such as the following:

- Pneumothorax will often show a visceral pleural line on upright films or deep sulcus sign on supine films. If tension is present, the mediastinum will be shifted away from the side of 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 a Hampton hump; a wedge-shaped, pleural-based infiltrate representing an area of infarction; and the Westermark 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 31.

17. Is bedside ultrasound useful in identifying the cause of emergency chest pain?

In recent years there has been increasing focus on the use of bedside ultrasound by emergency practitioners. Multiple studies have demonstrated that with proper training, emergency physicians can be quite adept at using bedside ultrasound to answer focused clinical questions. Bedside ultrasound can rapidly and accurately elucidate multiple sequelae of conditions causing emergent chest pain, including pericardial effusion, pleural fluid, pneumothorax, and gross cardiac dysfunction. More advanced users have been able to detect more subtle findings, such as tamponade, contractility abnormalities in MI, and right heart strain in PE. In many circumstances, these studies are more accurate and rapid than plain radiographs. Although such findings are important supplements to clinical care, it is important to realize the limitations of such studies, including user variability (in both acquisition and interpretation of ultrasounds), image quality of bedside ultrasound machines, and factors that may confound imaging.

18. Are there any other 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 MI and unstable angina (ACSs) may remain unrelieved. Thus use of nitroglycerin as a diagnostic test should be avoided.
- Antacids or GI cocktails, consisting of viscous lidocaine and an antacid, often 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 often 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 and palpation.

19. Are there any other useful diagnostic imaging studies to help determine the cause of chest pain?

- Select patients at low to moderate risk for ACS may be risk stratified with coronary computed tomography (CT) angiography.
- Aortic dissection may be diagnosed by a thoracic arteriogram, a rapid-sequence CT scan with IV contrast, magnetic resonance imaging (MRI), or a transesophageal echocardiogram.
- A suspected PE may be confirmed by a ventilation-perfusion scan, spiral CT of the thorax, or conventional pulmonary angiography.
- Esophageal rupture may be diagnosed by an esophagogram with a water-soluble contrast material.

20. What special considerations must be taken into account when evaluating chest pain in patients patients who are geriatric, have diabetes, or are female?

- 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 show atypical symptom patterns more often than men. This is likely because of the higher prevalence of less common causes of ischemia, such as vasospastic and microvascular angina.

21. Is provocative stress testing useful in the emergent assessment of chest pain?

The current standard of care suggests that it is not only important to rule out an ongoing cardiac emergency but also to stratify patients for risk of an imminent major adverse cardiac event. Stress testing is an important tool for this purpose. Stress testing is a noninvasive tool to screen patients with emergent chest pain for intervenable coronary vascular lesions that may predispose them to future adverse cardiac events. It is important to note that stress testing is not appropriate in patients with a very low pretest probability for disease, because stress testing is more likely to evoke a false-positive result than a true-positive result and therefore will not change management and may lead to unnecessary use of valuable ED resources. On the other hand, stress testing in a patient with an extremely high pretest probability for a coronary vascular lesion can also result in false negatives and should be done in conjunction with cardiology. Therefore, good judgment is required to determine who is appropriate for ED stress testing. Imaging stress testing, such as stress echocardiography and myocardial perfusion studies, may produce lower false-positive and false-negative rates. Not all stress testing must be performed emergently. If patients are reliable, are at low risk for an intervenable coronary lesion, and have access to good follow-up care, it may be reasonable to administer stress tests to them as outpatients.

22. Approximately 2% to 4% of patients with chest pain caused by acute MI 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

Acknowledgments

Special thanks to Eric Wong, MD, who contributed to the previous version of this chapter.

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. Twenty-five percent of patients ultimately diagnosed with ACS do not have a primary complaint of chest pain.
4. Relief of chest pain by nitroglycerin or antacids is not diagnostic for either cardiac or noncardiac disease.
5. Chest pain in patients who are elderly or have diabetes is more commonly an emergent illness than in the general population, but often presents in atypical fashion because of underlying neuropathy.

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QUESTIONS

1. You are a physician in a tertiary referral center with full cardiac and surgical facilities. You examine a 56-year-old male with severe, 8 out of 10, retrosternal chest pain radiating to his back for the past 2 hours that woke him up from sleep. He felt very short of breath until a nurse outfitted him with nasal canula. He denies recent infection and has no other past medical history. His examination demonstrates a middle-aged man in obvious distress, jugular venous distention to 2 cm above the sternal notch, normal cardiac sounds, and clear lungs. He has bilateral 1+ pitting pedal edema, and peripheral pulses are thready. His ECG demonstrates sinus tachycardia ST elevations greater than 2 mV in leads III and AVF with less than 2 mV depressions in V5 and V6 that are new from a prior ECG. The most appropriate next step is
   a. To order a stat CT with angiogram
   b. To order high-sensitivity cardiac troponin T
   c. To place a stat consult to interventional cardiology
   d. To administer 0.4 mg of sublingual nitroglycerin

   The correct answer is *c.* This is a patient who shows typical cardiac chest pain and history, and has ECG changes consistent with acute STEMI. Interventional cardiology should be immediately consulted so that the cardiac catheterization laboratory can be activated to minimize potential myocardial ischemia. If there is clinical concern for other conditions such as aortic dissection, further workup can be performed (such as a CT scan) while the catheterization laboratory is being prepared, or in the catheterization laboratory itself; thus *a* is incorrect. Because laboratory testing should not delay time to cardiac catheterization and troponin may not be elevated in the early stages of STEMI, *b* is also incorrect. Although nitroglycerin can be used to treat cardiac chest pain, it should be used with extreme caution or avoided in patients with conditions that are dependent on preload, such as right-sided STEMI or aortic dissection, which are the two most likely diagnoses for this patient. Thus *d* is incorrect.
2. A 41-year-old male with type I diabetes and well-controlled hypertension had severe retrosternal chest pressure associated with dyspnea that occurred early this morning while eating breakfast. He had spent the night with friends drinking heavily and smoking cigars, which he admits he does several nights each week, resulting in frequent episodes of heartburn. His pain was relieved by ranitidine, magnesium aluminum hydroxide, and simethicone. His ECG shows a left bundle branch without significant change from prior ECGs. Examination, vital signs, chest radiograph, and initial cardiac biomarkers are all unremarkable 3 hours after onset of pain. Which of the following is least likely contribute to a missed MI in the ED?
   a. Incorrectly failing to recognize an STEMI by modified Sgarbossa criteria
   b. Initial evaluation by a first-year emergency medicine resident
   c. Relief of symptoms by GI cocktail
   d. The patient's age and gender

   The correct answer is *a.* Failure to correctly interpret ECGs is also a common reason for a missed diagnosis of ACS. Although a new left bundle branch block may be evidence of an ischemic change, in this instance the patient's ECG is unchanged from prior ECGs, and therefore it does not indicate evidence of new ischemia and would not meet Sgarbossa criteria. All other answers are known contributors to missed MIs.
3. A 24-year-old female with a past medical history of anxiety and IV heroin abuse presents with chest pain and mild shortness of breath on exertion. She has had multiple pneumonias in the last year. The examination is significant for fever to 102.6°F (39.2°C; responsive to acetaminophen), flat neck veins, diffuse wheezing with a respiratory rate of 22, a regular heart rate at 89 beats per minute without abnormal heart sounds, and track marks on both arms without evidence of cellulitis. She is not hypoxic at rest and is otherwise hemodynamically stable. Her ECG is unremarkable. A radiologist has read her chest radiograph as having patchy opacities consistent with pneumonia or pulmonary edema. She lives with her parents, is able to perform her activities of daily living (ADLs), and has a primary care physician with next-day appointments. Which of the following is most appropriate management for this patient?
   a. Admit her to the medical department for IV antibiotics and echocardiography.
   b. Discharge her to home with oral antibiotics and plan for her to visit her primary care physician tomorrow.
   c. Send troponin and pro–brain natriuretic peptide (pro-BNP) to rule out acute cardiac dysfunction.
   d. Collect blood and sputum cultures and place the patient in ED observation for IV antibiotics.

The correct answer is *a.* This is a patient at high risk for endocarditis because of her IV drug use. The presence of pneumonia does not exclude this and could be concurrent or possibly be a result of seeding from septic emboli. IV drug use also puts her at risk for HIV and HIV-related opportunistic infections such as *Pneumoncystis* pneumonia, which will not respond to traditional antibiotics. She should not be discharged without further workup (*b*). Fever makes cardiogenic pulmonary edema unlikely in this patient. In addition, cardiac enzymes and pro-BNP will not rule out endocarditis, which is the most concerning condition for this patient (*c* and *d*).

CHAPTER 16

ABDOMINAL PAIN, NAUSEA, AND VOMITING

Rick A. McPheeters, DO, FAAEM, and Juliana Karp, MD

ABDOMINAL PAIN

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

Evolving patterns of pain commonly 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 often signals the need for surgical intervention.

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

3. Which tests for peritoneal irritation are best?

Rebound tenderness during the physical examination is the traditional 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, highly sensitive tests for peritoneal irritation are the heel-drop jarring (Markle) and hop tests. Among patients with appendicitis, these tests have reasonable sensitivities and generally outperform 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, however, 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 as an initial symptom.

Abstract

This chapter is dedicated to the evaluation and management of undifferentiated nontraumatic acute abdominal pain and the disease states that are commonly diagnosed in this patient population. In addition, nausea and vomiting are both commonly encountered in the ED. Symptoms, etiologies, and treatment are discussed. Key points in diagnosing and managing nausea and vomiting are presented.

Keywords:

abdominal pain, acute abdominal pain, undifferentiated abdominal pain, nontraumatic abdominal pain, appendicitis, bowel obstruction, small bowel obstruction, cholecystitis, mesenteric ischemia, peritonitis, diagnostic testing in abdominal pain, patients with physical findings in abdominal pain, renal colic, ovarian torsion, abdominal pain in the elderly, nonsurgical causes of abdominal pain, nausea, vomiting, gastroenteritis, antiemetics

6. Discuss the pitfalls of 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 lipase, liver function studies, and alkaline phosphatase) and the liberal use of ultrasound or computed tomography (CT) scan is prudent in this age group.

7. What other factors should be sought in the history that may alter significantly the presenting symptoms 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. Advanced imaging (predominantly CT) has had the greatest impact on lowering the negative laparotomy rate.

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 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 lasting longer than 48 hours 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, conventional surgical wisdom proscribes the use of narcotic analgesics until a firm diagnosis is established. In a review article, Ranji and co-workers found that pain control with opiates may alter the physical examination findings, but these changes result in no significant increase in management errors. 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 level helps to determine whether there is antecedent anemia. An elevated WBC count suggests significant pathologic findings but is nonspecific. Elevated urinary specific gravity reflects dehydration, and an increased urinary bilirubin level 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 electrolyte, glucose, blood urea nitrogen, and creatinine tests 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 in the initial evaluation of suspected small bowel obstruction?

No. Abdominal CT has been shown to be significantly superior to plain films both in its diagnostic accuracy and in determining the level and cause of the obstruction. Exceptions include unavailable CT, expected delay in obtaining CT scans, the patient is *in extremis,* or instability negating transport to the CT suite.

15. Is oral contrast necessary when performing CT scans for suspected appendicitis?

No. In the ED, patients with acute abdominal pain and suspected appendicitis, oral contrast does not improve the diagnostic accuracy of CT and only delays the time to diagnosis.

16. Do all patients with uncomplicated appendicitis require surgery?

Urgent appendectomy remains the standard of care. Minneci and associates, however, have shown that nonoperative management with antibiotics may be a viable option. Further study is necessary to better define patient selection, long-term success rates, safety, and cost effectiveness.

17. A 7-year-old child comes to the ED with acute abdominal pain and 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 5 years, abdominal pain that is intermittent and of more than 3 months' duration is functional in more 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. Diabetic ketoacidosis (DKA)
2. Food poisoning
3. Pneumonia
4. Pelvic inflammatory disease

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

Patients with established DKA often come 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 with DKA have no such pathologic findings. 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 whose conditions are not stabilized. If symptoms persist despite adequate correction of DKA, then an underlying reason for surgery 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 has been cited 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 and co-workers 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.

NAUSEA AND VOMITING

21. Vomiting? Do I really need to read this section when there are so many more interesting topics 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.

22. What causes vomiting?

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

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

23. 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

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 intravenous (IV) rehydration and electrolyte replacement may be necessary.

24. List the common causes of vomiting.

See Table 16-1.

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

Yes, particularly during the first year of life. These include gastrointestinal atresia, malrotation, volvulus, Hirschsprung disease, gastroesophageal reflux, pyloric stenosis, intussusception, and inguinal hernia. (Vomiting in children presents considerations not covered in this chapter. See Chapter 64.)

26. Can the character of the vomit help me 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 often 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 35).

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

- Ask about 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.
- Discuss the 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.

Table 16-1. Common Causes of Vomiting

	GASTROINTESTINAL	NONGASTROINTESTINAL
Functional	Gastroparesis, irritable bowel syndrome	Pregnancy
Infectious/ inflammatory	Gastroenteritis, hepatitis, appendicitis, cholecystitis, pancreatitis	Pneumonia, meningitis, sepsis
Mechanical	Small bowel obstruction, ileus, gastric outlet obstruction	Renal calculi, ovarian torsion, testicular torsion
Medication side effects	NSAID-induced gastritis, didanosine-induced pancreatitis, valproic acid-induced pancreatitis	Digoxin, theophylline, aspirin, iron, opiates, antibiotics, chemotherapy, radiation therapy
Neurologic/ psychiatric	N/A	Increased intracranial pressure, vestibular disorders, bulimia nervosa and binge-eating disorders
Toxicologic/ metabolic	Alcoholic gastritis and pancreatitis, acetaminophen-induced hepatitis, chronic cannabis use	diabetic ketoacidosis, uremia, hypercalcemia

N/A, Not applicable; *NSAID*, nonsteroidal antiinflammatory drug.

- 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.

28. What do I look for during 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.

29. 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. Patients with diabetes and elderly patients can hide serious infections and metabolic disturbances. Be careful with these patients.

30. 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.

31. How should I treat the vomiting patient?

- Always remember to protect the airway. Patients with altered mental status should be placed on their sides to prevent aspiration. Intubate the patient's airway early when necessary.
- IV fluids usually are indicated for rehydration; normal saline or lactated Ringer solution is preferred. In some patients, especially children, oral rehydration may be preferred.
- Nasogastric suction can be therapeutic and diagnostic and may be indicated when there is a suspicion of a gastrointestinal bleed or small bowel obstruction.
- Medications to relieve nausea and vomiting must be used judiciously, especially in patients with altered mental status, hypotension, or uncertain diagnosis.
- Determine and, if possible, treat the underlying cause.

32. What medications should I use?

See Table 16-2.

Table 16-2. Antiemetic Medications

GENERIC NAME	TRADE NAME	INDICATION	DOSAGE
Palonosetron	Aloxi	Vomiting with chemotherapy	0.25 mg IV before chemotherapy
Meclizine	Antivert	Vertigo and motion sickness	25 mg PO qid
Dolasetron	Anzemet	Vomiting associated with anesthesia or chemotherapy	12.5 mg IV or up to 100 mg PO
Hydroxyzine	Atarax	Nausea, vomiting, anxiety	25-100 mg PO or IM tid or qid
Diphenhydramine	Benadryl	Motion sickness	25-50 mg PO or IV qid
Nabilone	Cesamet	Nausea and vomiting with chemotherapy	1-2 mg PO bid
Prochlorperazine	Compazine	Nausea, vomiting, anxiety	10 mg PO, IM, or IV qid, 25 mg PR bid (black box warning: elderly patients with dementia-related psychosis†)
Doxylamine + pyridoxine*	Diclegis	Nausea and vomiting of pregnancy	Dosage varies, 2-4 tabs in divided doses qid
Dimenhydrinate	Dramamine	Nausea, motion sickness	50-100 mg 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,	15-30 mL q 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 mg/kg IV or 1 mg PO bid (only on day of chemotherapy); also comes in patch
Dronabinol	Marinol	Refractory nausea and vomiting with chemotherapy	Dosage varies

Continued

Table 16-2. Antiemetic Medications—cont'd

GENERIC NAME	TRADE NAME	INDICATION	DOSAGE
Promethazine	Phenergan	Nausea, vomiting, motion sickness, anxiety	12.5-25 mg PO, PR, or IV qid (black box warning: children younger than 2 years; severe tissue injury, gangrene†)
Metoclopramide	Reglan	Nausea, vomiting, gastroesophageal reflux, gastroparesis	5-10 mg PO or IV dosage varies (black box warning: tardive dyskinesia†)
Chlorpromazine	Thorazine	Nausea, vomiting, anxiety	10-25 mg PO qid or 25 mg IM qid (black box warning: elderly patients with dementia-related psychosis†)
Trimethobenzamide	Tigan	Nausea and vomiting	300 mg PO tid or qid, 200 mg IM tid or qid
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	4-8 mg PO, IV, or IM; also comes in ODT form (black box warning: QT prolongation†); new warnings are being issued in pregnancy, data unclear

bid, Twice a day; *IM*, intramuscularly; *IV*, intravenously; *ODT*, orally disintegrating tablet; *PO*, per os (by mouth); *PR*, per rectum; *qid*, four times a day; *tid*, three times a day.

*From Koren G, Clark S, Hankins GD, et al: Effectiveness of delayed-release doxylamine and pyridoxine for nausea and vomiting of pregnancy: a randomized placebo controlled trial. *Am J Obstet Gynecol* 203: 571.e1–571.e7, 2010.

†From U.S. Food and Drug Administration: MedWatch: The FDA safety information and adverse event reporting program. Available at www.fda.gov/safety/medwatch; accessed 1-14-15.

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QUESTIONS

1. The ultrasound technician calls to notify you that your patient's pelvic ultrasound shows good arterial flow but absent venous flow of the left ovary. What should you do next?
   a. Treat for pelvic inflammatory disease.
   b. Consult an obstetrics and gynecology specialist.
   c. Discharge the patient to home with instructions for timely follow-up consultation with an obstetrics and gynecology specialist.
   d. Order a CT scan to better delineate the etiology of her pelvic pain.
   The correct answer is *b.* Absence of venous flow is more sensitive (67%) than arterial flow (46%) on ultrasound imaging for ovarian torsion, mandating emergent surgical evaluation.
2. A 25-year-old male with a past medical history of nephrolithiasis documented by CT comes to the ED with recurrent right flank pain consistent with previous episodes. Laboratory studies demonstrate normal renal function and microscopic hematuria without pyuria. He has right costovertebral angle tenderness, and his abdominal examination is normal. The patient's pain is well controlled with ketorolac, and his vital signs are normal. What is the next step in this patient's ED evaluation?
   a. CT
   b. Intravenous pyelogram
   c. ED-performed renal ultrasound
   d. Formal renal ultrasound
   The correct answer is *c.* Repeat CT scanning is unlikely to change management in patients with a high pretest probability of recurrent renal colic and low likelihood of an alternative diagnosis. Therefore its risk of excessive radiation generally outweighs its benefits in this patient population. Evidence of high-grade hydronephrosis, however, readily seen on ED-performed renal ultrasounds, could potentially change this patient's urgent diagnostic and therapeutic plan with little to no added risk and cost.
3. A 43-year-old obese female presents to the ED on Monday morning for persistent right upper quadrant colicky abdominal pain. Her last menstrual period was 2 weeks ago and regular. This is her third visit to the ED in the last 2 months for the same complaint. She has a positive Murphy sign, but otherwise her physical examination is normal. She is afebrile and tachycardic to 110 beats/min. A complete blood count, liver function tests, lipase test, and ultrasound examination remain normal, and her human chorionic gonadotropin (HCG) test is negative. On her previous visits, two separate right upper quadrant ultrasounds only showed biliary sludge without evidence of cholecystitis. On her second visit, a CT scan of the abdomen and pelvis was also performed and demonstrated no pathologic abnormality. With the fact that cholecystitis remains high on the differential, what is the next best course of action?
   a. Consult a representative from the surgery department.
   b. Repeat a CT scan.
   c. Repeat a right upper quadrant ultrasound.
   d. Perform hepatobiliary iminodiacetic acid (HIDA) scan.
   The correct answer is *d.* In general, right upper quadrant ultrasound is the initial imaging of choice to evaluate biliary disease because of its test characteristics and availability. CT is not routinely recommended, because it has a much lower sensitivity than ultrasound for gallbladder disease but can be useful if other diagnoses are suspected. Nuclear medicine scans have been found to be superior to both ultrasound and CT in diagnosing cholecystitis but unfortunately are not routinely available in the ED around the clock. HIDA scans are therefore most useful when the diagnosis remains uncertain and when they are available (i.e., weekdays).
4. In the diagnosis of the vomiting patient, it is important to:
   a. Always order laboratory tests and radiographs.
   b. Carefully focus on the gastrointestinal system.
   c. Utilize a thorough history and physical, especially in the young and elderly.
   d. Always aggressively treat with intravenous fluids and antiemetics.
   The correct answer is *c.* The work-up is always tailored to each individual patient. Some patients have a self-limited illness and require no testing while others are critically ill. Always be more careful in the young and the elderly.
5. All of the following are true in uncomplicated gastroenteritis except:
   a. Acute gastroenteritis is usually a self-limited disorder.
   b. True abdominal tenderness is not usually present in acute gastroenteritis.

c. Acute gastroenteritis may consist of vomiting only.
d. Laboratory testing and radiographs are seldom useful in acute gastroenteritis.
The correct answer is *c.* Acute gastroenteritis usually consists of both vomiting and diarrhea. It can be dangerous to make the diagnosis of gastroenteritis with vomiting only.

6. Common causes of nausea and vomiting other than gastrointestinal disorders include:
a. Infections
b. Metabolic disturbances
c. Myocardial ischemia
d. All of the above
The correct answer is *d.* There are many causes of nausea and vomiting other than gastrointestinal disorders. Always consider a wide differential initially. Take a thorough history and physical every time and do not initially focus on the gastrointestinal system.

CHAPTER 17

HEADACHE

Nicole M. Dubosh, MD, and Jonathan A. Edlow, MD, FACEP

1. How common are headaches, and what percentage of patients in the ED 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 headache, are conditions that threaten life, limb, brain, or eye, and are treatable (Table 17-1). 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 a tension or migraine headache. A headache that is described as a *first* or *worst* headache, or even substantially different from prior headaches, requires close evaluation. A sudden, severe onset, commonly described as "The worst headache I have ever had," is classic for an 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

Abstract

Headache is a relatively common presenting complaint of patients coming to the ED. Nearly everyone has a headache at some point in their lives, and whereas most headaches are benign, there are some types that have life-threatening causes. It is the role of the emergency physician to identify those with a serious, underlying cause. Obtaining a careful history and physical examination, including a neurologic examination, is prudent for identifying those patients who warrant neuroimaging and further workup. The emergency physician should have a lower threshold for performing a workup to look for a life-threatening cause of headache in patients who are older, have headaches that are new or different from their typical headaches, are immunocompromised (i.e., cancer, HIV), have focal neurologic findings on physical examination, and have risk factors for neurovascular lesions.

Keywords:

headache, subarachnoid hemorrhage (SAH), migraine, temporal arteritis

Table 17-1. Red Flags in Patients with Headaches

HEADACHE CHARACTERISTICS	DIFFERENTIAL DIAGNOSIS	POSSIBLE WORKUP (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, posttraumatic headache	Neuroimaging of brain and possibly cervical spine

CT, Computed tomography; *CVST*, cerebral venous sinus thrombosis; *ESR*, erythrocyte sedimentation rate; *LP*, lumbar puncture; *MRI*, magnetic resonance imaging; *SAH*, subarachnoid hemorrhage.

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?

- Do you get headaches often? 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.
- How bad is this headache? Have you had headaches this severe in the past? These questions assess the severity.
- Did the headache start suddenly or gradually? If sudden, what were you doing at the time it began? These questions go after the onset.
- 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 the result of that same etiologic cause.
- 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.
- What treatment have you used at home, and was it helpful? This can also help if a headache has responded in the past 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 temperature, pulse, or respiration may be caused by infection or toxins.

- Palpate the temporal arteries, sinuses (see Question 21), temporomandibular 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?

With advances in imaging technology, approximately 90% to 95% of SAHs are detected on CT scans of the head. Research shows that this number approaches 100% if the CT scan is performed within 6 hours of headache onset and is interpreted by a neuroradiologist. The rapid decline in sensitivity over time from headache onset is caused by 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. The need to perform an LP to rule out SAH in patients with an early negative CT is an area of controversy and ongoing research.

10. What are the CSF findings in an 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 with 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 caused by hemoglobin catabolism) is almost always found later (until about 2 weeks after 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 an SAH?

There are many tests, but none are perfect. Clearing of blood from tubes collected earlier, as well as those collected later, is commonly used and is helpful. However, unless the last tube contains no cells, SAH is still a possibility. Wasting a few milliliters of CSF between the first and last tubes facilitates this diagnosis. An elevated opening pressure suggests an 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, you must factor in the timing of the LP (with respect to the onset of the headache) in interpreting the LP results.

Table 17-2. Differential Diagnosis and Workup for Acute, Severe Headache

PATHOLOGIC PROCESS	CLINICAL CHARACTERISTICS	WORKUP
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: occipitonuchal 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; *LP*, lumbar puncture; *MRI*, magnetic resonance imaging.

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 workup 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 the following (Table 17-2):

- Pituitary apoplexy
- Cervical artery dissections
- Cerebral venous sinus thrombosis
- Posterior reversible encephalopathy syndrome (PRES; 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 pathophysiologic cause 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, and throbbing, and are commonly associated with photophobia and nausea. The headache may also be nonthrobbing. 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 patients with migraine 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, their frequency, and what workup 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 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/h, 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 40 to 60 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, including 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, 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 come 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, the 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 17-3).

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 commonly 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.

Table 17-3. Selected Medications for Acute Migraine Attacks

MEDICATION	DOSAGE 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	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	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
Dexamethasone	10-25 mg IV	A single IV administration decreases migraine recurrence

CAD, Coronary artery disease; *GI*, gastrointestinal; *HTN*, hypertension; *IM*, intramuscularly; *IV*, intravenously; *PVD*, peripheral vascular disease; *SQ*, subcutaneously.
*Assumes average-size adult patient.

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. The overuse, or prolonged use, of over-the-counter analgesics should be avoided, because these can lead to "medication overuse headaches." 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 73.

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, 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 nonspecific and may be a function of how

hard one is pressing. Finally and very important, 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 common complaint among patients with AIDS, 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 patients who have AIDS, 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 come 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. The headaches 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, VI, and $V_{1\text{-}2}$. Cavernous sinus disease may present as only a retroorbital 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 experience 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 workup 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 the result of 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 when the patient is standing and better when he or she is lying down), similar to a postdural puncture headache. Occasionally, patients will develop neurologic 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 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 you should get the tests that are needed, trying to balance radiation exposure with the need for an accurate diagnosis.

31. Is high blood pressure causing my patient's headache?

One potential mistake is to diagnose hypertensive urgency in patients with headache who have 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 reason 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 the result of 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 workup 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. CT scanners may miss 5% to 10% of SAHs, but the sensitivity markedly improves if the imaging is performed within 6 hours of headache onset. LP is needed if SAH is a major diagnostic concern, although this is an area of controversy in the field.
3. Patients who are HIV positive 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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QUESTIONS

1. A 68-year-old female arrives at the ED with a headache and jaw claudication. Her symptoms started gradually and have been present for 1 week. Her vital signs are normal. Her physical examination is notable for tenderness to her right temple. Her neurologic examination is otherwise normal. Laboratory reports are notable for ESR of 60. What is the next best step?
   a. Arrange for an emergent temporal artery biopsy.
   b. Initiate treatment with high-dose prednisone.
   c. Perform an MRI.
   d. Treat her pain with IV narcotics.
   The correct answer is *b.*
2. What type of headache is characterized by unilateral, intermittent pain episodes and with associated autonomic symptoms and is more prevalent in males?
   a. Cluster headache
   b. Migraine
   c. SAH
   d. Tension headache
   The correct answer is *a.*
3. Which of the following patients with headaches can be most safely discharged from the ED without brain imaging?
   a. A 45-year-old male with HIV who has headache and fever, and has experienced a seizure for the first time.
   b. A 60-year-old female with 2 weeks of a gradually worsening frontal headache, generalized malaise, and a 10-pound weight loss.
   c. A 21-year-old female with an abrupt-onset severe headache and vomiting that is resolving in the ED.
   d. A 37-year-old female with a history of migraines who has a 2-day history of a severe, pounding headache that was refractory to nonsteroidal antiinflammatory drugs (NSAIDs).
   The correct answer is *d.*

SYNCOPE, VERTIGO, AND DIZZINESS

Katherine M. Bakes, MD

CHAPTER 18

1. **Do I need to be concerned by a complaint of dizziness?**
   Yes. Approximately 5% of patients who come to the ED with dizziness will have a serious neurologic condition, such as ischemic stroke, intracranial hemorrhage, brain neoplasm, demyelinating disease, and cerebral infection. Risk factors for serious neurologic etiologies include age 60 years and older, risk factors for stroke (e.g., atrial fibrillation, hypertension, and diabetes), chief complaint of imbalance, and any focal neurologic abnormality (other than nystagmus).

2. **How do I approach the vague and ill-defined complaint of dizziness?**
   Start your history with an open-ended questions. Patients should be allowed to describe their symptoms without prompting, so you can understand what they mean by *dizzy. Dizzy* can describe a sensation of vertigo (the illusion of motion), lightheadedness (presyncope or frank syncope), or disequilibrium (imbalance), each with disparate causes.

3. **What causes dizziness?**
   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 vestibular system, located in the petrous portion of the temporal bone, is made up of a semimembranous labyrinth that is filled with fluid (endolymph) and contains the utricle, the saccule, and the three semicircular canals. The utricle and saccule respond to gravitational forces and linear movement, whereas the semicircular canals, oriented in the three planes of space, respond to rotational movements. Otoliths rest on the hair cells of the utricle and the saccule. The mass of the otoliths is needed to stimulate the hair cells in response to gravitational pull (force = mass $\times$ acceleration). When the head turns, endolymph bends and stimulates the hair cells within the semicircular canals. Impulses from the hair cells are sent to the brain via the eighth cranial nerve. The nucleus of the eighth cranial nerve interconnects with other cranial nerves, the cerebellum, and the sensory and motor tracts to coordinate visual and motor responses.

5. **How do you differentiate central versus peripheral vertigo?**
   This is an anatomic distinction. Peripheral vertigo is caused by a dysfunction of the inner ear or vestibular nerve, whereas central vertigo is from etiologies of the brain or brain stem. Benign paroxysmal positional vertigo (BPPV), vestibular neuritis, and Meniere disease are common causes of peripheral vertigo, whereas vertebrobasilar ischemia, multiple sclerosis, cerebellar infarction/hemorrhage, and basilar migraine are causes of central vertigo.

6. **What are the common characteristics of peripheral vertigo?**
   The mnemonic *DR FLIP* reminds you that the Epley maneuver, which flips the patient, helps BPPV.

   Deafness (unilateral, best detected with the Weber test)
   Ringing in the ears (tinnitus)
   Fatigable on repeated testing (central suppressive mechanisms still function)
   Latency after Dix-Hallpike maneuver
   Intense symptoms (with head movement to one side)
   Positional in nature

Abstract

Syncope, vertigo, and dizziness are common ED complaints. This chapter discusses in detail how to differentiate between serious and benign etiologies of these symptoms.

Keywords:

vertigo, syncope, benign paroxysmal positional vertigo (BPPV), Meniere disease, vestibular system, dizziness, Dix-Hallpike maneuver, Epley maneuver, San Francisco syncope rule, Boston syncope rule, nystagmus, head thrust maneuver, vestibular neuronitis, vasovagal

Table 18-1. Key Points for the Main Causes of Peripheral Vertigo

Benign Paroxysmal Positional Vertigo
Most common cause (50%)
Hearing not affected
Recurrent; <1 minute paroxysms elicited with head turning
Normal between brief attacks
Result of 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
Meniere Disease
Less common cause (10%)
Less acute onset (hours) and can last for weeks at a time
Associated with hearing loss, tinnitus
Caused by swelling of the semimembranous labyrinth (vestibular and cochlear components)
Responds to diuretics or fluid restriction
Can lead to permanent hearing loss

7. What are the characteristics of central vertigo?

- Cranial nerve deficits (as well as other neurologic signs)
- Vertical nystagmus (not seen in peripheral vertigo)
- Ataxia (with gait impairment and not only with head turning)

8. What are the key points for the main causes of peripheral vertigo?

See Table 18-1.

9. What should be included in the physical examination of a patient with vertigo?

Examine the eyes for ocular palsies (central etiology) and document the presence and characteristics of nystagmus. Examine the ears for foreign bodies, infection, perforation, and cholesteatomas. Vertigo associated with unilateral hearing loss (in the absence of other neurologic signs) points to a peripheral etiology, where these functions anatomically reside in close proximity. Because patients often do not appreciate subtle changes in hearing, the Weber and Rinne tests should be utilized for a more objective and sensitive assessment. Perform a full neurologic examination, including cranial nerves, gait, stance, and cerebellar function. The Dix-Hallpike maneuver will diagnose BPPV, whereas the Epley maneuver effectively treats it.

10. How is nystagmus evaluated in the workup of vertigo?

The presence of nystagmus supports vertigo as the cause of "dizziness." Horizontal or rotary nystagmus can be found in peripheral or central etiologies, and thus does not exclude brain or brain stem pathologic abnormality. Vertical, changing-direction, and nonsuppressible nystagmus are pathognomonic for a central etiology. Because the brain's central suppressive mechanisms are intact, patients with peripheral etiologies will be able to suppress their nystagmus (and the sensation of vertigo) by visually fixating on a stationary object. Suspect a central etiology in patients who have very active nystagmus and prefer to keep their eyes closed.

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 vestibuloocular 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. Although not pathognomonic, a patient with unidirectional horizontal nystagmus (a positive head thrust test) and

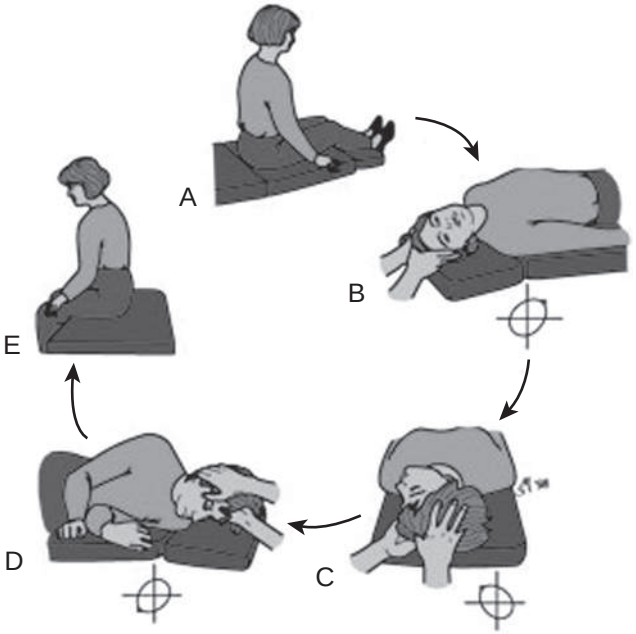

Figure 18-1. Epley maneuver.

no other neurologic signs is more likely to have a peripheral etiology of vertigo. A negative head thrust test should raise suspicion for a central etiology of vertigo.

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, to a supine position with the head hanging down at 30-degrees' extension. Nystagmus, often toward the dependent eye or forehead, is characteristically associated with a delay of a few seconds (as otoliths move through endolymph) and fatigable symptoms of vertigo on repetition (intact central suppressive mechanisms).

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 the first attempt and up to 98% after two attempts. The technique involves performing the Dix-Hallpike maneuver, then turning the head to the opposite side while the patient is still supine, and then turning the patient over in the same direction before asking the patient to rise (Fig. 18-1). It is important to allow adequate time for the otoliths to settle between each change in position. Because of the natural fatigability of symptoms, the patient's sensations cannot be relied on to guide the time spent in each position. Thus the examiner should note the time it takes for symptoms and nystagmus to abate during the Dix-Hallpike maneuver and apply that time to each position of the Epley maneuver.

14. How do I treat peripheral (and central) vertigo?

Meniere disease is treated with diuretics or salt/fluid restriction. BPPV is treated by the Epley maneuver. Nonspecific vestibular suppressants include the following:

- Anticholinergics (e.g., scopolamine transdermal)
- Antihistamines (meclizine 25 mg orally [per os; PO] every 6 hours as needed for vertigo and diphenhydramine 25 to 50 mg PO every 4 to 6 hours)
- Benzodiazepines (diazepam 5 to 10 mg PO every 6 hours)

Peripheral vertigo not amenable to repositioning maneuvers is best monitored by an ear, nose, and throat (ENT) physician, who can do more specialized vestibular testing to localize the lesion.

Central vertigo requires evaluation by a neurologist or neurosurgeon. Magnetic resonance imaging (MRI) to evaluate the posterior fossa and brainstem is recommended.

15. What is syncope?

Syncope is 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. Seizures may mimic syncope.

16. What are the odds of determining the cause of a syncopal episode?

Despite extensive and expensive ED workups, 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 pregnancy, **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), is often correctable but easily overlooked. Seizures don't cause, but can mimic, syncope. Vertebrobasilar insufficiency and subarachnoid hemorrhage (SAH) indicate 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, preexcitation, 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
- 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.

- Obtain vital signs and evaluate and treat for immediate life threats.
- Check a bedside glucose level and consider naloxone for any patient with persistent altered mental status.
- 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.
- 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, particularly if the patient is taking anticoagulants.

21. 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, particularly if the patient did not have time to protect himself or herself from injuries (e.g., facial trauma). Similarly, syncope associated with exercise, or while the patient was 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 any previous episodes of syncope, syncope associated with upper extremity exertion (e.g., subclavian steal syndrome), and the presence of cardiac risk factors. A family history of sudden death may suggest Brugada, preexcitation, 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 patient who has had a generalized seizure awakens slowly with prolonged confusion or postictal state. Both may have trauma. The Denver Seizure Score, or the Δ bicarbonate level plus twice the Δ anion gap (AG) {[24 – bicarbonate] + [2 × (AG – 12)]}, can be used to differentiate unwitnessed loss of consciousness as either syncope or seizure. If blood is collected within 30 minutes of the event, a score of greater than 20 has a high likelihood of being a 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 syncope from abrupt effort or exercise syncope may have aortic stenosis or hypertrophic cardiomyopathy. Look for narrow pulse pressure, systolic murmur, or change in murmur with the Valsalva maneuver. 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. A patient who has returned to normal mental status is unlikely to have hypoglycemia as the cause of syncope. If anemia is suspected on clinical examination (pale skin and pale conjunctivae), a bedside hematocrit or hemoglobin test is indicated. 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 syndrome, and helps in risk stratification for ACS. Check for markers of cardiac disease, such as ischemia, infarction, arrhythmias, preexcitation, 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, age greater than 65 years, abnormal ECG, and unexplained syncope who have suspected heart disease should be admitted and evaluated for ACS. Echocardiography, exercise treadmill testing, Holter ECG monitoring, and electrophysiologic studies may be useful during the inpatient stay.

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 but do help identify risk factors for serious conditions (Table 18-2). The San

Table 18-2. Syncope at High Risk for Cardiac Etiology

HISTORICAL	ED EVALUATION
Age > 65 years	Abnormal vital signs
Cardiovascular disease history (especially heart failure)	Systolic blood pressure < 90
Lack of prodrome	Evidence of congestive heart failure
Exertional	Abnormal electrocardiogram
Chest pain with event	Hematocrit < 30%
Palpitations with event	
Family history of sudden death	

Francisco syncope rule predicted short-term adverse events in patients with the following signs: abnormal ECG result, shortness of breath, systolic blood pressure less than 90 mm Hg, hematocrit level less than 30%, and CHF by history or examination. Unfortunately, this rule was found to have only 75% sensitivity on external validation. The Boston syncope rule has demonstrated excellent sensitivity, but still requires external validation. The rule recommends admission for any patient with ACS, conduction disease, worrisome cardiac history (e.g., dysrhythmia, pacemaker), valvular heart disease, family history of sudden death or conduction abnormality, volume depletion (e.g., gastrointestinal bleed, hematocrit < 30%), persistent abnormal vital signs in the ED, or a primary central nervous system etiology.

Acknowledgment

The editors and author of this chapter would like to acknowledge and thank Dr. William F. Young, Jr. for his previous contributions to this chapter.

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QUESTIONS

1. All of the following are common manifestations of BPPV, except:
   a. Latency with the Dix-Hallpike maneuver
   b. Inability to suppress the nystagmus with visual fixation
   c. Fatigability
   d. Symptoms with head turning
   The correct answer is *b.*
2. What suggests a peripheral etiology of vertigo?
   a. Hearing loss without other neurologic signs
   b. Vertical nystagmus
   c. Fixed gait ataxia
   d. Negative head thrust test
   The correct answer is *a.*
3. All of the following are risk factors for a serious adverse event after syncope, except:
   a. Bradycardia on ECG
   b. Pulmonary edema
   c. Age greater than 65 years
   d. Prolonged prodrome before the event
   The correct answer is *d.*

SEIZURES

Shawn M. Varney, MD, FACEP, FACMT

CHAPTER 19

1. What is a seizure?

A seizure is an episode of abnormal brain function caused by excessive and aberrant electrical neuronal discharge and may result in characteristic motor findings recognized as *seizure activity*. In addition to tonic-clonic muscle activity (*tonic* refers to muscle stiffening; *clonic* refers to muscle jerking), 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, because seizures are a common presenting problem in the ED. Prolonged excessive electrical activity in the brain directly causes neuronal destruction, especially in the hippocampus.

2. How are seizures classified?

In general, seizures are divided into two groups: generalized and focal (Table 19-1). Generalized seizures affect a large volume of brain tissue during the abnormal electrical activity, whereas focal seizures involve a specific brain area. Focal seizures, whether simple or complex, may lead to bizarre manifestations, including hallucinations, memory disturbance, visceral symptoms (abdominal symptoms), and perceptual distortions, and may lead to a misdiagnosis of a psychiatric problem.

3. What are the causes of seizures?

Primary seizures are recurrent episodes of abnormal electrical brain activity without a recognized underlying cause and are classically referred to as *epilepsy*. Secondary seizures (also called *provoked seizures*) usually have a nonneurologic underlying condition. Table 19-2 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 seizure mimics include syncope, hyperventilation syndrome, migraines, movement disorders, narcolepsy, and psychologic disorders. *Pseudoseizure* is a special category and is discussed later (see Question 16).

5. What should my priorities be in managing a patient who is actively experiencing a seizure?

Clinical priorities are airway, breathing, and circulation (ABCs). Give direct attention to the airway first, although it is rare that a seizure patient needs to have his or her airway intubated. Suction oral secretions to prevent aspiration, and position the patient on his or her side. Give supplemental oxygen to treat the increased oxygen demand caused by the generalized muscle activity. Supplemental ventilation (bag-valve-mask) is rarely needed. Do not place objects into the patient's mouth that might be bitten off (including fingers) and thus become an obstructing foreign body. Nasopharyngeal airways may provide a patent airway and improve ventilation. Note the patient's blood pressure, pulse, and capillary refill. Check the temperature and administer antipyretics in appropriate doses when needed. Gently restrain the patient or place a blanket or sheet under the head to help the patient avoid self-harm.

Diagnostic priorities should focus on likely reversible causes of seizures, such as hypoglycemia, metabolic disturbances, and potential toxicologic exposures.

6. What do I do if the seizure does not stop?

Intravenous (IV) benzodiazepines are the first-line therapy. Order and administer them immediately. Most seizures last for less than 1 to 2 minutes. Lorazepam (2 to 4 mg IV) is the conventional first choice because of its longer duration of action. However, it also may have a slightly slower onset of

Abstract

Seizures are abnormal neuronal discharges, usually causing motor activity, and are a common presenting problem in the ED. An essential part of emergency care is to know how to identify true seizures, perform a tailored evaluation, and treat patients who have had seizures. Seizures result from many causes, especially from toxicologic exposures. General treatment principles include sufficient doses of benzodiazepines, but may require phenytoin/fosphenytoin, valproic acid, or Levetiracetam. General anesthesia with propofol or a barbiturate may be needed for status epilepticus.

Keywords:

Seizure, anticonvulsants, pseudoseizure, status epilepticus

Table 19-1. Classification of Seizures

Type
Generalized
Tonic-clonic (grand mal)
Absence (petit mal)
Atonic (drop attacks)
Myoclonic
Tonic
Clonic
Partial or Focal
Simple partial
Complex partial
Partial with secondary generalization

action compared with diazepam (5 to 10 mg IV), which may also be used. Diazepam may also be administered by the intraosseous and rectal routes, but is not recommended for intramuscular use because of uneven uptake.

Once the seizure has ceased, anticonvulsants are used to prevent recurrence. Although phenytoin and fosphenytoin are the drugs of choice, they are not always successful, may cause cardiovascular compromise when rapidly infused, and are not effective for toxin-induced seizures. Table 19-3 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*. Immediate seizure-abortive intervention with benzodiazepines is indicated along with simultaneous attention to the ABCs, screening for the underlying cause, and immediate treatment of life-threatening etiologies. Table 19-4 gives an algorithm for managing the patient with status epilepticus.

8. Which historical and physical findings suggest a seizure?

Historical features include a history of a seizure disorder or brain neoplasm, a preceding aura (warning symptoms and signs of an imminent seizure), known precipitants (strong emotions, flashing lights, stress, lack of sleep), and circumstances surrounding the event (compliance with medication regimen, drug/alcohol use or withdrawal, head trauma, infection). Physical findings associated with seizures include tongue biting, bowel or bladder incontinence, witnessed seizure activity, trauma from the seizure, lateralizing abnormalities, loss of consciousness, and a postictal state of confusion or somnolence.

9. In addition to the neurologic examination, what other parts of the physical examination are important?

A complete head-to-toe examination may reveal causes of and trauma from the seizure. Specifically, examine the skin (meningococcemia or stigmata of liver failure), head (trauma), and neck (nuchal rigidity may indicate meningitis or subarachnoid hemorrhage). The neurologic examination is most important. Focal neurologic findings, such as focal paresis after the seizure (Todd paralysis), may indicate a focal cerebral lesion (e.g., tumor, abscess, or cerebral contusion). Evaluation of the cranial nerves and the fundi can reveal 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 a patient with a prior history of seizures who has 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. There are no perfect tests with definitive thresholds to determine whether a major motor seizure occurred, but some test options are creatine phosphokinase (stays elevated up to 24 hours), anion gap (elevated if done within 1 hour), or prolactin (elevated if drawn within 20 minutes of seizure and compared to a 6-hour level).

Table 19-2. Etiologies of Secondary (Provoked) Seizures

CATEGORY	EXAMPLE
Drugs/toxins (multiple)	Anticholinergics/antihistamines Anticonvulsants (carbamazepine, valproic acid) Antidepressants (bupropion, tricyclics) Camphor CO, CN, hydrogen sulfide, azides GHB Gyromitra (mushroom), hydrazine Iron Isoniazid Lidocaine Lithium Metaldehyde Opioids (meperidine, propoxyphene, tramadol) Organophosphates/carbamates Salicylates Sympathomimetics (amphetamines and derivatives, cocaine) Synthetic cannabinoids Theophylline
Central nervous system lesions	Hypertensive encephalopathy Intracranial hemorrhage Mass lesions Structural Trauma (recent and remote) Vascular lesions
Infectious diseases	Cerebral abscess Cerebral parasitosis Encephalitis HIV Meningitis
Metabolic	Fever ("febrile seizures") Hepatic encephalopathy High anion-gap acidosis Hypocalcemia Hypoglycemia, hyperglycemia Hypomagnesemia Hyponatremia, hypernatremia Hypothyroidism, hyperthyroidism Uremia
Other	Subtherapeutic antiepileptic drug levels Withdrawal (barbiturates, ethanol, sedative-hypnotics) Eclampsia

CO, Carbon monoxide; *CN*, cyanide; *GHB*, γ-hydroxybutyric acid.

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. Nevertheless, it is reasonable to check serum electrolytes (sodium, calcium, magnesium), glucose, renal and liver function, toxicology screen, and complete blood count. A pregnancy test for women of childbearing years may affect the choice of antiepileptic therapy and/or disposition. An electrocardiogram may rule out seizure mimics. The routine use of lumbar puncture in patients with new-onset seizures is not indicated.

Table 19-3. Anticonvulsants

DRUG	ADULT DOSAGE
Phenytoin	15-20 mg/kg IV at <50 mg/min
Fosphenytoin	15-20 mg PE/kg IV at 100-150 mg PE/min; may be given IM
Phenobarbital	20 mg/kg IV at 50 mg/min; may be given IM; may repeat dose in 10 min
Valproate	20-40 mg/kg IV over 1 hr
Levetiracetam	1500 mg IV at 100 mg/min
Pentobarbital	5 mg/kg IV at 25 mg/min, then titrate to EEG; intubation required
Isoflurane	Via general endotracheal anesthesia

IM, Intramuscularly; *IV*, intravenously; *PE*, phenytoin sodium equivalents.

Table 19-4. 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 Lorazepam, given at 2 mg/min, 0.1 mg/kg IV up to 4 mg per dose, may repeat in 5 min (*or* diazepam, 0.15 mg/kg IV up to 10 mg per dose, may repeat in 5 min; *or* midazolam 0.2 mg/kg up to 10 mg IM or 0.2 mg/kg IV load, followed by infusion of 0.05-0.2 mg/kg/hr)
10-20 min	Phenytoin, 20 mg/kg IV at 50 mg/min (to 30 mg/kg if seizures continue); *or* fosphenytoin, 20 mg/kg PE IV at 150 mg/min Valproate, 20-40 mg/kg IV at 3-6 mg/kg/min; may give additional 20 mg/kg after 10 min Levetiracetam, 1000-3000 mg IV load at 2-5 mg/kg/min
30 min	And/or General anesthesia with midazolam, 0.2 mg/kg initial infusion at 2 mg/min, then continuous infusion of 0.05-2 mg/kg/hr Propofol, 2 mg/kg IV, then 5 mg/kg/hr; may repeat 2 mg/kg in 5 min Phenobarbital, up to 20 mg/kg IV at 50 mg/min; may repeat in 10 min Pentobarbital, 5-15 mg/kg at 50 mg/min, then 0.5-5 mg/kg/hr*

IV, Intravenous; *IM*, intramuscular; *PE*, phenytoin sodium equivalents.
*By this time a critical care physician would be involved.

12. What imaging studies are indicated?

In the patient with a first-time seizure, emergent noncontrast head computed tomography (CT) is recommended when suspecting a structural lesion (intracranial bleeds, masses, large strokes, or trauma). 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 receiving anticoagulation therapy; and patients who are immunosuppressed and HIV positive. Emergent neuroimaging should also be considered in the patient with first-time seizure who is older than 40 years or who had a partial seizure. In addition, patients with a prior history of seizures who have a new or different seizure pattern should undergo imaging studies.

13. What should be the disposition of the patient who has a seizure?

Patients who come to the ED with any of the following should be considered for admission to the hospital for evaluation and therapy: persistent altered mental status, central nervous system (CNS)

infection, new focal abnormality, new intracranial lesion, underlying correctable medical problem (e.g., significant hypoxia, hypoglycemia, hyponatremia, dysrhythmia, and alcohol withdrawal), acute head trauma, status epilepticus, and eclampsia. In the patient with a history of seizures who has a simple seizure and a subtherapeutic anticonvulsant level, correct the level before discharge. Patients with new-onset seizures who have normal workups in the ED and are medically stable may be considered for discharge.

14. What discharge instructions should the patient receive?

Follow-up consultation with the patient's primary care physician or a consulting neurologist should be arranged. Inform the patient of the possibility of another seizure, and instruct him or her to avoid working with hazardous machines, driving, and performing other activities that may result in serious injury if the he or she has another seizure. Also, most states have mandatory reporting laws to the Department of Motor Vehicles if the patient has a driver's license.

15. Should I start antiepileptic medication before discharge in the patient with a new seizure?

In general, no, but 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 prescribed anticonvulsants until seen in a follow-up visit and further testing (i.e., electroencephalogram [EEG]) is completed.

16. What is a pseudoseizure?

Pseudoseizures (psychogenic nonepileptic seizures [PNESs]) are functional events that may mimic seizures in their motor activity or behavior but are not caused by abnormal electrical discharges in the brain. In general, patients with PNES have underlying anxiety or hysterical/histrionic personality disorders. PNESs are 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 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 PNES. Simultaneous video and EEG monitoring can help to differentiate a true seizure from a PNES. Exercise caution in diagnosing PNES, because 5% to 50% of patients with PNES also have epilepsy.

17. Name some etiologies of seizures that generally do not respond to the usual medications, and name the antidote (adult doses).

- Eclampsia: Magnesium sulfate 6 g IV over 15 minutes, then 2 g/h. Consider this etiology in women between 20 weeks' gestation and 6 weeks' postpartum.
- Hypoglycemia: Dextrose 0.5 to 1 g/kg IV
- Hyponatremia: 3% hypertonic saline 100 to 200 mL over 1 hr
- Isoniazid (INH) ingestion: Pyridoxine (vitamin B_6) 5 g IV given over 10 minutes, or 1 g for each gram of INH ingested.

18. What are simple febrile seizures?

See Chapter 62, Seizures in Infancy and Childhood.

Acknowledgment

The editors and author of this chapter would like to acknowledge and thank Dr. Kent Hall for his previous contributions to this chapter.

KEY POINTS: SECRETS FOR SEIZURES

1. Regardless of the agent chosen, the primary goal is to stop the seizure as rapidly as possible.
2. Always check a blood glucose level in a patient having a seizure early in the resuscitation process.
3. Drugs of choice for status epilepticus include benzodiazepines (first-line therapy), followed by phenytoin/fosphenytoin or valproate, and finally levetiracetam, propofol, or barbiturates.
4. Obtain images for seizure patients with focal neurologic deficits, persistent altered mental status, fever, recent trauma, persistent headache, history of cancer, history of anticoagulation, HIV (AIDS), and when timely follow-up care cannot be ensured.

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QUESTIONS

1. Which of the following medications is the initial drug of choice for terminating active seizures?
   a. Phenytoin/fosphenytoin
   b. Valproic acid
   c. Phenobarbital/pentobarbital
   d. Lorazepam/diazepam
   e. Propofol
   The correct answer is *d.*
2. Which of the following actions is inappropriate in a patient with a seizure?
   a. Administering medications to stop the seizure
   b. Performing a finger sweep of the mouth to remove a foreign body
   c. Checking a blood glucose level
   d. Inserting a nasopharyngeal airway
   e. Placing a blanket or padding under the patient's head
   The correct answer is *b.*
3. All of the following are indicated upon disposition from the ED in a patient with a provoked seizure, except:
   a. Identifying a potential etiology for the seizure
   b. Notifying the patient and the Department of Motor Vehicles according to state mandatory reporting laws
   c. Initiating anticonvulsant therapy
   d. Arranging follow-up with the patient's primary care provider or a neurologist
   e. Performing a noncontrast brain CT scan if the following are present: focal neurologic deficits, persistent altered mental status, fever, recent trauma, persistent headache, history of cancer, history of anticoagulation, or HIV (AIDS), or when timely follow-up care cannot be ensured.
   The correct answer is *c.*

ANAPHYLAXIS

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CHAPTER 20

1. What is anaphylaxis?

Anaphylaxis is a serious allergic reaction that is mediated by immunoglobulin E (IgE), 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?

An anaphylactoid reaction is a potentially fatal syndrome clinically similar to anaphylaxis but 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.

Anaphylaxis is caused by 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 (urticaria or rash)
- Mucous membranes (angioedema)
- Upper respiratory tract (edema and hypersecretion)
- Lower respiratory tract (bronchoconstriction)
- Gastrointestinal symptoms (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 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 percentage of patients who come to the ED with anaphylaxis have no known history of allergies or anaphylaxis?

25%

7. What is the role of diagnostic studies?

There is no immediate role for diagnostic studies in the ED, because diagnosis and treatment is 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 may be measured in the ED. There is a role for skin testing either before administration of an antigen or in follow-up referral to determine the exact allergens involved.

Abstract

Acute allergic reactions are sometimes life threatening and need immediate treatment. Their recognition, differential diagnosis, and treatment is discussed in this chapter.

Keywords:

allergy, anaphylaxis, anaphylactoid reaction, C1 esterase deficiency, hereditary angioedema (HAE), epinephrine administration

8. What is the differential diagnosis?

Conditions include hereditary angioedema (HAE), septic and cardiogenic shock, asthma, croup and epiglottitis, vasovagal syncope, and any acute cardiovascular or respiratory collapse of unclear origin.

9. 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 the result of a capillary leak mediated by histamine release. It may be treated by the administration of antihistamines (i.e., orally, intramuscularly [IM], or intravenously [IV]) or epinephrine (i.e., subcutaneously or IM).

10. What is HAE? 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 HAE.

11. How does the treatment of HAE differ from that of anaphylaxis?

HAE 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, HAE 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 examination in the ED, treat the patient as if he or she were having an allergic reaction. If there is minimal or no response to therapy, consider IV fresh frozen plasma (FFP), which contains C1 esterase inhibitor, or C1 esterase inhibitor concentrate.

12. Should I treat HAE and drug-induced angioedema in the same way?

In known C1 esterase deficiency (HAE), C1 esterase concentrate is treatment of choice rather than FFP; theoretically, FFP may worsen the symptoms because HAE is a complement-mediated process. If the cause is unknown or thought to be drug induced and C1 esterase inhibitor is unavailable, 2 to 4 units of FFP should be administered. Please also note there are other recently approved agents used to treat HAE, such as ecallantide and icatibant, although availability may be limited in the ED setting.

13. Summarize the initial treatment for life-threatening forms of anaphylaxis.

- 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.
- 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 IM. 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 dextrose 5% in water (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 or metaproterenol.
- 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.
- For patients in full cardiac arrest, administer 1:10,000 epinephrine, 1 mg slow IV push or 2 to 2.5 mg epinephrine diluted in 10 mL NS 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 non–cardiac arrest situation.

14. 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 IV, 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, 50 to 150 μg/kg IV over 1 minute followed by 1 to 5 mg/hr IV infusion may be helpful in patients resistant to epinephrine who are on long-term β-adrenergic blocking agents, such as propranolol. Corticosteroids have limited benefit because of the delayed (4 to 6 hours) onset of action but may be beneficial in patients with prolonged bronchospasm or hypotension.

15. What percentage of children who require IM epinephrine require a second dose?

20%

16. 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.

17. What is biphasic anaphylaxis? How common is it?

Biphasic anaphylaxis is 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 later. 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 as follows:

- 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

18. 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 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.

19. 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. A single dose of hydrocortisone (250 to 1000 mg IV) or methylprednisolone (125 to 250 mg IV) should be administered.

20. 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 a minimum of 2 to 4 hours. Patients who experience rebound or continue to have life-threatening symptoms (e.g., bronchospasm, hypotension, or upper airway obstruction) should be admitted.

21. 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 epinephrine 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, and should be taken concomitant with the administration of IM epinephrine.

22. Is there an advantage of IM over subcutaneous epinephrine injection?

Yes, if it is 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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QUESTIONS

1. What is the weight limit for administering 0.15 mg of IM epinephrine (EpiPen Jr) versus the adult dosage of 0.3 mg IM epinephrine?
   a. 20 kg
   b. 30 kg
   c. 50 kg
   d. 60 kg
   The correct answer is *b.*
2. What special consideration must be given to patients with anaphylaxis who are taking long-term β-blockers?
   a. Higher doses of epinephrine must be given
   b. Give glucagon if there is limited response to epinephrine
   c. IV epinephrine is preferred over IM epinephrine
   d. These patients are more predisposed to hypoglycemia
   The correct answer is *b.*
3. What is the most common medication that causes anaphylaxis?
   a. Nonsteroidal antiinflammatory drugs (NSAIDs)
   b. Opioids
   c. Penicillin
   d. Radiocontrast media
   The correct answer is *c.*

LOW BACK PAIN

Ryan A. Pedigo, MD, and Robert S. Hockberger, MD

CHAPTER 21

1. Can I skip this chapter?

Not if you anticipate a career that involves caring for adults. Low back pain (LBP) is the most common musculoskeletal complaint leading to an ED visit. The vast majority (up to 85%) of individuals will have LBP at some point in their lives; one in four adults report having LBP in the past 3 months. It is the most common cause of activity limitation in people younger than 45 years of age and the third most common cause in people older than age 45 (after heart disease and arthritis). The cost of diagnosis, treatment, disability, lost productivity, and litigation as a result of 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 common causes of LBP?

Idiopathic back pain is also referred to as *musculoskeletal back pain, acute lumbosacral sprain, lumbago,* and other similar terms; importantly, there is no specific identifiable anatomic injury when patients have these diagnoses. Although most patients will feel better within 4 weeks, the majority will also have a recurrence within 1 year. Patients with idiopathic LBP have asymmetric pain in the paraspinal muscles of the lumbar spine that is worse with activity.

Lumbar disk herniation is LBP that often radiates down the leg in the area served by a compressed and irritated nerve. Herniations that occur at L4-5 or L5-S1 cause pain that radiates down the lateral and posterior aspects of the leg, respectively, termed *sciatica.* Nerve root impingement can also be a result of other etiologies, such as spinal stenosis or more life-threatening etiologies such as malignancy or abscess (see Question 3). The challenge in emergency medicine is finding the three or four patients out of 100 who have emergent causes of LBP and treating them aggressively, while preventing excessive imaging and testing in patients who do not have features indicative of a serious cause of their pain.

3. What are the emergent causes of LBP?

Although almost all causes of acute back pain are self-limited and benign, there are a few life-threatening diagnoses that should always be considered and ruled out by a thorough history and physical examination (Table 21-1).

Epidural abscess (or other spinal infection such as vertebral osteomyelitis) should be considered in high-risk patients with back pain and fever. Risk factors include immunocompromised states (e.g., HIV/AIDS, alcoholism, diabetes), older age (most commonly ages 60 to 70 years), intravenous drug use, and recent spinal surgery. Emergent magnetic resonance imaging (MRI) is the diagnostic test of choice.

Ruptured abdominal aortic aneurysm (AAA) should be considered in older individuals who have LBP and risk factors for atherosclerosis. A contained AAA rupture can cause significant back pain and be a sentinel event to a fatal rupture. An AAA is defined as an aortic diameter over 3 cm, with the risk of rupture increasing with size. ED bedside ultrasound can quickly and accurately evaluate for this diagnosis

Cauda equina syndrome occurs when there is compression of the cauda equina, typically around the L1 area in adults. This is usually from a herniated disk but can be caused by anything that impinges on the distal spinal canal (e.g., metastatic disease, epidural abscess, hematomas). The classic presentation is severe LBP, associated saddle anesthesia, bilateral lower extremity weakness, and urinary incontinence/retention. The most sensitive finding is urinary retention, so a postvoid residual (PVR) test should be checked in individuals for whom the diagnosis is considered (with either urinary catheterization or ultrasound). A PVR over 100 mL is abnormal, and anything over 300 mL is markedly abnormal. The negative predictive value of a normal PVR for ruling out cauda equina syndrome is nearly 100%. Emergent MRI is the diagnostic test of choice.

Abstract

This chapter discusses the evaluation and management of patients who come to the ED with low back pain (LBP). It discusses common causes of LBP, highlights red flag features (signs and symptoms) that should prompt further evaluation, and recommends a minimalist, cost-efficient approach to management in the absence of red flags.

Keywords:

low back pain (LBP), lumbar disc herniation, vertebral osteomyelitis, vertebral metastases, vertebral fracture, straight leg raise (SLR) test, erythrocyte sedimentation rate (ESR)

Table 21-1. Differential Diagnosis of Low Back Pain

MECHANICAL SPINE DISORDERS	NONMECHANICAL SPINE DISORDERS	VISCERAL DISEASE
Lumbar strain	Malignancy	Abdominal aortic aneurysm
Degenerative disk/facet disease	Multiple myeloma	Pelvic organs
Herniated disk	Metastatic cancer	PID
Spinal stenosis	Spinal column or cord cancer	Prostatitis
Spondylolysis	Lymphoma	Renal disease
Spondylolisthesis	Infection	Pyelonephritis
Congenital spinal disease	Septic discitis	Nephrolithiasis
Traumatic fracture	Osteomyelitis	Gastrointestinal disorders
Osteoporotic compression fracture	Epidural abscess	Pancreatitis
	Shingles	Penetrating ulcer
	Inflammatory arthritis	Cholecystitis

PID, Pelvic inflammatory disease.

Spinal fracture should be considered in individuals who have a history of antecedent blunt trauma before the onset of pain. There should be a low threshold to image (with radiography or computed tomography [CT]) patients who have LBP after significant blunt trauma. In individuals with risk factors for osteoporosis (e.g., elderly, bedridden, chronic corticosteroid use), the risk of a fracture is much higher; there is a 10-fold increase in fracture risk for individuals taking corticosteroids for a chronic condition.

Malignancy should be suspected in individuals with LBP who have known cancer or experience worsening back pain that lasts longer than 4 weeks, often is worse at night, does not respond to routine analgesics, and is associated with recent weight loss. Further testing to identify the primary site of malignancy should be undertaken; malignancies that commonly metastasize to bone include prostate, breast, kidney, thyroid, and lung (Pb KTL, or "lead kettle").

4. How should I focus my history?

The following historical features, termed *red flag features*, should be investigated routinely to assess for an emergent cause in all patients who have a chief complaint of LBP (Table 21-2).

- Radiation of pain suggests radiculopathy from a disk herniation or mass lesion impinging on a nerve root.
- Aggravation of pain in the back and calves with walking (termed *pseudoclaudication*) and alleviation of pain with bending forward suggests spinal stenosis. Pseudoclaudication can be distinguished from claudication caused by peripheral vascular disease by the alleviation with bending forward (which widens the spinal canal and relieves stenosis) and from the duration of pain after rest, which is typically longer (e.g., 15 minutes with spinal stenosis versus 5 minutes with vascular disease).
- A history of recent trauma should raise concern for spinal fractures.
- A history of malignancy or symptoms consistent with malignancy (e.g., pain at night, persistent chronic worsening pain, unexplained weight loss) makes metastatic disease more likely.
- Immunocompromised patients (diabetes, HIV/AIDS, chronic steroid use) and those with fever are at risk for epidural abscesses.
- Elderly patients and those taking steroids for chronic conditions are at higher risk for fractures, even with minor trauma.
- Neurologic symptoms, such as urinary retention, saddle anesthesia, or bilateral leg numbness, cause concern for cauda equina syndrome.

5. How should I focus my physical examination?

All patients with LBP should receive a complete neurologic examination, focusing on lower extremity strength, sensation, and reflexes (Table 21-3). Mechanical spine disorders, with the exception of herniated lumbar disks or severe spondylolisthesis, should not compromise neurologic function. Red flag features of the physical examination include fever, midline spinal tenderness, and significant

Table 21-2. Red Flag Features of Low Back Pain

RED FLAG FEATURES	POSSIBLE CAUSE	IMAGING
Age >50 years	Fracture, malignancy	LS spine radiography
Trauma	Fracture	LS spine radiography
Fever, intravenous drug use, recent infection	Infection	MRI
Unexplained weight loss, history of cancer	Metastases	LS spine radiography
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 radiography
Immunosuppression or steroid use	Fracture, infection	LS spine radiography, MRI, or CT
Midline spinal tenderness	Fracture, infection, malignancy	LS spine radiography

CT, Computed tomography; *LS*, lumbosacral; *MRI*, magnetic resonance imaging.

Table 21-3. 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

neurologic deficits, including saddle anesthesia (see Table 21-2). A straight leg raise (SLR) test should be performed to assess for sciatica, but a positive result is not necessarily a red flag sign. An abdominal examination is important to assess for visceral disease, including an AAA, and rectal tone and sensation should be assessed if there is any concern for cord compression.

6. 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, commonly (but not always) radiates distal to the knee, usually is associated with a positive SLR test, and may be associated with neurologic impairment.

7. 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 results in pain that radiates down the involved leg past the knee; 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. Dorsiflexion of the foot at the point of pain should worsen the pain, and plantar flexion should alleviate it.

8. What imaging or laboratory testing should be routinely performed?

None. In the absence of any red flag features (see Table 21-2), imaging is estimated to change clinical decision making only once for every 2500 studies performed. Despite popular belief, studies have shown that routine imaging does not improve patient reassurance or decrease anxiety, but it does increase patient radiation exposure, ED length of stay, and health care costs. If concerning features are present, appropriate imaging should be ordered (e.g., MRI for cord compression, cauda equina, or epidural abscess; radiography or CT for bony pathology such as fractures). When spinal infection or malignancy is suspected, an erythrocyte sedimentation rate (ESR) should be obtained. An elevated ESR (usually greater than 60 to 80 mm/h) 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). Patients often come to the ED expecting imaging, so explaining early on that imaging is unnecessary for uncomplicated back pain, and only increases their bill and radiation exposure, is important to ensure that they understand why imaging is not being performed.

9. What should I know about children who come to the ED with back pain?

Back pain is rare in children. LBP that interferes with activities previously enjoyed by a child may be indicative of a serious underlying pathologic condition. Spondylolysis and spondylolisthesis resulting from 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 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. AAA
2. Cauda equina syndrome
3. Lumbar disk herniation with severe neurologic compromise
4. Spinal malignancy
5. Spinal infection (e.g., vertebral osteomyelitis, epidural abscess)

11. How should patients with LBP be treated in the ED?

Quickly. There is no need to await definitive diagnosis before providing pain relief. Oral or parenteral nonsteroidal antiinflammatory drugs (NSAIDs) and application of superficial heat are first-line agents. Parenteral narcotics may be necessary to provide adequate analgesia in patients with severe discomfort.

12. Who should be hospitalized for treatment?

Patients with LBP may require hospitalization to expedite evaluation and treatment when an emergent cause of LBP (e.g., epidural abscess, cauda equina) is suspected or when patients experience severe pain requiring continued administration of parenteral analgesics.

13. How should patients with musculoskeletal LBP be treated as outpatients?

Bed rest is not recommended for patients with acute LBP in the absence of acute disc herniation associated with severe pain. In general, patients who remain active recuperate faster and

experience less disability 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 cost, side effects (e.g., 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 do not 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, or develop any of the red flag features previously mentioned.

15. What happens to patients with LBP when they leave the ED?

The prognosis for patients experiencing a first episode of idiopathic or 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 underlying 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. However, recurrence rates are also high, and patients should be educated that this is likely to be a continuing problem for them in the future. Performing exercises that strengthen the abdominal and back muscles (after the acute pain has subsided), avoiding activities that twist and torque the back, and maintaining good cardiovascular health may decrease the incidence and severity of LBP recurrences.

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QUESTIONS

1. A 35-year-old male who uses IV heroin has had malaise, tactile fevers, and atraumatic LBP for the past day. His vital signs are significant for a heart rate of 110 beats/min and a temperature of 37.9°C. Examination shows midline tenderness over T3 but a normal neurologic examination. An ESR is 80 mm/h, and a CT of the abdomen and pelvis with IV contrast is normal. What is the next step in management?
   a. Perform an MRI
   b. Reassurance, NSAIDs, and a 1-week follow up visit with his primary medical doctor
   c. Perform lumbar spine radiography
   d. Perform an abdominal ultrasound to rule out an AAA

   The correct answer is *a.* The diagnostic test of choice in evaluating for an epidural abscess is an MRI. This patient has multiple risk factors for an epidural abscess (low-grade temperature, history of IV drug use, localized midline tenderness), and a negative CT does not have sufficient negative predictive value to rule out the diagnosis. Reassurance, *b,* would be inappropriate. Because the patient has already received a CT scan, ***c*** would be inappropriate, and *d* is inappropriate because the patient is 35 years old (although if he had a history of a connective tissue disease or early AAA, it would be reasonable).
2. A 60-year-old male with a history of metastatic renal cell carcinoma is experiencing LBP. What is the most sensitive finding when evaluating for cauda equina syndrome?
   a. Decreased rectal tone
   b. Increased PVR volume
   c. Saddle anesthesia
   d. Paralysis of the bilateral lower extremities

   The answer is *b.* Urinary retention (rather than incontinence) is the most sensitive finding in cauda equina syndrome. Although the other answer choices can be found in cauda equina syndrome, they are not as sensitive.
3. Which patient should undergo imaging on their first ED visit for LBP?
   a. A 40-year-old female who has LBP after lifting with right paraspinal tenderness in the lumbar spine
   b. A 30-year-old male with lupus, who is taking prednisone and has midline lumbar spinal tenderness after a bicycle accident but has a normal neurologic examination
   c. A 45-year-old female who complains of severe LBP radiating down the posterior aspect of her right leg into her ankle with a normal neurologic examination but a positive straight leg test. She has required three doses of morphine for her pain.
   d. A 45-year-old male with a history of basal cell carcinoma removal from the face in 2008, who has atraumatic LBP with bilateral paraspinal tenderness in the lumbar spine

   The answer is *b,* a patient taking steroids for a chronic condition and who has undergone blunt trauma. Chronic corticosteroid use greatly increases the risk of fracture in this patient, especially because he has midline spinal tenderness. Radiographs are not indicated in the other patients; note that in choice *d,* basal cell carcinoma essentially never is metastatic and its mention is a distractor.

CHAPTER 22

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 distance 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, a positive accommodative test, which is simply pain precipitated by accommodation, is suggestive. Pain with either maneuver suggests ciliary spasm.

2. What typical findings help with the differential diagnosis of the red eye?

See Table 22-1.

3. What is conjunctivitis?

Conjunctivitis is inflammation of the bulbar and palpebral conjunctivae. Viral conjunctivitis is usually bilateral with clear tearing and may be associated with an upper respiratory infection (URI). A preauricular lymph 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 sign) (Fig. 22-1).

Bacterial conjunctivitis initially may be unilateral with purulent 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 sulfacetamide drops alone or in combination with trimethoprim (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?

Endophthalmitis is 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 involving posterior eye structures. Both tend to be unilateral. Orbital cellulitis is most often the result of direct spread from ethmoid sinusitis or pansinusitis, whereas periorbital cellulitis often follows trauma, bites, or foreign body.

7. How do I differentiate clinically between periorbital and orbital cellulitis?

The two may be difficult to distinguish clinically, especially in children. Periorbital (preseptal) 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.

Abstract

This chapter discuss eye complaints that are often seen in the ED, some of which, such as sudden loss of vision, are true emergencies. The diagnosis and treatment of common eye disorders are discussed in some detail.

Keywords:

red eye, conjunctivitis, iritis, orbital cellulitis, periorbital cellulitis, cavernous sinus thrombosis, central retinal artery occlusion, glaucoma, afferent pupillary defect (APD), subconjunctival hemorrhage

Table 22-1. 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 (haloes)
Pain	Gritty	Moderate	Severe
Conjunctival injection	Diffuse with limbic sparing	Perilimbic	Perilimbic
Cornea	Clear	Keratotic precipitates	Steamy or hazy
Pupil size	Normal	Constricted or dilated	Fixed and dilated
Pupillary light response	Normal	Poor and painful (+ consensual photophobia)	Poor or none if fixed
Intraocular pressure	Normal	Normal	Elevated

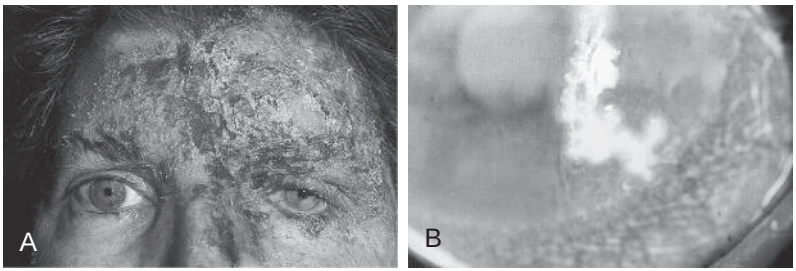

Figure 22-1. A, Ocular zoster, suggested by involvement of the nasociliary branch of V_1, manifested by lesions on the tip of the nose (Hutchinson sign). **B,** Classic herpes simplex dendrite staining brightly with fluorescein. *(**A** from Kanski JJ:* Clinical ophthalmology: a synopsis, *New York, 2004, Butterworth-Heinemann; **B** from Reeves SW, et al: Corneal infections. In Vander JF, editor:* Ophthalmology secrets, *ed 3, St. Louis, 2007, Elsevier Mosby, Fig. 8-11, p 97.)*

Orbital (postseptal) 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 (divisions 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. The mnemonic *POTOMAC* can be remembered for the following structures that traverse the cavernous sinus:

Pituitary
Ophthalmic branch of the trigeminal nerve (V_1)
Trochlear nerve (IV)
Oculomotor nerve (III)
Maxillary branch of the trigeminal nerve (V_2)
Abducens nerve (VI)
Carotid artery

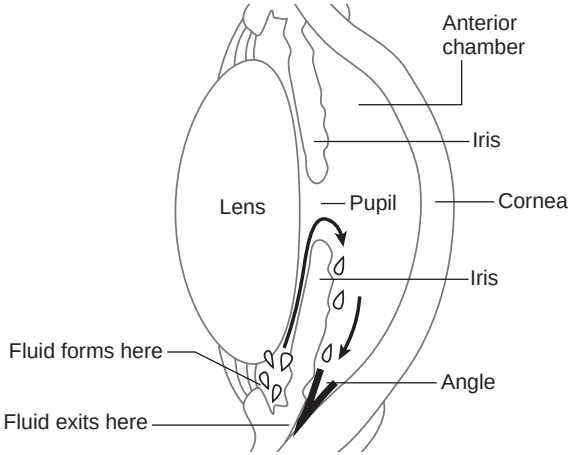

Figure 22-2. Cross section of the eye.

In thrombosis, 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 exhibit perilimbic injection or "flush," ciliary spasm, and a constricted miotic pupil. A consensual photophobia can be a clue to iritis as the etiology of unilateral injection, versus conjunctivitis, which will only cause direct photophobia. Because both may exhibit photophobia, bilateral iritis can be 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, pupillary dilation or mydriasis (e.g., from reduced illumination) causes the thickened iris to abut the lens, preventing adequate aqueous humor drainage through the pupil. Once the pressure rises high enough, the outflow tract (angle of Schlemm canal) is also narrowed, preventing drainage and quickly elevating globe pressures (Fig. 22-2). 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 headache.

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. Definitive management involves creating a hole or bypass tract through the iris for drainage (laser iridotomy).

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 taking anticoagulant medication 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, which does not pass the corneal edge. 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 historically are not recommended as outpatient therapy. However, consensus is evolving regarding short-course therapy for uncomplicated corneal abrasions. Nonsteroidal antiinflammatory drugs, such as ketorolac or diclofenac, are useful for pain relief. Topical corticosteroids should generally be used only 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 have a pupil that is dilated as a result of taking a medication. If 1% pilocarpine fails to constrict the pupil, it is pharmacologically blocked, most commonly by phenylephrine, a scopolamine patch (if it has been handled), or aerosolized anticholinergics/β-agonists. Other causes of a unilateral dilated pupil include posttraumatic 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 agent. 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 to 6 hours) or 2% to 5% homatropine (1 to 2 days); never use atropine (1 to 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, shine a light toward the normal eye, and after several seconds swing it 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
5. Retinal detachment

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 syndrome
2. Argyll-Robertson pupil
3. Adie pupil
4. Posttraumatic or medication-induced mydriasis
5. Third nerve palsy

Table 22-2. Common Causes of Nontraumatic Loss of Vision

Transient Monocular
Amaurosis fugax
Temporal arteritis
Migraine
Persistent Monocular or Binocular
Central retinal artery occlusion
Central retinal vein occlusion
Retinal detachment or hemorrhage
Vitreous or macular hemorrhage
Optic or retrobulbar neuritis
Macular degeneration
Acute Binocular
Migraine
Vertebral basilar insufficiency
Cerebrovascular disease
Toxins (e.g., methanol, salicylates, quinine)
Hysteria
Malingering

19. What are common causes of a miotic pupil?

The two most common causes of a miotic pupil are Horner syndrome and an Argyll-Robertson pupil. The clinical manifestations of Horner syndrome include ptosis, miosis, and anhidrosis (in a cold ED, check for dilated conjunctival vessels). Bronchogenic carcinoma, stroke, and brachial plexus pathology may present with Horner 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 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 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 22-2.

22. Describe the presentation and treatment of central retinal artery occlusion 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 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 partial pressure of carbon dioxide (pCO_2) (e.g., using a 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 diabetic patients with an obscured red reflex and retinal details. Nontraumatic retinal detachments are more common in patients with significant myopia. Patients

Table 22-3. 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

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 30s, and there is a 40% association with current or future diagnosis of 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 22-3). 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.

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QUESTIONS

1. A 32-year-old female comes to the ED with painful vision loss in the left eye. On examination you find a normal cornea with photophobia, blurred optic disc margins, presence of spontaneous venous pulsations, and decreased visual acuity. What is the next step in management?
   a. Ocular massage and CT angiogram of brain
   b. CT of the head for suspected intracranial mass
   c. Intravenous steroids, and consult ophthalmologic and neurologic specialists
   d. Timolol, acetazolamide, and consult an ophthalmologist
   The correct answer is *c.* This patient has signs indicative of optic neuritis. Steroid therapy should be initiated early, along with consultation with ophthalmologic and neurologic specialists.
2. A 40-year-old contact lens wearer has eye pain and blurry vision. On examination you find a grayish area in the central cornea that has fluorescein uptake. How should the patient be treated?
   a. Tetracaine drops and a follow-up visit with the primary care physician
   b. Erythromycin ointment and a follow-up consultation with an ophthalmologist
   c. Sulfacetamide with trimethoprim drops, cool compresses, follow-up care as needed
   d. Moxifloxacin drops, follow-up visit to an ophthalmologist the next day
   The correct answer is *d.* The patient has a corneal ulcer. With the history of contact lenses, therapy should be aggressive. Topical fluoroquinolone therapy with moxifloxacin should be given, and the patient should have urgent ophthalmologic follow-up care.
3. What is a possible complication of pupillary dilation, and a reason this is rarely done by an emergency physician?
   a. Acute angle-closure glaucoma caused by obstruction of Schlemm canal
   b. Inconvenient duration of photophobia
   c. Discomfort with mydriatics
   d. Cost of medication
   The correct answer is *a.* Pupillary dilation, via relaxation of the iris muscle and obstruction of the Schlemm canal, can induce acute narrow-angle glaucoma. Rarely should an emergency provider dilate the pupil of a patient with a shallow anterior chamber.

CHAPTER 23

NONTRAUMATIC EAR, NOSE, AND THROAT EMERGENCIES

Dowin Boatright, MD, MBA, and Christopher Davis, MD

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 common causes. Less commonly seen causes include foreign bodies; tumors; coagulopathies; use of anticoagulant drugs such as aspirin, clopidogrel, 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 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 the *Kiesselbach plexus* or *Little 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 age 50 years. The hemorrhage tends to be more severe, with patients often swallowing large amounts of blood.

4. List the key questions to ask the patient.

- Is there a prior history of nosebleeds?
- Is there a history of excessive alcohol use or bleeding dyscrasias?
- Was trauma involved? Was nose picking?
- On which side did the bleeding start?
- Have there been any recent sinus infections or surgeries?
- Is there warfarin, clopidogrel, direct thrombin inhibitor, 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 23-1). While obtaining the history and quickly assessing airway, breathing, and circulation (ABCs), have the patient pinch the nose firmly (bilateral nasal, 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.

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

Abstract

This section discusses nontraumatic ear, nose, and throat (ENT) pathology, including epiglottitis, epistaxis, otitis externa, mastoiditis, and esophageal foreign body.

Keywords:

sinusitis, mastoiditis, epistaxis, epiglottitis, otitis externa, esophageal foreign body

Table 23-1. 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 (Afrin)	Antibiotic ointment
4 × 4 gauze	0.25% phenylephrine (Neo-Synephrine)	Foley catheter or commercial balloon
		Rolled 4 × 4 gauze with silk suture

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(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 the Kiesselbach 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 and inserting it horizontally back into the nostril, along the floor of the nasal cavity. Once in place, moisten the packing 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. The literature demonstrates an initial failure rate (defined as a recurrence of symptoms in 7 days) for chemical cautery and nondissolvable packing to be 20% and 23% respectively.

The use of topical tranexamic acid to control epistaxis was compared with anterior nasal packing in a small, randomized control trial. In this study, 500 mg of tranexamic acid was dissolved in 5 mL of saline and applied topically to the nasal mucosa. This approach resulted in quicker hemostasis, shorter visits, and higher patient satisfaction. Although this technique is promising, further external validation is necessary.

7. Any 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?

- The pack (any type) should be left in place for 2 to 3 days.
- Treat each patient who has packing with prophylactic antistaphylococcal antibiotics (cephalexin or trimethoprim-sulfamethoxazole) while the pack is in place to prevent sinusitis or toxic shock syndrome. Sinusitis may occur because the paranasal sinuses cannot drain properly with a pack in place.
- Any recurrent epistaxis that fails to respond to direct firm pressure for 10 minutes should be seen in the ED.
- 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 catheter 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 catheter in this position with an umbilical clamp placed just outside the nose. Because the catheter will be stretched a bit, place gauze between the nose and the 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 (ear, nose, and throat; 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 consultation, 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, patients in critical condition 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 should be ordered.

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 cotton swab or small balloon-tipped catheter can do also do the trick. Using an aural speculum when guiding the swab will prevent adherence of glue to the external auditory structures.

15. What symptoms do patients with nasal foreign bodies show?

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 is 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 face mask 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 meglumine diatrizoate (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% lidocaine. Objects that are visualized may be removed with bayonet forceps or a Kelly clamp. Smooth objects, such as coins, in the esophagus for fewer than 24 hours can be removed by placing the patient in the Trendelenburg position (head down), passing a Foley catheter beyond the object, expanding the balloon, and withdrawing the catheter. Because of potential complications, this procedure is best performed with the use of 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 to 2 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, owing to gas formation, are associated with esophageal perforation. Sharp objects should be removed endoscopically.

KEY POINTS: ESOPHAGEAL FOREIGN BODIES

1. In the patient with the sensation of an esophageal foreign body, esophagoscopy should be considered with persistent symptoms or an uncertain diagnosis.
2. Because glucagon commonly elicits vomiting, it may cause esophageal perforation and it should not be used.

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 they are localized to the esophagus. Otherwise, their location in the GI system should be monitored with serial radiography until elimination is confirmed.

KEY POINTS: NATURAL HISTORY OF GI FOREIGN BODIES

1. Of foreign bodies, 80% to 90% pass through the GI tract without significant problems.
2. The following often require surgical removal: sharp or long (>6.5 cm) objects, disk or button batteries, and items that have not migrated as seen 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 for 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 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 for 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 having medical emergencies.

Locally, mucoceles and osteomyelitis can develop. Orbital complications are the most common, 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 finger-stick glucose level 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), maxillary sinus tenderness, and worsening of symptoms after improvement.
2. A single Water 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 pain
- Hoarseness or muffled ("hot potato") voice

Signs
- Lymphadenopathy
- Drooling
- Respiratory distress
- Extreme pain with palpation of the larynx

29. What is the thumbprint sign?

The thumbprint sign is 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 β-hemolytic streptococci. In most cases, no organism is found, pointing to a viral cause. With the introduction of the *H. influenzae* type B (Hib) vaccine in children, the reservoir for *H. influenzae* has decreased dramatically so that epiglottitis is now seen more often 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 β-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, partial pressure of carbon dioxide (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. An otic suspension of polymyxin B, neomycin sulfate, and hydrocortisone (Cortisporin) works well because it has antibacterial, antiinflammatory, and drying properties, as well as a nontoxic pH.

Additionally, unlike Cortisporin solution, neomycin and polymyxin B sulfates and hydrocortisone otic 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. Topical ciprofloxacin, a second-generation fluoroquinolone antibiotic, also has demonstrated efficacy in the treatment of otitis externa. Ciprofloxacin has an excellent safety profile without evidence of ototoxicity. Studies have shown clinical efficacy in 95% of cases when administered topically. Additionally, the combination of ciprofloxacin with fluocinolone, a corticosteroid, has been shown to be more effective in the treatment of otitis externa than ciprofloxacin alone or Cortisporin with fluocinolone.

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 and is 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 the combination of benzocaine, butamben, and tetracaine hydrochloride (Cetacaine). A needle cover should be cut to provide a needle guard for an 18-gauge needle, exposing no more than 1 cm of the needle. The guarded needle is inserted at the most fluctuant portion of the abscess. If available, ultrasound with an endocavitary probe can help identify location of the abscess during drainage. In a randomized control trial of patients with suspected peritonsillar abscess, a negative ultrasound for

abscess was associated with no complications at 2 days. The same study showed that ultrasound reduces the need for ENT referral (7% versus 50%) and CT scanning (0% versus 35%). Occasionally, trismus can be so pronounced that ultrasound guidance is impossible. In situations like this, consider using a video laryngoscope to improve visualization. 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. There is some evidence to suggest that patients with a peritonsillar abscess who receive 10 mg IV dexamethasone have decreased pain at 24 hours, and are able to more quickly return to normal activities and dietary intake.

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 emergent nonsurgical and surgical airway procedures.
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.

ACUTE MASTOIDITIS

43. What is mastoiditis?

Mastoiditis is a suppurative infection of the mastoid air cells. Mastoiditis occurs when the thin, bony septae between air cells are destroyed by bacteria. Acute mastoiditis is classified by having symptoms present for less than 1 month.

44. How do I make the diagnosis?

Acute mastoiditis is characterized by ear pain, erythema and swelling over the mastoid, and displacement of the auricle. Of patients with acute mastoiditis, 80% have an associated acute otitis media. Laboratory results, including white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) level, may be elevated but have low utility in making the diagnosis. Whereas the diagnosis is clinical, imaging of the temporal bone helps define the stage of mastoiditis, which ultimately guides treatment. Contrast-enhanced CT of the temporal bone is the preferred imaging modality.

45. What are the complications?

Mastoiditis can lead to facial nerve palsy, hearing loss, labyrinthitis, osteomyelitis, neck abscess, meningitis, venous sinus thrombosis, and epidural empyema.

46. How do I treat mastoiditis?

Treatment will require intravenous antibiotics such as ceftriaxone or cefotaxime to provide coverage against *S. pneumonia, S. pyogenes,* and *S. aureus.* Antipseudomonal coverage is required if the patient has a history of recurrent otitis media or recent antibiotic therapy. If there is evidence of complications, an ENT specialist should be consulted for possible tympanostomy tube placement and mastoidectomy.

Acknowledgment

The editors gratefully acknowledge the contributions of Danielle Raeburn, MD, and Katherine Bakes, MD, authors of this chapter in previous editions.

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QUESTIONS

1. Which of the following regarding epiglottitis is false?
   a. Fever, pain with swallowing, and sore throat are common complaints.
   b. Lateral neck films have a sensitivity greater than 90% for the detection of epiglottitis.
   c. In most cases of epiglottitis, no bacterial etiology is found.
   d. Direct visualization is the gold standard for diagnosis of epiglottitis.

   The correct answer is *b.* Lateral neck films have poor sensitivity for the detection of epiglottitis. No bacterial etiology is found for most cases of epiglottitis, and direct visualization remains the gold standard for diagnosis. Most patients show some combination of fever, pain with swallowing, and sore throat.
2. Which of the following is true regarding epistaxis?
   a. Most cases of posterior epistaxis are safe for discharge.
   b. Patients with anterior epistaxis and hypertension should have aggressive blood pressure management.
   c. Local trauma is the most common cause of anterior epistaxis.
   d. The international normalized ratio (INR) is useful for guiding treatment in most cases of anterior epistaxis.

   The correct answer is *c.* Local trauma causes the majority of cases of anterior epistaxis. Patients with posterior epistaxis are almost never safe to discharge, and many will require ICU admission. Neither aggressive blood pressure control nor an INR is needed to manage most cases of anterior epistaxis.
3. Concerning a peritonsillar abscess, each of the following is true except:
   a. Smokers, males, and those with periodontal disease are at an increased risk for peritonsillar abscess.
   b. Needle aspiration and antibiotics are the treatment of choice for most patients.
   c. Ultrasound reduces the need for ENT referral in patients with a peritonsillar abscess.
   d. The uvula is typically midline in a peritonsillar abscess.

   The correct answer is *d.* The uvula is typically deviated away from the side of a peritonsillar abscess. Smoking, male sex, and periodontal disease have been associated with risk for peritonsillar abscess. Ultrasound has been shown to decrease the need for ENT referral among patients with peritonsillar abscesses, and needle aspiration and antibiotics are the treatment of choice.

CHAPTER 24

DENTAL AND ORAL SURGICAL EMERGENCIES

Colin T. Galbraith, DMD, and Mark J. Glasgow, DDS

1. **For what conditions should I emergently consult the dental team versus the oral surgery team? Which other conditions require urgent follow-up care (24 to 48 hours)?**
   See Table 24-1.

2. **What are the important anatomic structures of the orofacial region?**
   Important structures coursing through the orofacial region include the cranial nerves, major and minor salivary glands and their ducts, muscles of mastication and facial expression, and numerous blood vessels and lymph nodes. Teeth may be present in various states of repair, depending on the patient's age and history of oral hygiene and dental restorations. Healthy gingiva and mucosa should appear pink in color without edema, erythema, or bleeding, although patients with dark complexion may have splotchy areas of dark pigmentation The submandibular and sublingual glands should be palpable on the floor of mouth and submandibular region.

3. **How are teeth numbered?**
   Different tooth numbering systems exist, including the Universal, Palmer, and ISO systems. In the United States, the Universal system is the most common numbering method (Figs. 24-1 and 24-2), but the ISO system is commonly used as well. When communicating with consulting services or other facilities, it is helpful to clarify which tooth is being discussed (e.g., tooth 11, the upper left canine).

4. **How should I examine the orofacial region?**
   Use bright lighting. Palpate the neck for any masses or lymphadenopathy. Perform a standard cranial nerve examination. Look for facial asymmetry or injury. Examine the lips, inner cheeks, and gums. Dentures and orthodontic retainers should be removed. Retract the upper and lower lips until taut to expose the depths of the maxillary and mandibular vestibules. Use a tongue depressor to evaluate the lingual vestibules, floor of the mouth, and ventral surface of the tongue. Inspect the soft and hard palate, as well as the tonsils, uvula, and oropharynx. The uvula and soft palate should rise symmetrically. Palpate the teeth, mandible, and maxilla for mobility and pain. Gingival lacerations or bruising may be signs of underlying fractures or dental trauma. Retract the cheeks and ask the patient to bite his or her teeth together to evaluate the dental occlusion.

5. **How do you examine the temporomandibular joint (TMJ)?**
   Palpate the TMJ approximately 1 cm anterior to the tragus. Ask the patient to open and close their mouth to rule out trismus and assess any clicking, popping, or crepitus. Normal mouth opening ranges from about 40 to 60 mm, measured between the incisal edges of the central incisors. Watch for deviation of the mandible on opening or closing. Ask the patient to move the mandible as far left and right as possible. Palpate the masseter and temporalis muscles to check for myofascial pain or trigger points.

6. **How do I assess open TMJ lock?**
   Open and closed TMJ locks are typically caused by articular disc dislocation. You must first determine whether the condition is acute or chronic, because some patients have limited function for months or years. Imaging the joints with plain film radiographs or computed tomography (CT) scan can evaluate the condition of the mandibular condyle and glenoid fossa, which is especially useful when ankylosis is suspected. Magnetic resonance imaging (MRI) is useful for evaluating the articular disc and its movement during opening and closing movements. In the acute setting, open lock is typically caused by condylar dislocation anterior to the articular eminence. If the dislocation is unilateral, the patient's chin will be deviated away from the affected side.

Abstract

Dental and oral surgical problems are commonly encountered in the ED. This chapter discusses in some detail their evaluation and treatment.

Keywords:

dental, oral, teeth, mouth, face, temporomandibular joint (TMJ) dislocation

Table 24-1. Guidelines for Managing Oral and Facial Consultations

EMERGENT DENTAL CONSULTATION*	EMERGENCY ORAL SURGERY CONSULTATION	URGENT DENTAL OR ORAL SURGERY FOLLOW-UP CARE
Fractured, avulsed, or luxated teeth	Uncontrollable bleeding from the oral cavity, face, head, or neck	Parotid or other salivary gland swelling
Alveolar housing fractures	Open facial fractures including frontal sinus, orbital, zygomatic, maxillary, nasal, and mandibular fractures	Lost or fractured dental restorations or dentures
Infections within the oral cavity	Cellulitis or abscesses involving the oral cavity, face, head, and neck (e.g., Ludwig's angina)	Chronic dental conditions such as gingivitis, periodontitis, exposed bone, or soft-tissue lesions
Refractory pain or bleeding from surgery site or dental extraction	Noma	TMJ pain, clicking, or popping
Gingival lacerations	Facial lacerations involving facial nerves, uncontrolled arterial bleeding, muscles of mastication and facial expression, the parotid, or submandibular, sublingual or salivary glands	Loose or broken dental implant
Failed reduction of an open or closed lock of the TMJ	Disease processes that require a surgical airway	ANUG or ANUP
		Sexually transmitted diseases of the oral cavity
		Natal or neonatal teeth

ANUG, Acute necrotizing ulcerative gingivitis; *ANUP*, acute necrotizing ulcerative periodontitis; *TMJ*, temporomandibular joint.
*Severe or unusual conditions should be elevated directly to the oral surgery service.

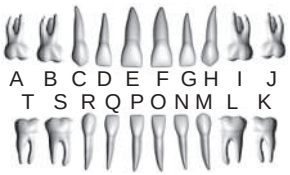

Figure 24-1. Primary teeth, Universal numbering system.

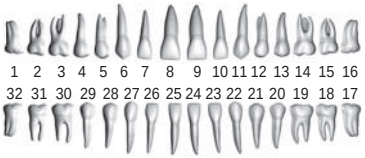

Figure 24-2. Permanent teeth, Universal numbering system.

7. How do you treat open TMJ lock?

Treatment of open lock requires either local anesthesia into the TMJ or deep sedation. Place your thumbs over the posterior mandibular teeth or posterior alveolar ridge, with your fingers beneath the inferior border of the mandible. Gauze padding should be used to prevent laceration of your thumbs from sharp teeth. Press downward and backward on the posterior teeth with upward pressure underneath the patient's chin. After reduction, limited mouth opening should be enforced for 2 to 4 weeks with either an elastic head wrap or Ivy loops with maxillomandibular elastics. Soft diet, nonsteroidal antiinflammatory drugs (NSAIDs), and warm compresses are encouraged. Recurrent open lock may require surgical intervention.

8. How do I examine the parotid gland and parotid duct?

Visually inspect for asymmetry, swelling, erythema, and cutaneous fistulas. Palpate the parotid gland for masses, crepitus, pain, fluctuance, and asymmetry. Verify cranial nerve VII function. Locate the Stensen duct inside the cheek, adjacent to the first maxillary molar, and dry the area with gauze. While gently retracting the cheek, palpate the parotid gland from posterior to anterior, and you should see clear saliva emerge from the duct. Blood is a sign of injury, whereas pus is a sign of

sialadenitis. To determine whether the duct has been lacerated, cannulate the Stensen duct with a flexible IV catheter; then gently inject up to 1 mL of sterile milk, propofol, or methylene blue. If the liquid emerges from the site of laceration, the duct has been compromised.

9. What are some causes of parotid swelling?

- Bacterial or viral infection (e.g., paramyxovirus, Epstein-Barr virus, cytomegalovirus)
- Salivary gland tumors
- HIV parotitis
- Trauma (edema, hematoma, or sialocele)
- Salivary stones
- Autoimmune diseases (e.g., Sjögren syndrome)
- Sarcoidosis
- Wegener granulomatosis
- Chronic recurrent parotitis
- Pneumoparotid (from wind instruments, coughing, or dental work)
- Kimura disease
- Lymphoma
- Radiation sialadenitis
- Polycystic disease

10. Which sensory nerves innervate the orofacial structures, and how can they be anesthetized?

The maxillary and mandibular branches of the trigeminal nerve (cranial nerves V2 and V3) provide sensation of the teeth, gingiva, mucosa, anterior two thirds of the tongue, and the skin of the midface and lower face. The glossopharyngeal nerve (cranial nerve IX) provides sensation for the posterior two thirds of the tongue, tonsils, and pharynx. Local infiltration can be used to anesthetize most of the orofacial structures; however, nerve blocks are useful for anesthetizing large areas and for distancing the anesthetic administration from the site of injury or infection. Most of the terminal nerve branches of cranial nerves V2 and V3 can be blocked, including the nasopalatine nerve, greater and lesser palatine nerves, inferior alveolar nerve, lingual nerve, long buccal nerve, mental nerve, and the posterior/middle/anterior superior alveolar nerves.

11. How are dental injuries treated?

For all tooth injuries, instruct the patient to eat a soft diet for 2 weeks and to follow up with a dentist as soon as possible. The dentist will monitor the tooth vitality and radiographic appearance over time for evidence of pulpitis, necrosis, resorption, and ankylosis. Contaminated wounds and avulsed teeth that are reimplanted should be treated with tetanus prophylaxis. Reposition and reimplant teeth with the appropriate local anesthetic. Obtain a panoramic radiographic unless a CT scan is indicated. Fractured teeth may require root canal therapy and dental restorations as an outpatient.

12. What is a dental concussion and how is it treated?

Dental concussions occur when an impact to a tooth does not result in displacement or mobility of the tooth, but the periodontal ligament may be injured and the tooth's vitality may be compromised. The tooth may be sensitive to palpation or percussion. No immediate treatment is required.

13. What is a subluxation, and how is it treated?

Subluxation occurs when an injured tooth is mobile but without displacement from its original position. Bleeding from the gingival sulcus is a common finding. Depending on the degree of mobility, a splint can be applied to stabilize the tooth for 1 to 2 weeks or the tooth can be left untreated.

14. What is luxation of a tooth, and how is it treated?

Luxation occurs when a tooth is displaced in a buccal or lingual direction. Alveolar housing or root fractures are likely. Rinse the socket and exposed root surface with sterile saline; then grasp the tooth with gauze and reposition it back into the socket. Reducing the tooth may require pulling the tooth slightly out of the socket in order to redirect the root apex around the fractured bone. Splint the tooth for 4 to 6 weeks to stabilize the tooth and the alveolar housing fracture. Immediate extraction is indicated only for those teeth deemed hopeless or considered an aspiration risk.

15. What is intrusion of a tooth, and how is it treated?

Intrusion is the displacement of the tooth into the socket. Intruded teeth will often spontaneously erupt over time, depending on the depth to which they were intruded and the developmental stage of the root apex. When primary teeth are intruded, they require early extraction if the developing permanent tooth bud is disrupted. Otherwise, no immediate treatment is required. The patient's dentist will decide between spontaneous eruption versus orthodontic or surgical repositioning.

16. What is extrusion of a tooth and how is it treated?

Extrusion is the displacement of a tooth partially out of its socket. Rinse the exposed root surface with sterile saline and press back it into the socket. Stabilize the tooth with a splint for 1 to 2 weeks.

17. How is an avulsed tooth treated by emergency medical services (EMS) on scene?

Avulsion, or complete displacement of a tooth out of its socket, requires rinsing the tooth and reimplanting it into the socket. Handle the tooth only by the crown, avoiding contact with the root surface. If the tooth is not reimplanted at the scene, possible transport media, in preferential order, include Hanks balanced salt solution, milk, sterile saline, or saliva. Avoid placing the tooth into tap water, juice, or soda. Primary teeth should not be reimplanted. Teeth reimplanted within the first 30 minutes have the best prognosis; any tooth out of the socket for more than 2 hours has a poor prognosis and should not be reimplanted.

18. How should I treat avulsion of a tooth in the ED?

Examine the tooth for evidence of root or crown fracture. If the tooth is intact and has been out of the socket for less than 60 minutes, rinse the tooth and its socket with sterile saline. Use manual pressure to reimplant the tooth and splint it for 1 to 2 weeks. If the tooth has been out of the socket for more than 60 minutes, gently remove any attached soft tissue with gauze, reimplant the tooth into the socket, and then splint it for 2 to 4 weeks. Administer antibiotics for 1 week: doxycycline twice daily if the patient is older than 12 years of age, and penicillin VK four times a day if the patient is younger than age 12 years. If the tooth or a portion of the tooth was not found at the scene, consider a chest radiograph to rule out aspiration. Teeth may also be swallowed or displaced into the nasal cavity, maxillary sinus, tongue, or lips.

19. What are the tooth fracture classifications, and how are they treated?

- Ellis class 1: Fracture is through enamel only, and no immediate treatment is needed (Fig. 24-3).
- Ellis class 2: Fracture is through enamel and dentin only, and there is no exposed pulp. If the patient complains of sensitivity, apply a dentinal sealant or glass ionomer.

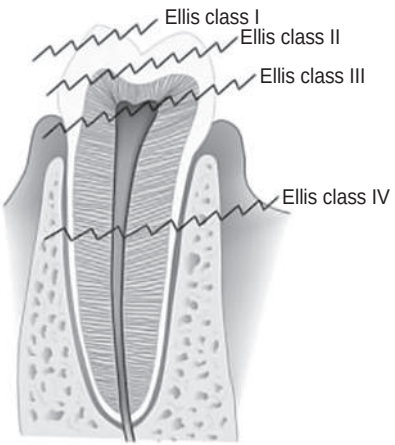

Figure 24-3. Ellis classifications of dental fractures.

- Ellis class 3: Fracture is through enamel and dentin with exposed pulp. Apply calcium hydroxide to the exposed pulp and dentin.
- Ellis class 4: Fracture is through the tooth root. Splint the tooth for 1 to 2 weeks if the fracture is in the middle or apical thirds of the root. The tooth will need extraction if the fracture is in the coronal third.

20. What are the signs of maxillary and mandibular fractures?

- Mobility
- Crepitus
- Gingival lacerations
- Bleeding from the gingival sulcus
- Ecchymosis
- Open bite
- Cross bite
- Premature tooth contact
- Step-off deformity between teeth
- Trismus
- Paresthesia or anesthesia
- Pain to palpation

Studies show the ability to bite into and crack a wooden tongue depressor has a negative predictive value greater than 90% for mandible fractures and a positive predictive value around 65%.

21. What imaging should be ordered for known or suspected facial fractures?

Panoramic radiographs are often adequate for simple, isolated mandible fractures. A plain-film mandible series (includes Towne view, anteroposterior view, and right and left lateral oblique views) can help visualize displaced segments. CT scans are indicated for complex fractures of the mandible or suspected fractures of the midface, orbits, skull, or cervical spine. CT-angiography (CTA) should be considered for patients at risk for a cerebrovascular injury based on the mechanism and clinical findings.

22. What is an alveolar housing fracture?

An alveolar housing fracture is a fracture through the alveolar bone of the mandible or maxilla that surrounds and supports the teeth, often associated with tooth luxation or fracture. Signs include gingival lacerations, ecchymosis, and mobility of the alveolus. When multiple teeth are displaced and mobile *en bloc,* there is a high likelihood of an alveolar housing fracture. Consider panoramic radiography or CT of the face. Irrigate gingival lacerations and exposed bone with sterile saline. Manually reduce the displaced alveolus and any luxated teeth; then splint the affected teeth for 4 to 6 weeks. Some fractures may require open reduction internal fixation or maxillomandibular fixation. Repair gingival lacerations with 3-0 or 4-0 chromic gut or Vicryl sutures. Nondisplaced fractures may not require any surgical treatment. Instruct patients to avoid chewing with the affected segment for 6 weeks.

23. How are mandible fractures classified?

Mandible fractures are classified by anatomic location, including symphysis, parasymphysis, body, ramus, coronoid process, subcondylar, condylar neck, condylar head, and alveolar housing (Fig. 24-4). They are further described by the fracture pattern as simple, linear, nondisplaced, displaced, mobile, nonmobile, greenstick, comminuted, pathologic, monocortical, or bicortical.

24. Which fractures require antibiotics?

Open fractures of the maxilla and mandible require antibiotics. Open fractures include any fracture that communicates through a laceration of the skin, mucosa, or gingiva, as well as any fracture that extends through the socket of an erupted tooth. Prescribe penicillin VK 500 mg four times a day for 1 week, or clindamycin 300 mg four times a day for patients allergic to penicillin. Prescribe children weight-based doses of amoxicillin or clindamycin.

25. How are odontogenic abscesses treated?

Incision and drainage with removal of the offending tooth or root canal therapy are the desired treatments. In the ED, the incision and drainage may be performed at bedside if the abscess is in an accessible location, with the patient discharged to home on antibiotics and a follow-up appointment for extraction or root canal. Severe or deep space infections may require drainage in the operating room.

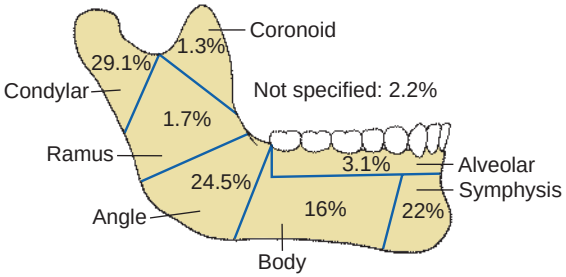

Figure 24-4. Anatomic distribution of mandibular fracture sites. *(From Hupp JR, Ellis E, Tucker MR:* Contemporary oral and maxillofacial surgery*, ed 5, St. Louis, 2008, Elsevier Mosby, Fig. 24-11, p 499.)*

26. Which spaces are typically involved with infections of odontogenic origin?

Infections originating from the maxillary teeth typically perforate through the thin buccal plate to the maxillary canine and buccal spaces, but they may also involve the palate, the preseptal space around the eye, the infratemporal space, the parapharyngeal space, or paratonsillar spaces. Infections originating from the mandibular teeth spread to the buccal and sublingual spaces, extending into the submental, submandibular, sublingual, and masticator spaces. Severe infections may spread to the parapharyngeal, retropharyngeal, pretracheal, or prevertebral spaces, and down to the mediastinum.

27. What are the indications for admitting odontogenic infections?

Indications include impending airway compromise, sepsis, dehydration, inability to tolerate foods or liquids by mouth, uncontrolled pain, and deep space involvement.

28. What is alveolar osteitis?

Alveolar osteitis, or dry socket, is delayed healing and inflammation of the bony tooth socket after dental extraction. The condition is likely caused by fibrinolytic lysis and subsequent premature loss of the blood clot. Symptoms typically develop 3 to 4 days after the extraction and include pain, foul taste, and foul smell. Mandibular third molar sockets are most commonly affected. The condition is not associated with infectious signs such as erythema, fever, swelling, or purulence.

29. How is alveolar osteitis treated?

No antibiotics are indicated, and treatment is palliative. The socket should first be irrigated with sterile saline to dislodge any food debris. Do not curette the socket. Medicated dressings containing eugenol can be placed into the socket for temporary relief. Nonresorbable dressings must be replaced every 2 days until symptoms improve. The condition is usually self-limited to about 2 weeks with or without treatment.

30. What is Ludwig's angina?

Ludwig's angina is cellulitis with or without abscess formation of the submental, submandibular, and sublingual spaces bilaterally. Signs and symptoms may include

- Severe swelling
- Erythema extending down to the chest
- Elevated and/or protrusive tongue
- "Hot potato" voice
- Dysphagia
- Dysphonia
- Dyspnea
- Crepitus
- "Tripoding"
- Stridor
- Inability to tolerate secretions
- Airway deviation or collapse on CT

Treatment includes securing the airway, emergent incision and drainage of all involved spaces, extraction of the offending tooth, and antibiotics.

31. What is Lemierre syndrome?

Lemierre syndrome is septic thrombophlebitis of the internal jugular vein, usually resulting from paratonsillar or lateral pharyngeal infection. The resulting septic emboli may cause cavernous sinus thrombosis or travel to the heart, lungs, liver, bone, and joints.

32. What are some causes of gingival bleeding and potential treatments?

Bleeding is caused by:

- Trauma
- Periodontal disease
- Postoperative wound
- Thrombocytopenia
- Vitamin deficiency
- Blood dyscrasias
- Leukemia

First-line treatment of intraoral bleeding is direct gauze pressure and exploration to determine the source. Extraction sockets can be sutured with or without topical agents such as Gelfoam, Surgicel, and topical thrombin. Other treatments include electrocautery or chemical cautery, vessel ligation, vitamin K, tranexamic acid, and transfusion with fresh frozen plasma or platelets. Interventional radiology can be used for angiography and embolization of intractable or deep space hemorrhaging.

33. Describe the process for closing perioral and intraoral lacerations.

Close lacerations only after manipulating alveolar fractures or displaced teeth to avoid disrupting the wound closure.

- Anesthetize the area with local anesthesia, typically 1% or 2% lidocaine with 1:100,000 epinephrine.
- Use pulse irrigation and a soft brush or gauze to remove debris. Perform conservative surgical debridement with a scalpel or scissors to remove devitalized tissue and foreign bodies, but keep tissue removal to a minimum because of the prolific vascularity of the face and mouth.
- Achieve hemostasis with manual pressure, electrocautery, silver nitrate, or vessel ligation.
- Explore the wound to its depth to determine the complexity of the laceration and to inspect the underlying bone for fractures. Remove foreign bodies, such as tooth fragments and gravel.
- Close wounds in layers from inside to outside (e.g., from deep to superficial and from intraoral to extraoral). Lacerations of the gingiva and mucosa can usually be closed as a single layer with resorbable sutures such as 3-0 or 4-0 Vicryl or chromic gut. (Small lacerations up to about 1 cm in the mucosa, gingiva, and tongue do not require closure.)
- Irrigate full thickness lip or cheek lacerations again after mucosal closure.
- Take care to reapproximate the vermillion border of the lips to avoid a noticeable step-off.
- 5-0 or 6-0 nonresorbable, monofilament sutures should be used for skin closure, including the "dry" mucosa of the lips.
- Remove skin sutures 5 to 7 days after placement.

34. How do I close a wound if tissue has been avulsed?

In general, these types of wounds are beyond the expertise of most emergency medicine physicians, and they should be referred to an oral or plastic surgeon.

35. How do you treat animal bites to the orofacial region?

Irrigate all wounds with copious sterile saline under pressure. Because of the high vascularity of the face, small puncture wounds can be left to heal secondarily or closed primarily to reduce the risk of scarring. Larger lacerations and tissue avulsions should be repaired as described in Questions 33 and 34. Determine the type of animal bite and rabies status. Rabies vaccination should be considered for wild bats, raccoons, skunks, and unknown dogs. Administer tetanus vaccine or immune globulin as indicated. The need for antibiotic prophylaxis for dog bites is controversial, because only 5% will otherwise become infected. When given, antibiotics should cover *Pasteurella multocida* (amoxicillin with clavulanic acid, or for patients allergic to penicillin, cefuroxime or doxycycline). Unlike dog bites, all cat bites should be given antibiotic prophylaxis, because 80% will otherwise become infected. Infecting organisms include *P. multocida, Bartonella henselae,* and *Clostridium tetani*. Water fowl, reptiles, amphibians, and mammals each carry a unique flora of bacteria and viruses and should be treated appropriately. Human bites are at risk for *Pseudomonas* and *Streptococcus* infection, as well as viruses such as HIV, hepatitis B, and hepatitis C. Animal hoof

injuries may contaminate the wound with pathogens from the ground or barnyard, such as *Escherichia coli, Clostridium,* and *Staphylococcus*.

36. What is noma and how is it treated?

Noma, or cancrum oris, is an opportunistic, polymicrobial infection that causes destruction of the lips, cheeks, mucus membranes, and facial bones. Caused by bacterial infection, the disease is found primarily in patients with malnutrition, dehydration, immunocompromise, poor oral hygiene, and/or unsanitary living conditions. The disease typically progresses rapidly and without pain, but its effects are disfiguring, debilitating, and permanent. Treatment involves surgical debridement, nutrition, and empiric penicillin treatment.

37. Patients taking bisphosphonate medications are at risk of developing what intraoral condition?

Bisphosphonate-related osteonecrosis of the jaws (BRONJ), defined as any area of exposed maxillary or mandibular bone that has persisted for more than 8 weeks in patients with a history of bisphosphonate use without a history of radiation to the area. IV bisphosphonates (e.g., zolendronate, pamidronate, ibandronate) carry the highest risk, but PO bisphosphonates (e.g., alendronate, risedronate) and antiresorptive medications such as denosumab have also been implicated. The exposed bone may develop spontaneously or after trauma to the oral mucosa or gingiva (including tooth extraction). Initial treatment includes analgesics and antibacterial mouth rinse, but as the lesion progresses, drug holiday, debridement, antibiotic therapy, and surgical resection may be required. Avoiding intraoral trauma in patients with a history of these medications is the best method of prevention.

KEY POINTS

1. Press downward and backward on the posterior teeth with upward pressure underneath the patient's chin to reduce open temporomandibular joint (TMJ) lock.
2. To determine if the parotid duct has been lacerated, cannulate the Stensen duct with a flexible IV catheter; then gently inject up to 1 mL of sterile milk, propofol, or methylene blue.
3. When primary teeth are intruded, they require early extraction if the developing permanent tooth bud is disrupted; otherwise, no immediate treatment is required.
4. If an avulsed permanent tooth is not reimplanted at the scene, possible transport media, in preferential order, include Hanks balanced salt solution, milk, sterile saline, or saliva.
5. Studies show the ability to bite into and crack a wooden tongue depressor has a negative predictive value greater than 90% for mandible fractures and a positive predictive value around 65%.
6. Ludwig's angina is cellulitis with or without abscess formation of the submental, submandibular, and sublingual spaces bilaterally.

Acknowledgment

The editors and author of this chapter would like to acknowledge and thank Drs. Richard D. Zallen and Valerie Byrnside for their previous contributions to this chapter.

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QUESTIONS

1. A father calls the emergency department stating that his 10-year-old daughter's front tooth was just knocked out after a fall from a bunk bed. The tooth landed on the hardwood floor and appears to be intact. Before taking his daughter to the ED or dentist's office for splinting, what should the father do to increase the tooth's prognosis for survival?
   a. Place the tooth into a cup of apple juice.
   b. Rinse the tooth briefly in water and immediately reimplant it into the socket.
   c. Wipe the root of the tooth dry with paper towels.
   d. Nothing. 10-year-old children never have permanent central incisors, and primary teeth should never be reimplanted.

   The correct answer is *b.*
2. A 34-year-old homeless male comes to the ED after three days of dysphagia, dysphonia, and tooth pain. While the patient is trying to describe his symptoms, you notice he has a protruding tongue, audible stridor, and difficulty tolerating his secretions. On examination the patient has erythema, crepitus, and firm edema of the submandibular and submental regions extending down to his upper chest. His pulse is 107, BP 104/71, T38.4, RR 28, and SpO_2 90% on RA. What immediate steps should be taken?
   a. Intravenous dexamethasone, fluids, and bedside suction
   b. Nasopharyngoscopy to evaluate edema of his larynx
   c. CT scan of the head and neck with and without contrast, upright anteroposterior and lateral chest films, blood culture
   d. Secure airway, begin empiric antibiotics and fluids, consult oral and maxillofacial surgery department, prepare for transportation to operating room

   The correct answer is *d.*
3. A 21-year-old female unrestrained driver is brought to the ED after crashing her car at high speed into a retaining wall. The patient is intoxicated, combative, and has significant bleeding coming from a full-thickness lower-lip laceration that penetrates down to the bone and extends laterally 6 cm toward the left angle of the mandible. After securing her airway and sedating the patient, you begin to examine the laceration. All of the following structures are at risk of injury from this laceration except:
   a. The mental nerve
   b. The supratrochlear nerve
   c. The marginal mandibular branch of cranial nerve VII
   d. The facial artery
   e. Tooth 21.

   The correct answer is *b.*

CHAPTER 25

TRANSIENT ISCHEMIC ATTACK AND CEREBROVASCULAR ACCIDENT

Richard Byyny, MD, MSc

1. What is a cerebrovascular accident (CVA) or stroke?

Stroke is a sudden spontaneous impairment of blood flow to a region of the brain, caused by either vascular occlusion or rupture. 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?

There are two major classes of stroke: ischemic and hemorrhagic. Ischemic stroke causes 90% of all CVAs in the United States, and hemorrhagic stroke accounts for the remaining 10%. Unfortunately, the timing and duration of neurologic dysfunction does not indicate which type of stroke the patient is experiencing; therefore rapid evaluation is critical in these patients. The critical diagnostic study to differentiate between the two is a noncontrast computed tomography (CT) of the head.

3. What are the causes of ischemic stroke?

- Thrombotic
  - Atherosclerosis
  - Vasculitis
  - Small vessel disease
- 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 types of hemorrhagic stroke?

These can be classified as either intracerebral hemorrhage (ICH), which causes approximately 70% of hemorrhagic strokes, or subarachnoid hemorrhage (SAH), which is responsible for approximately 30% of hemorrhagic strokes.

5. What are the causes of ICH?

The most common etiologies of ICH are anticoagulants, hypertension, bleeding disorders, amyloid angiopathy, illicit drug use (usually sympathomimetics), and vascular malformations.

6. What is the most common cause of SAH?

Cerebral aneurysms are the most common cause of SAH, accounting for approximately 80%. However, SAH may also be caused by arteriovenous malformations and vertebral artery dissection. A small proportion are caused by perimesencephalic hemorrhage, a type of bleeding that does not have a known source and almost universally has a benign course.

7. What are some of the potential mimics of acute stroke?

Disorders that mimic stroke include:

- Postictal Todd palsy
- Hypoglycemia

Abstract

This chapter provides an overview of transient ischemic attacks, hemorrhagic and thrombotic stroke, and the recognition and treatment of these conditions.

Keywords:

stroke, transient ischemic attack (TIA), cerebrovascular accident (CVA), tissue plasminogen activator (tPA), subarachnoid hemorrhage (SAH), head computed tomography (CT), National Institute of Neurological Disorders and Stroke (NINDS), European Cooperative Acute Stroke Study (ECASS), thrombolysis

- Complex migraine
- Conversion disorder
- Bell palsy
- Acute spinal cord compression
- Brain tumor
- Systemic infection
- Multiple sclerosis

8. What is the definition of a transient ischemic attack (TIA)?

The classic definition of TIA has been based on time (i.e., <24 hours of symptoms), although 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 cut-off point can reliably determine whether underlying ischemic infarction has occurred, in 2009 the American Heart Association and American Stroke Association (AHA/ASA) transitioned to a tissue-based description of TIA (i.e., transient symptoms with lack of tissue injury confirmed by neuroimaging).

9. Why should I be concerned about a TIA?

TIAs are associated with a high risk of early acute stroke (up to 10% within the first 2 days).

10. Are there prognostic scoring systems that can be used to determine the risk associated with a TIA?

There are several scoring systems that can be used to prognosticate the patient's subsequent risk of having an ischemic stroke. One of the more widely studied and used prognostic scores is the $ABCD^2$ score (Table 25-1). The results of the score can help predict the likelihood of having an infarct in the next 48 hours. Evidence suggests that rapid evaluation and initiation of preventive measures within 24 hours may significantly reduce the risk of recurrent stroke.

11. Do patients with suspected TIA have to be admitted to the hospital?

Although many hospitals admit patients with TIA, there are an increasing number that use ED-based observation units to accomplish the workup. Some hospitals even have outpatient-based TIA clinics to rapidly perform the necessary diagnostic studies and initiate care.

12. 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 25-1. $ABCD^2$ 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 (2-day stroke risk)	1 point
High: Total 6-7 pts (8.1% risk)	
Moderate: 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.

13. How do I approach a patient with acute stroke symptoms?

The patient should be evaluated immediately. As with all patients who have acute conditions, airway, breathing, and circulation (ABCs); intravenous (IV) access; oxygen; and monitoring are the critical first steps. 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 (BPs) are important to recognize; however, immediate reductions are unnecessary and should not distract the provider from other aspects of the patient's care. The final management will depend on whether the stroke is hemorrhagic or ischemic. Many hospitals have dedicated stroke protocols and stroke teams. It is important to be familiar with your institution's practice.

14. Why is the time of onset an important historical factor 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 strokelike symptoms, then the last time the patient was awake and normal is considered the time of onset. In patients who are unable to communicate effectively, using the prehospital personnel for history and timing can be extremely valuable.

15. What bedside tests should be performed on patients with suspected CVA?

The patient should have a finger-stick glucose test performed. Hypoglycemia is a well-known cause of neurologic symptoms and can cause focal neurologic deficits mimicking a stroke. Hypoglycemia and hyperglycemia are contraindications to thrombolytic therapy.

16. What laboratory tests should be performed on patients with suspected CVA?

Whereas no laboratory testing can confirm a CVA, a number of tests are important to assess for other causes of the patient's symptoms and to help evaluate the patient's eligibility for therapy. A basic metabolic panel can help assess for electrolyte derangements that are known to cause neurologic symptoms, such as hyponatremia. A complete blood count can be helpful to evaluate the platelet count in patients with hemorrhagic stroke. Studies of coagulation can help guide or exclude patients from therapy in both hemorrhagic and ischemic stroke.

17. What imaging test should be performed on patients with suspected CVA?

The most important early imaging test is noncontrast CT of the brain. This helps with one of the first branch points of therapy, which is distinguishing hemorrhagic stroke from ischemic stroke. Some hospitals use emergent MRI to evaluate patients with possible stroke, but this is much less common.

KEY POINT: IMAGING IN STROKE

Rapidly obtaining a noncontrast CT of the head should be a priority in working up the patient with suspected CVA.

18. What historical factors are typical for SAH?

The most well described complaint is that of a "thunderclap" headache or sudden onset of the worst headache a patient has ever had. Up to 15% of patients with sudden onset of the worst headaches of their lives have SAH. However, patients with SAH can have seizure, syncope, depressed mental status, or even focal neurologic deficits. It is important if the patient complains of headache to assess whether the headache is new or different for the patient. Additionally, it is helpful to ask for the timing between headache onset and maximal intensity.

19. What is the sensitivity of noncontrast CT of the head for SAH?

The literature is evolving with regard to the sensitivity of noncontrast CT as the technology for CT scanning improves. It is also dependent on who is reading the imaging; however, a reasonable estimate is 95% within 24 hours, 80% at 48 hours, 70% at 72 hours, and 50% at 5 days. It is important to note that the literature is consistent in demonstrating a decreasing sensitivity of head CT from headache onset. A more recent study has suggested that if the time from headache ictus to CT scan is 6 hours or less, the sensitivity is as high as 100%; however, this result has not been replicated yet.

20. If the noncontrast head CT is negative, what is the next step in a caring for patient with suspected SAH?

The literature is evolving with regard to the answer to this question as well; however, most experts still recommend a lumbar puncture. The reasoning is based upon the fact that the noncontrast head CT lacks 100% sensitivity, and the risk for missing one patient with SAH could be catastrophic. There has been a recent Bayesian analysis of the data to suggest that CT angiography of the brain can be used to assess for aneurysmal SAH (the most common type of SAH). The absence of aneurysms on CT angiography would reduce the risk of aneurysmal bleeding and effectively rule out SAH. However, this practice has yet to be prospectively studied.

21. 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 source in those with neck trauma (including even minor trauma) or cervical spine fractures, young patients (<45 years old), and those with neck pain.

22. 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.

23. What role do prehospital personnel play in patients with suspected stroke?

Besides stabilization of the acute condition, prehospital personnel 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.

24. What medications should be started in the ED for acute stroke?

This depends on the type of stroke. Hemorrhagic stroke therapy is centered on reducing hemorrhage. Ischemic stroke therapies are centered on restoring the flow in blocked vessels.

25. What is the appropriate time frame from symptom onset to administration of systemic thrombolytics?

This is sometimes called the *thrombolytic window*. Initial literature suggested that the maximum window was 3 hours. However, the publication of European Cooperative Acute Stroke Study (ECASS) III in 2008 and the on-going SITS-ISTR trial have extended the potential window for systemic thrombolytics from 3 hours to 4.5 hours.

KEY POINTS: RAPID TREATMENT OF ISCHEMIC STROKE WITH TISSUE PLASMINOGEN ACTIVATOR

1. Door to physician ≤10 minutes
2. Door to stroke team ≤15 minutes
3. Door to CT initiation ≤25 minutes
4. Door to CT interpretation ≤45 minutes
5. Door to drug (≥80% compliance) ≤60 minutes
6. Door to stroke unit admission ≤3 hours

26. Do systemic thrombolytics save lives in patients with possible stroke?

There has never been shown to be a mortality benefit for systemic administration of tissue plasminogen activator (tPA) in patients with suspected CVA.

27. What is the evidence for tPA in acute ischemic stroke?

Alteplase (also called *tPA*) is the only thrombolytic currently approved by the U.S. 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 (modified Rankin scale) at 3

months if given within 3 hours of symptom onset, with a number needed to treat (NNT) of 6. In 2008, ECASS III showed similarly improved functional outcome within the 3- to 4.5-hour timeframe (NNT, 14).

28. What is the risk of tPA?

The primary risk of tPA is systemic bleeding, particularly ICH. For the NINDS trial, ICH with tPA was 6.4% versus 0.6% for the nontreatment group or a number needed to harm (NNH) of 17. For ECASS III, ICH with tPA was 2.4% versus 0.2%, or NNH of 45 (7.9% versus 3.5%, or NNH of 23 if the 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. Angioedema may occur in 1% to 5% of patients.

29. 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.

30. What are the indications and contraindications for tPA?

The indications and contraindications are presented in Table 25-2. It is important to note that the NINDS trial, the ECASS III trial, and the AHA guidelines all vary slightly in their inclusion and exclusion criteria. Once again, be familiar with the criteria used in your institution's stroke protocol.

31. Why is there controversy with tPA for acute ischemic stroke?

The controversy is multifactorial. Some providers are legitimately concerned about the potential for ICH that can occur with tPA therapy and the lack of mortality benefit. Proponents note that obtaining an independent functional outcome is an important outcome, especially given the burden of

Table 25-2. 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 previous 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; *ECASS*, European Cooperative Acute Stroke Study; *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.

ischemic stroke in the United States. Additionally, critics note that the original NINDS trial may have had skewed results because, in spite of randomization, there was a difference in stroke acuity between the treatment arms. The NINDS trial recruited half of its subjects within 0 to 90 minutes from symptom onset, which may not be generalizable to everyday ED practice, in which the majority of patients often will not fall into this time window. There is concern that widespread community use will have different results, although part of this difference likely results from protocol violations, an argument supporting rigorous adherence to stroke protocol.

KEY POINTS: MORTALITY AND MORBIDITY OF TPA

1. tPA does not appear to confer a mortality benefit.
2. Patients appear to have improved functional outcomes.
3. There are serious risks associated with major bleeding complications.

32. Is informed consent required before tPA administration?

Informed consent requirements will vary depending on institutional protocols. The practice of shared decision making, however, is important. If possible, the risks, benefits, and alternatives should be explained and confirmed to be understood by the patient or a surrogate decision maker. In a patient whose current functional state impairs assessing his or her understanding and no surrogate decision maker is available, it is valid to administer tPA without a formal consent. If the patients is able to refuse, it is important to explain the outcomes in that scenario as well. In community observational studies, the reported range of ICH in patients treated within 3 hours is still 0.5% to 1%. Furthermore, improved functional outcome can occur without therapy (NINDS, 26% in placebo arm; ECASS III, 45%).

33. 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.

KEY POINTS: SUCCESSFUL ADMINISTRATION OF TPA

1. Adhere to your institution's stroke alert protocol.
2. Be exact about the time of onset and document it.
3. Immediately consult with a neurologist.
4. Expedite the head CT and final radiology reading.
5. Send labs early (i.e., finger-stick glucose, complete blood count [CBC], prothrombin time [PT]/partial thromboplastin time [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.

34. Are there alternatives to systemic tPA for acute ischemic stroke?

Intraarterial thrombolysis and mechanical clot disruption remain investigational and are often limited to academic institutions. Intraarterial 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.

35. How should I manage ICH in the setting of tPA?

Consider new ICH if the patient has a sudden neurologic decline, new headache, nausea or vomiting, and sudden BP rise within 24 hours. In this case you should immediately stop the tPA, perform a noncontrast head CT, and send for laboratory reports (i.e., type and cross match, PT, PTT, platelets, fibrinogen). There are no prospectively collected data on the therapy of ICH after administration of tPA; however, there are expert opinions provided in guidelines which recommend the following:

- 10 units cryoprecipitate
- 6 to 8 units of platelets (or one single donor unit)
- Neurosurgical consultation for possible hematoma evacuation

36. What are the indications for aspirin therapy in the patient with acute ischemic stroke?

All patients with acute ischemic stroke who are not candidates for tPA, have no evidence of associated ICH, and have no other contraindications for aspirin (e.g., allergy) should receive 324 mg of aspirin. In the International Stroke Trial, administration of aspirin within 48 hours reduced 14-day recurrent stroke from 3.9% to 2.8%.

37. What are the indications for heparin in ischemic stroke?

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 heparin early in patients with embolism 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.

38. How do I approach hypertension in the patient with acute ischemic stroke?

Permissive systemic hypertension may be critical to maintaining cerebral perfusion, and aggressive lowering may result in clinical deterioration. Most experts recommend lowering BP slowly by 15% only if systolic BP (SBP) is 220 mm Hg or greater or diastolic BP (DBP) is 120 mm Hg or greater. The important exception is when thrombolytic therapy is being considered. In this setting, one to two dosages of labetalol IV 10 to 20 mg over 1 to 2 minutes are allowed if SBP is greater than 185 mm Hg or DBP is greater than 110 mm Hg.

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, you should defer to recommendations obtained during consultations, but a modest reduction of elevated BPs is reasonable to consider in this setting.

39. What should I do to treat the patient who is taking anticoagulant drugs and who has hemorrhagic stroke?

Approximately 12% to 14% of patients with hemorrhagic stroke will be on oral anticoagulants. In general, for patients taking traditional oral anticoagulants, it has been recommended to reverse the effects of the medication completely by using both vitamin K and fresh frozen plasma (FFP). More recently, plasma-derived factor concentrates (prothrombin complex concentrates, PCCs) have been introduced. They have been shown to more rapidly normalize an abnormal international normalized ratio (INR); however, no mortality or morbidity benefit has been found to date. For patients with specific defects, such as hemophilia, the factor deficiency should be completely reversed, and those patients with thrombocytopenia should receive platelets.

40. What about patients taking novel oral anticoagulants (NOACs)?

More recently, pharmaceutical manufactures have released direct thrombin inhibitors and direct factor Xa inhibitors. Emergency medicine practitioners have been faced with the challenge of treating patients with life-threatening hemorrhages who are taking these medications. Typical studies of coagulation, such as the PTT/PT and INR, may not accurately reflect the degree of anticoagulation in these patients. Unfortunately, there are yet to emerge high-quality randomized trials to support specific strategies of reversal. However, PCCs may have some efficacy, as does hemodialysis. Other antifibrinolytic agents, such as tranexamic acid or aminocaproic acid, can also be considered. Some institutions may have access to testing with thromboelastogram to direct the administration of reversal therapies. Because this is an evolving area of treatment, it is wise to use a team-based approach to management, which may include neurosurgeons, hematologists, and representatives from the pharmacy and the blood bank.

KEY POINTS: MANAGEMENT OF ICH

1. Reverse any anticoagulants or antiplatelet agents.
2. Administer antiepileptic drugs.
3. Control mean arterial pressure (MAP).
4. Position the head of the bed.
5. Control intracranial pressure (ICP).

Acknowledgment

The editors and author of this chapter would like to acknowledge and thank Dr. Michael M. Liao for his previous contributions to this chapter.

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QUESTIONS

1. Which of the following therapies is not helpful in ischemic stroke?
   a. Heparin
   b. Aspirin
   c. tPA
   d. Warfarin
   e. None of the above
   The correct answer is *a*.
2. True or false: A noncontrast CT of the head can rule out SAH?
   The correct answer is *false*.
3. Which of the following is not a contraindication for tPA?
   a. Any history of ICH
   b. Bleeding ulcer 14 days ago
   c. Major surgery in the last 14 days
   d. Extremity trauma to any long bone
   e. Serious head trauma in the last 3 months
   The correct answer is *d*.
4. Which is true about TIA?
   a. TIAs and stroke can be distinguished clinically.
   b. A patient who has symptoms for 60 minutes is not at increased risk of stroke.
   c. Patients with TIA do not need a cranial imaging.
   d. Patients with TIA have an increased risk of stroke.
   The correct answer is *d*.

CHAPTER 26

MENINGITIS

Maria E. Moreira, MD

1. What is meningitis, and why is it important?

Meningitis is an inflammatory disease of the tissues surrounding the brain and spinal cord. The mortality rate 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 26-1.

3. Which organisms are most commonly involved in each age group?

See Table 26-2.

4. Who is at risk for meningitis?

Those aged older than 60 years and younger than 5 years 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
- 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

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 sign: Flexion of the neck results in flexion of the knees and hips

Abstract

This chapter provides an overview of meningitis, including presentation, diagnostic workup, and treatment.

Keywords:

meningitis, lumbar puncture (LP), fever, cerebrospinal fluid (CSF), Listeria *meningitis*

Table 26-1. Causes of Meningitis

INFECTIOUS CAUSES	NONINFECTIOUS CAUSES
Bacteria	Neoplastic
Viruses	Collagen vascular
Fungi	Drugs (i.e., antibiotics and antiinflammatory medications)
Parasites	
Tuberculosis	

Table 26-2. Organisms Most Commonly Involved by Patient Group

AGE OR CONDITION	MOST COMMONLY ENCOUNTERED ORGANISMS
Newborns	Group B or D streptococci, non–group B streptococci, *Escherichia coli*
Infants and children	*Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae*
Adults	*S. pneumoniae, H. influenzae, N. meningitides,* staphylococci, streptococci, *Listeria* species
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.

- Kernig 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 the previously mentioned physical findings.)

These findings are often absent in the very young and in older patients.

7. List the presenting signs of meningitis in infants.

- Bulging fontanel (may not be present if patient is dehydrated)
- Paradoxic irritability (quiet when stationary, cries when held)
- High-pitched cry
- Hypotonia
- Skin over the 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 cerebrospinal fluid (CSF) is the only way to diagnose meningitis.

9. What tests should be done before doing an LP?

- Funduscopic examination: Check for papilledema and presence or absence of spontaneous venous pulsations.
- 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
  - Clinical suspicion for recent trauma or subarachnoid bleed
- Coagulation studies and platelet count: Order if there is suspicion for bleeding disorder.

10. What is the most common error in ED management of meningitis?

The most common error is 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 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: Caused by 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 are the contraindications to performing an LP?

- There are no absolute contraindications.
- Use caution in patients with possible increased intracranial pressure, thrombocytopenia or other bleeding diathesis, or suspected spinal epidural abscess.
- Be cautious in patients with severe thrombocytopenia (platelet counts $<50{,}000/mm^3$) or with an elevated international normalized ratio (INR; >1.4). In these situations consider correcting the abnormality before performing the LP.

13. 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.

14. 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.

15. What can cause a falsely elevated intracranial pressure?

Intracranial pressure can be elevated by a tense patient, the head being elevated above the plane of the needle, marked obesity, or muscle contraction.

16. Which laboratory studies should be ordered on the CSF?

Four tubes are usually collected, each containing 1 to 1.5 mL of CSF. 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: Microbiology
- Tube 2: Glucose and protein
- Tube 3: Cell count and differential.

17. What findings on LP are consistent with bacterial meningitis?

See Table 26-3.

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 a traumatic LP has occurred, correct the CSF protein result for the presence of blood by subtracting 1 mg/dL of protein for each 1000 RBCs.
3. A high CSF protein level associated with a benign clinical presentation should suggest fungal disease.

Table 26-3. Findings Consistent with Bacterial Meningitis

PARAMETER	FINDING
Opening pressure	In range of 20-50 cm H_2O
Appearance	Cloudy
White blood cell count	1000-5000 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.

Table 26-4. 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 years	Vancomycin + broad-spectrum cephalosporin
Age >50 years	Ampicillin + broad-spectrum cephalosporin + vancomycin
Impaired cellular immunity	Ampicillin + ceftazidime + vancomycin
Head trauma, neurosurgery, CSF shunt	Vancomycin + ceftazidime
Patients with severe β-lactam allergies	Vancomycin + moxifloxacin + trimethoprim-sulfamethoxazole (if need *Listeria* coverage)

CSF, Cerebrospinal fluid; *IV*, intravenously; *Rx*, recipe or prescription.

18. Which antibiotics should be prescribed when the causative organism is unknown?

See Table 26-4.

19. 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, altered cerebral blood flow, and hearing loss. 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.

20. 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 (for children older than 1 month, 10 mg/kg every 12 hours; younger than one month, 5 mg/kg every 12 hours.). Other accepted prophylaxis regimens for *Neisseria meningitides* include the following: ciprofloxacin 500 mg single dose (not recommended for pregnant or lactating women or patients younger than 18 years of age.); ceftriaxone 250 mg intramuscular (IM) dose for adults or 125 mg IM for children (used in pregnancy); or a single oral dose of azithromycin 500 mg for adults (pediatric dose 10 mg/kg). Note that azithromycin has been used if ciprofloxacin resistance has been detected. 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. Rifampin dose recommended for *H. influenza* prophylaxis is 20 mg/kg (maximum 600 mg) once daily for four days (10 mg/kg for children younger than 1 month). Individuals exposed to someone with other types of meningitis, especially viral, do not need prophylactic antibiotics.

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QUESTIONS

1. Which of the following is a sign of meningitis in infants?
   a. Cries when stationary
   b. Hypertonia
   c. Sunken fontanel
   d. Cries when held
   The correct answer is *d*.
2. Which of the following statements is true about meningitis diagnosis and management?
   a. CT scan of the brain needs to be performed before LP.
   b. Transient leg paresthesias is the most common sequela of LP.
   c. When interpreting LP results subtract one WBC for every 700 RBCs.
   d. Never administer antibiotics before LP, because it will affect the results of the CSF analysis.
   The correct answer is *c*.
3. Which of the following groups need treatment for *Listeria monocytogenes*?
   a. Newborns
   b. Patients older than 50 years
   c. Teenagers
   d. Head trauma patients in their 30s
   The correct answer is *b*.

BREATHING AND VENTILATION

Jeffrey Sankoff, MD, and David B. Richards, MD, FACEP

CHAPTER 27

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 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 pulse.

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 of saturation.

Abstract

This chapter addresses the assessment of breathing and ventilation with particular attention to the application of noninvasive and invasive ventilation use.

Keywords:

breathing, ventilation, oxygenation, pulse oximetry, end-tidal capnography, noninvasive ventilation

5. When might the pulse recorded from the pulse oximeter be different than that shown on the cardiac monitor?

This occurs in situations where electrically conducted beats do not result in a subsequent pulse (i.e., electromechanical dissociation) and can provide valuable clinical information.

6. How can pulse oximetry be useful?

Pulse oximetry is useful when monitoring arterial O_2Hb saturation in cardiopulmonary disorders, monitoring oxygen saturation during conscious procedural sedation (previously referred to as *conscious sedation*) or airway management, or in patients with a decreased level of consciousness, as well as in quantifying the arterial O_2Hb saturation response to therapeutic interventions.

7. In which situations can pulse oximetry yield false readings?

Situations in which the usefulness of pulse oximetry is limited include vasoconstriction, excessive movement, low O_2Hb saturations (<83%), exposure of the measuring sensor to ambient light sources, and in the presence of certain types of nail polish. Oxygen saturation measurements may be falsely elevated in the presence of carboxyhemoglobin and falsely decreased in the presence of methemoglobin or sulfhemoglobin.

8. 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 carbon dioxide (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.

KEY POINTS: PULSE OXIMETRY

1. Pulse oximetry measures oxygenation, not ventilation.
2. Poor peripheral perfusion is a common reason pulse oximeters provide unreliable readings.

9. What is end-tidal CO_2 ($EtCO_2$) monitoring?

An $EtCO_2$ monitor is 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 may be either qualitative or quantitative. Qualitative detectors (generally colorimetric) are used immediately after intubation to detect the presence of CO_2 in exhaled gas, and are used only to determine the proper placement of the endotracheal tube. Quantitative $EtCO_2$ detectors continuously monitor exhaled CO_2, displaying its concentration in both numeric and graphic 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.

10. When is $EtCO_2$ monitoring useful?

$EtCO_2$ is being used in a number of ways:

- During conscious procedural sedation
- In patients with sepsis or shock 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

11. 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 face mask, and face mask 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% over and above the atmospheric concentration (21% at sea level). As a result, a nasal cannula can deliver an FiO_2

between 25% and 45%. A simple face mask relies on an oxygen flow of 5 to 10 L/min, with a resulting FiO_2 ranging from 35% to 50%. A face mask with an oxygen reservoir has a constant flow of oxygen so that higher concentrations of oxygen can be achieved. A properly fitted face mask with an oxygen reservoir with a 15-L/min flow rate can deliver up to 85% FiO_2.

12. What is noninvasive ventilation?

It is a means of delivering positive-pressure ventilation without placement of 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 and in many cases can help avoid the need for intubation in some patients.

13. What forms of noninvasive ventilation are available to emergency physicians?

The two most useful forms of noninvasive ventilation used are mask continuous positive airway pressure (CPAP) ventilation and bilevel positive airway pressure (BiPAP). With each method, a tight-fitting mask is placed over the patient's face and high-flow air with or without supplemental oxygen is delivered through the circuit in order to augment the patient's breathing 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 additional 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, thus easing the work of breathing.

14. In what circumstances would noninvasive ventilation be preferred over standard invasive ventilation?

Noninvasive ventilation has been shown to be most useful in many conditions, including in patients with cardiogenic pulmonary edema, pneumonia, asthma, COPD, and nocturnal hypoventilation. There is some evidence to suggest that it may also confer some benefit in patients with asthma and pneumonia with hypoxia. Noninvasive ventilation is most beneficial in those settings where the anticipated time needed for its use is short. The best examples are in patients with acute exacerbations of CHF or of COPD. In properly selected patients, CPAP is particularly useful in the treatment of pulmonary edema, and BiPAP is useful in a patient with respiratory distress caused by COPD. Patients with CHF exacerbations have been shown to improve more rapidly with noninvasive ventilation than without it and rarely need to have their airways intubated when noninvasive ventilation is employed. Although overall mortality is not improved with the use of noninvasive ventilation in these patients, this is likely the result of the underlying cardiac disease and not the failure of noninvasive treatment. Patients with COPD are notoriously difficult to wean from mechanical ventilators, and noninvasive ventilation can often be used to reverse the situation of patients with COPD in moderate respiratory distress who would otherwise have required standard invasive ventilation. Unlike in patients with CHF exacerbations, the use of noninvasive ventilation has been shown to improve outcomes in patients with COPD exacerbations. Lastly, some patients with advance directives forbidding mechanical ventilation may benefit from the respiratory support provided by noninvasive ventilation.

15. When is noninvasive ventilation contraindicated?

Noninvasive ventilation cannot be used in certain clinical settings. Principally, these are when the patient has an altered mental status or when patients are not breathing spontaneously. Other contraindications include when patients are at risk for, or are, vomiting or cannot tolerate a tightly fitted mask. Other clinical scenarios in which noninvasive ventilation would not be appropriate include anticipated prolonged need for ventilation and disorders that are not responsive to oxygen or enhanced ventilation (e.g., hemoglobinopathies, neuromuscular weakness).

16. How do I determine the initial ventilator settings in someone who has just had his or her airway intubated?

Ventilator settings should reflect the reason for which the airway was initially intubated and ventilation started, and afterward 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). Respiratory rate may also have an impact on oxygenation, but to a lesser degree. Initially, patients with intubation should be given

100% oxygen or an FiO_2 of 1.00. Subsequently, if arterial blood gas analysis reveals that the partial pressure of oxygen (PaO_2) is high, the FiO_2 and PEEP should be lowered incrementally to maintain an adequate oxygen saturation. It is desirable to reduce FiO_2 to less than 0.6 as soon as possible, because sustained levels higher than this may lead to alveolar tissue damage via free radical formation.

The main factors determining a patient's ventilatory status are tidal volume and respiratory rate. Changes in each are reflected by the partial pressure of carbon dioxide ($PaCO_2$) from arterial blood gas analysis. High respiratory rates and large tidal volumes decrease the CO_2 level, whereas the converse elevates the CO_2 level. Initially, the tidal volume can be estimated to be 6 to 8 mL/kg; for a 70-kg patient, that is 600 to 800 mL. The initial respiratory rate varies depending on the clinical situation. On average, it should be set between 10 and 16 breaths per minute.

17. Are ventilator settings always the same?

No, when you intubate a patient's airway, 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 the patient's respiratory center has millions of years of evolution backing it up, compared with your relatively few years of experience. Imagining how the patient's respiratory center would respond to the clinical situation and the etiology of the failure will help you to determine the best ventilator settings for the 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. Monitoring $EtCO_2$ and pulse oximetry can provide real-time feedback of the adequacy of the chosen settings. Other common ventilator settings for patients with closed head injury, CHF, metabolic acidosis, and sepsis are shown in Table 27-1. The most important thing to remember about ventilator settings is that they should not be static. As the patient's status changes over time, so too should the ventilator settings, reflecting the patient's need for more or hopefully less support as time goes on.

18. 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 IMV (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 minimum 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 occur in the IMV mode.

19. Are there different methods of delivering ventilation?

Ventilation can be provided in two ways: (1) by providing a set tidal volume (volume control), or (2) by providing a set inspiratory pressure (pressure control). With volume control modes, the provider sets a tidal volume, and the pressures that arise within the patient are a result of the interaction between the volume and the patient's characteristics (e.g., a large tidal volume in a small patient will generate high pressures). With pressure control modes, the provider sets an inspiratory pressure, and the resulting tidal volume will be determined by the interaction between the pressure and the patient (e.g., high pressure and small patient results in a high tidal volume).

20. What are the most commonly used methods?

The most commonly used volume control modes are AC and SIMV. With AC, all breaths administered by the ventilator are of the same tidal volume. The machine will give a minimum number of breaths per minute. The patient may initiate additional breaths on top of that minimum set rate, although all additional breaths will be of the same tidal volume. With SIMV, the ventilator administers a set minimum number of breaths of a preset tidal volume, and additional breaths initiated by the patient have varying tidal volumes dependent on patient effort.

The most commonly used pressure control mode is pressure support ventilation (PSV). With PSV the patient must initiate all breaths because there is no minimum rate administered by the machine,

Table 27-1. Initial Ventilator Settings According to Condition

CONDITION	TIDAL VOLUME (mL/kg)	RESPIRATORY RATE (BREATHS/MIN)	FiO_2	PEEP (cm H_2O)	COMMENTS
Asthma	5-8	6-10	100	5-10	Low RR will allow for lung deflation. Paralysis will be needed and hypercapnea WILL result. This may be tolerated until goals of treatment reached.
COPD	6-10	10-14	≤40	5	Lowest FiO_2 necessary to maintain SaO_2 of 90% or greater. Titrate down RR as soon as possible to allow patient to assume work of breathing.
Head injury	8	14	40	0	Normocapnea is goal. Low PEEP improves venous drainage from head and may improve cerebral perfusion pressure. Avoid hyperoxia if possible.
CHF	8	14	100	5	
Metabolic acidosis	8-10	18-22	50	5	If lungs are healthy, lower FiO_2 is all that is needed. If not, use higher concentration. High minute ventilation will offset metabolic acidosis.
Sepsis	6	12-16	100	5	Use higher PEEP to improve oxygenation. Avoid higher lung volumes.

CHF, Congestive heart failure; *COPD,* chronic obstructive pulmonary disease; *FiO_2,* fraction of inspired oxygen; *PEEP,* positive end-expiratory pressure.

and so spontaneous ventilation is a requirement for this mode. With each breath, the machine augments pressure to the predetermined rate, and tidal volume is determined by patient effort.

21. What is PEEP?

PEEP is positive end-expiratory pressure, or pressure that is applied during expiration. PEEP prevents collapse of alveoli at the end of expiration, leading to an increase in functional residual capacity and a recruitment of alveoli to participate in gas exchange. The end result is improved V/Q matching in the pulmonary circulation, thus improving oxygenation. On the flip side, higher PEEP can induce barotrauma, diminish venous return to the heart, and elevate intracranial pressure. PEEP is usually set at 2.5 or 5 cm H_2O. However, in certain clinical settings, especially those in which there is increased stiffness of the alveolar walls such as in acute respiratory distress syndrome (ARDS), much higher levels may be appropriate.

22. What is auto-PEEP?

Auto-PEEP occurs 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 side of the heart, and, consequently, hypotension. The increased airway pressures can result in barotrauma, pneumothorax, and inaccurate pulmonary artery catheter measurements. Auto-PEEP can be a particular problem in

the mechanical ventilation of patients with COPD or asthma. The immediate solution is to disconnect the ventilator circuit and allow full exhalation followed by appropriate changes to the ventilator settings.

23. What are the most common complications of mechanical ventilation?

The most common direct complication seen in the ED is barotrauma. High pressure can cause rupture of the alveolar wall, which in turn can lead to pneumomediastinum, pneumothorax, tension pneumothorax, pneumoperitoneum, and subcutaneous emphysema. Pneumonia tops 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.

24. How do I approach a patient on a ventilator with acutely worsening oxygenation or ventilation?

A systematic approach to this situation is necessary. First, remove the patient from the ventilator and provide manual ventilation to the patient by using a bag ventilatory device. Many problems involving a $30,000 ventilator can be solved with a $15 resuscitation bag. The DOPE mnemonic taught in pediatric life support can be helpful in remembering the remainder of the approach.

Displacement: Confirm that the endotracheal tube is in the proper place by using some combination of auscultation, measurement of CO_2 exchange, radiography, and direct visualization.

Obstruction: Confirm that the endotracheal tube is patent by passing a suction catheter down the lumen. Sometimes an endotracheal tube can become kinked simply as a result of patient positioning.

Patient: Consider various causes within the patient. First and foremost is the possibility of secretions obstructing large bronchi. Vigorous suctioning may remedy this situation. Next, consider pneumothorax. Confirm that there is no evidence of barotrauma, usually by a combination of physical examination and a chest radiograph.

Equipment: Confirm that the ventilator circuit and ventilator itself are functioning properly.

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 $PaCO_2$.
3. FiO_2 and PEEP affect the patient's oxygenation and pO_2.
4. Oxygenation and ventilation problems in patients using mechanical ventilators can be managed by removing them from the ventilator and following the DOPE mnemonic.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. John L. Kendall and Ryan D. Patterson for their previous contributions to this chapter.

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QUESTIONS

1. Pulse oximetry measures
   a. The effectiveness of a patient's ventilation
   b. A patient's heart rate
   c. The effectiveness of your patient's oxygenation
   d. Oxygenation accurately regardless of the patient's perfusion
   The correct answer is *c.*
2. Noninvasive ventilation is contraindicated for patients with
   a. Pneumonia
   b. Altered mental status
   c. CHF
   d. COPD
   The correct answer is *b.*
3. Ten minutes after you have intubated a patient's airway, an arterial blood gas evaluation shows that they have a pH of 7.15, a pCO_2 of 52, a pO_2 of 180, a HCO_3^- of 20, and an oxygen saturation of 97%. The best adjustment to make to their ventilator settings at this point would be to
   a. Increase the FiO_2
   b. Decrease the PEEP
   c. Increase the rate
   d. Decrease the tidal volume
   The correct answer is *c.*

ASTHMA, CHRONIC OBSTRUCTIVE PULMONARY DISEASE, AND PNEUMONIA

CHAPTER 28

Scott Felten, MD, FACEP, and Rita K. Cydulka, MD, MS

ASTHMA

1. What is asthma, and what are the presenting symptoms of asthma exacerbation?

Asthma is a heterogenous 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. This creates variable airflow limitation.

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
- Cystic fibrosis
- Bronchiectasis
- Parenchymal lung disease
- Vocal cord dysfunction

3. Which aspects of the asthmatic patient's history are important to the current exacerbation?

Primary considerations are exposure include common precipitants, such as the following:

- Viral upper respiratory tract infections
- Allergens
- Cold
- Exercise
- Obesity
- Occupational exposure
- Possible aspirin or nonsteroidal antiinflammatory 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 taking corticosteroids
- Possibility of weaning off corticosteroids
- Other comorbidities

Non-Caucasian race and lower socioeconomic standing are also risk factors for severity requiring hospitalization.

Abstract

Asthma, chronic obstructive pulmonary disease (COPD), and pneumonia are diseases commonly encountered in the ED. The diagnosis, treatment, and disposition of these conditions is discussed in some detail.

Keywords:

asthma, chronic obstructive pulmonary disease (COPD), pneumonia, Patient Outcome Research Team pneumonia-specific severity of illness (PORT PSI) scoring, atypical pneumonia, community-acquired pneumonia

4. Are there any helpful ancillary diagnostic tests?

Bedside spirometry, which is dependent on the cooperation and effort of the patient, 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 70% or greater of predicted (or personal best) indicates mild obstruction
- FEV_1 or PEFR 40% to 69% of predicted (or personal best) indicates moderate obstruction
- FEV_1 or PEFR less than 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 radiography 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 objective is to minimize morbidity from acute episodes, which includes 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 (Table 28-1). 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. Evidence does not suggest an improved benefit from intravenous (IV) β_2-agonists compared with aerosol. Early administration of systemic corticosteroids addresses the inflammatory component of acute asthma and has been demonstrated to prevent some hospitalizations, 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 using inhaled steroids. There is no added

Table 28-1. Medications Used to Treat Asthma and Chronic Obstructive Pulmonary Disease 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 IM
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	40-60 mg by mouth
Methylprednisolone	125 mg intravenously

COPD, Chronic obstructive pulmonary disease; *MDI*, metered dose inhaler.
*Exercise extreme caution in patients with known coronary artery disease.

benefit to IV corticosteroids compared with oral doses. Ipratropium should be added when treating severe exacerbations and is most effective in children and smokers. Aerosolized ipratropium should be added if FEV_1 or PEFR is less than 40% of the predicted value, because studies reveal that they increase pulmonary function modestly and decrease need for hospitalization. Hypoxemia is usually corrected by administration of supplemental oxygen with a goal of oxygen saturation of 90% to 95%. Epinephrine or terbutaline may be administered subcutaneously to patients unable to manage aerosolized treatments in severe exacerbations only. Theophylline is not recommended in the acute setting.

6. How can I determine whether my patients are improving?

Ask them how they feel, reexamine 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 each 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 is not 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 often difficult in patients who have asthma. Mechanical ventilation of patients with acute asthma presents special challenges, such as auto-PEEP and barotraumas (see Chapter 27).

8. How should I decide whether a patient can be discharged or requires hospitalization?

Disposition of patients is usually determined by their 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 that is 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 less than 50% of predicted and who continue to have moderate to severe symptoms after treatment, require hospitalization. If an ED observation capability exists, observation for 4 to 6 hours after steroid administration will decrease the number of inpatient admissions.

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 to 50 mg prednisone per day). Patients not already taking controller medications who have mild persistent asthma should start 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 whose symptoms are inadequately controlled by 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 should instructions to make 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 women with asthma aggressively to prevent maternal hypoxia and subsequent fetal morbidity and mortality. Patients should not be undertreated because of fear of teratogenicity, because 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 and ipratropium.
3. Reduce the likelihood of recurrence: Corticosteroids.
4. Provide objective measure of improvement: PEFR or FEV_1.
5. Provide adequate discharge planning: Education, medications, and follow-up care.

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, genetic predisposition, 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 with wheezing
  - Asthma
  - CHF
  - Pneumonia
  - Cardiogenic pulmonary edema
  - Foreign body aspiration
  - Anaphylaxis
  - Epiglottitis
  - Bronchitis
- In those who have dyspnea
  - Myocardial ischemia
  - Pericardial effusion
  - Cardiac tamponade
  - Pneumothorax
  - Pulmonary embolism
  - Pneumonia
  - Asthma
  - Acute respiratory distress syndrome (ARDS)
  - Bronchiectasis
  - Pulmonary fibrosis
  - Pleural effusion
  - Tuberculosis (TB)
  - Metabolic disturbances
  - Acidosis
  - 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. 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 dysrhythmias. The use of B-type natriuretic peptide (BNP) does not substitute for clinical judgment when trying to differentiate COPD from CHF, because a numeric cut-off value that differentiates between the two diseases remains elusive.

14. What is the role of pulmonary function tests (PFTs) for COPD?

In contrast to asthma, acute PFTs are less helpful in the emergency setting because of the difficulty that sick patients with COPD have in performing these tests properly. The formal diagnosis of COPD is made with spirometry and is positive when the ratio of FEV_1 to forced vital capacity (FVC) is less than 80% of that predicted for a matched control.

Differentiation of mild, moderate, and severe COPD relies on the FEV_1.

- Stage I (mild): FEV_1 80% or greater of predicted
- Stage II (moderate): FEV_1 50% to 79% of predicted
- Stage III (severe): FEV_1 30% to 49% of predicted
- Stage IV (very severe): FEV_1 less than 30% of predicted or FEV_1 less than 50% and chronic respiratory failure

Making this calculation without formal PFTs is generally not possible in the ED.

15. 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, carbon dioxide (CO_2) retention owing 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 gas measurements, when needed. Excessive supplemental oxygen in this small subset of patients can cause respiratory arrest secondary to loss of the 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. Studies have shown that combination therapy results in greater bronchodilator response and provides greater relief. Systemic corticosteroids are indicated in severe exacerbations of COPD. The use of methylxanthines (theophylline or aminophylline) remains controversial; they should be used only when there is inadequate response to short-acting bronchodilators. The use of newly prescribed inhaled long-acting β-agonists and long-acting anticholinergic drugs for COPD was associated with an increased risk of experiencing a cardiovascular event.

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 for patients who 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 whether my patient is improving?

Ask the patient how he or she feels, reexamine the patient, and monitor his or her 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. Measurement of partial pressure of CO_2 ($PaCO_2$) levels on a blood gas test for those patients in significant retention may also be helpful.

17. When should the airway of a patient with COPD be intubated?

Noninvasive modalities such as continuous positive airway pressure (CPAP) and bilevel 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%. Indications for intubation and mechanical ventilation include inability to tolerate NIV or not responding to NIV, acute deterioration exhibited by mental status change, increased respiratory distress with cyanosis, respiratory arrest, shock, severe acidosis (pH of 7.25), and hypercapnia ($PaCO_2$ >60 mm Hg).

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. These patients often take longer than an ED visit to recover and require hospitalization. Failure of a patient's

symptoms to improve while he or she is 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 instructions for close follow-up monitoring.

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 to 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 chronic use of 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 should instructions to make an appointment for a follow-up visit within several days.

20. When is ipratropium 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 and ipratropium.
3. Consider antibiotics if there are changes in sputum production.
4. Patients with COPD have less respiratory reserve and require admission more often 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 observation.

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. The ED serves as the point of entry for the majority of these admissions. When patients are properly identified and treated as outpatients, the mortality of community-acquired pneumonia (CAP) decreases significantly. 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 28-2 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 have fever associated with irritability, tachypnea, intercostal retractions, nasal flaring, and grunting, but notably, cough may be absent. Elderly or debilitated patients may have nonspecific complaints and findings, such as confusion or deterioration of baseline function, rather than classic symptoms. The most common organism is *S. pneumoniae*.

Table 28-2. 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

Modified from www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/infectious-disease/community-acquired-pneumonia/; accessed 9-15-15.

COPD, Chronic obstructive pulmonary disease; *CVA*, cerebrovascular accident; *ET*, endotracheal; *NT*, nasotracheal.

- 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 (Table 28-3). 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 caused by anaerobic oral flora, *S. pneumoniae*, *Legionella*, and *M. 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?

In addition to the symptoms mentioned in Question 23, findings consistent with pneumonia include the following:

- Fever
- Tachypnea
- Tachycardia
- Decreased oxygen saturation
- Altered mental status associated with severe illness

The physical examination may show evidence of alveolar fluid (inspiratory rales), consolidation (bronchial breath sounds), pleural effusion (dullness and decreased breath sounds), or bronchial congestion (rhonchi and wheezing).

Table 28-3. 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, including influenza)*	2-16	Atypical
Legionella species	2-8	Typical
Chlamydia pneumoniae	4-6	Atypical
Aspiration	6-10	Variable
Gram-negative bacilli *(Klebsiella, Pseudomonas)*	3-10	Typical
Others	3-5	Variable

Modified from www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/infectious-disease/community-acquired-pneumonia/; accessed 9-15-15.

*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.

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 radiograph 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 radiograph, but all patients who have a cough do not need chest radiography. Clinical judgment must be used along with the presence of clinical indicators. The American Thoracic Society (ATS) and the Infectious Diseases Society of America (IDSA) include radiographic findings as part of their definitions of pneumonia. IDSA 2007 pneumonia guidelines state, "In addition to a constellation of suggestive clinical features, a demonstrable infiltrate by chest radiograph or other imaging technique, with or without supporting microbiologica data, is required for the diagnosis of pneumonia."

An arterial blood gas test 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: white blood cell count and measurement of serum electrolyte levels. The use of sputum Gram stain and blood cultures is controversial.

27. What radiographic findings are helpful in making a microbiologic differential diagnosis?

Radiographic findings may suggest the underlying microbial source, but the overlapping variations in radiographic signs between different organisms may lead to misclassification. A radiograph is not diagnostic of a specific pathogen. An image of the chest 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 (Table 28-4).

28. How do I determine the disposition of a patient with pneumonia?

Once a diagnosis of pneumonia is strongly suspected by history, physical, and radiographic 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 older), 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 (Tables 28-5 to 28-7). 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 admission to an intensive care unit (ICU), several rules have been published. Patients requiring

Table 28-4. Radiographic Appearances of Community-Acquired Pneumonia

RADIOGRAPHIC PATTERN	SUGGESTED ORGANISMS
Lobar	*Streptococcus pneumoniae, Klebsiella* species, pneumonia caused by 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*†

Modified from www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/infectious-disease/community-acquired-pneumonia/; accessed 9-15-15.
*The development and resolution of radiographic findings may lag clinical findings by hours to days.
†Tuberculosis may take on almost any radiographic appearance, with some predilection for the upper lobes.

Table 28-5. Factors in Pneumonia Disposition Decision

PORT CHARACTERISTICS	POINTS GIVEN FOR PRESENCE OF CHARACTERISTIC
PORT PSI Scoring	
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 Radiographic 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 <60 mm Hg or oxygen saturation <90%	+10
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 28-6. PORT PSI Class Based on Point Totals and Mortality

POINTS CALCULATED FROM PSI	CLASS	MORTALITY(%)
<51	I	0.1
51-70	II	0.6
71-90	III	0.9
91-130	IV	9.5
>130	V	26.7

PORT, Patient Outcome Research Team; *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 admission to an intensive care unit.

Table 28-7. Other Factors (Not Part of PORT PSI) That Impact Disposition Decision

- Patient's clinical appearance
- Patient's ability to tolerate oral intake
- Patient's reliability
- Social factors, such as home support
- Clinical judgment of the physician (most important)

PORT, Patient Outcome Research Team; *PSI*, pneumonia-specific severity of illness.

ventilatory assistance or vasopressors and those who have altered mental status, multilobar or bilateral infiltrates, pleural effusion, age older than 65 years, comorbid conditions, respiratory rate greater than 30 breathes per minute, heart rate greater than 125 beats per minute, oxygen saturation level less than 90%, white blood cell count less than 3 or higher than 20, blood urea nitrogen (BUN) level greater than 11 mg/dL, pH less than 7.35, and sodium less than 130 mEq/L are among the patients who should be considered for an ICU admission. The patient's condition meeting criteria for concurrent systemic inflammatory response syndrome (SIRS) may influence the disposition as well.

29. What treatment should be started in the ED?

Supportive care, including oxygen and ventilatory support, should be provided 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 before transfer to a hospital floor or ICU.

30. Which antibiotic should I use?

The choice of antibiotic is based on the site of treatment and suspected pathogens. The suggestions in Table 28-8 should be used in consideration with the clinical picture, recent literature, local preference, and resistance patterns. Increasing evidence has strengthened the recommendation for combination empiric-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.

31. Has the epidemiology of pneumonia changed in recent years?

The epidemiology of CAP continues to change because of 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. *S. pneumoniae* continues to be the most common single agent and

Table 28-8. Empiric 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) *M. pneumoniae* *C. pneumoniae* *H. influenzae* Viruses Gram-negative bacilli*†	Fluoroquinolone alone* or a macrolide plus a β-lactam
Inpatient Not severely Ill	*Staphylococcus aureus**† *S. pneumoniae* *H. influenzae* Polymicrobial Anaerobes *S. aureus* *C. pneumoniae* Viruses	A macrolide and β-lactam or a fluoroquinolone‡ alone
Inpatient Severely ill	*S. pneumoniae* *Legionella* *Gram-negative bacilli* *M. pneumoniae* Viruses *S. aureus*	β-Lactam/β-lactamase inhibitor and azithromycin, or a fluoroquinolone‡; *Pseudomonas aeruginosa* possible: IV macrolide or IV fluoroquinolone and aminoglycoside, OR antipseudomonal quinolone and antipseudomonal β-lactam For methicillin-resistant *S. aureus*: Add vancomycin or linezolid

ATS, American Thoracic Society; *IDSA*, Infectious Diseases Society of America; *IV*, intravenous.
*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 caused by these organisms should be hospitalized.
‡Levofloxacin, gatifloxacin, moxifloxacin, or gemifloxacin.

is continually evolving resistance to a wider array of antibiotics. Viral and atypical agents are the most rapidly growing causes. *Pneumocystis carinii* pneumonia and TB are significant pathogens, particularly in the developing world. 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 (accessed January 23, 2015).

32. What is the role of the 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 commonly negative for specific organisms, and the results rarely change therapy. 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 coming 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. Guidelines suggest that blood cultures be obtained in the ED before initiating antibiotics in hospitalized patients who require ICU admission and in 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 patients and residents of nursing homes will be infected with different organisms and require additional antibiotic coverage.
5. Consider whether the presentation is typical or atypical when making therapy decisions.

WEBSITES

Cleveland Clinic Continuing Education program Disease Management: www.clevelandclinicmeded.com\diseasemanagement\pulmonary. Accessed January 23, 2015.

The Global Initiative for Chronic Obstructive Lung Disease: www.goldcopd.org. Accessed January 23, 2015.

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QUESTIONS

1. Which of the following is not a first-line therapy for moderate asthma exacerbations?
   a. β_2-Agonists
   b. Oxygen
   c. Ipratropium
   d. Corticosteroids
   The correct answer is *c.* Ipratropium is reserved for severe exacerbation.
2. Antibiotics should be considered in acute COPD exacerbations for patients who have all of the following except:
   a. Increased dyspnea
   b. FEV_1 <70%
   c. Increased sputum volume
   d. Need for mechanical ventilation
   The correct answer is *b.* Decreased FEV_1 helps to differentiate severity of COPD, not infectious cause.
3. What is the most common cause of community-acquired pneumonia?
   a. *S. pneumoniae*
   b. *S. aureus*
   c. *Klebsiella*
   d. *Pseudomonas*
   The correct answer is *a.*

CHAPTER 29

VENOUS THROMBOEMBOLISM

Stephen J. Wolf, MD

1. **What is the 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 DVT have concomitant PE when studied?**
   Fifty percent. Additionally, a similar percentage of patients with a diagnosed PE will have a concomitant DVT when studied.

4. **What are major risk factors for VTE?**
   - History of VTE
   - Immobilization (equivalent to bed rest ≥3 days)
   - Malignancy (treatment active, within 6 months, or palliative)
   - Postpartum (for up to 42 days)
   - Pregnancy (third > second > first trimester)
   - Recent surgery (≤4 weeks)

5. **List other minor risk factors for VTE.**
   - Advanced age
   - Cardiovascular disease (i.e., heart failure or congenital heart disease)
   - Circulating antiphospholipid antibodies (associated with systemic lupus erythematous)
   - Estrogen use (i.e., hormone replacement or oral contraceptives)
   - Indwelling vascular access
   - Inflammatory bowel disease (i.e., Crohn disease, ulcerative colitis)
   - Inherited thrombophilia (i.e., antithrombin III deficiency, factor V Leyden thrombophilia, protein C or S deficiency, and prothrombin gene mutation)
   - Obesity
   - Neurologic disease (i.e., cerebrovascular accident [CVA], paresis)
   - Renal disease (i.e., chronic kidney disease, end-stage renal disease, dialysis, nephrotic syndrome, or renal transplant)

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 symptoms 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 clinician's pretest probability to yield a posttest likelihood of disease. Thus the pretest probability should be used to determine when to initiate a patient workup and how to interpret the results of any test. See Figure 29-1 for a sample algorithm.

8. **When determining pretest probability for DVT, what are the Wells criteria?**
   - Malignancy (+1 point)
   - Paralysis/paresis/casted lower extremity (+1 point)

Abstract

Venous thromboembolism (VTE) and pulmonary embolism (PE) are common disorders that must de considered in patients coming to the ED with myriad symptoms. They often are true emergent conditions that may have serious morbidity if missed. This chapter describes an evidence-based approach to making this sometimes difficult diagnosis.

Keywords:

venous thromboembolism (VTE), deep venous thrombosis (DVT), pulmonary embolus (PE), Wells criteria, pulmonary embolism rule-out criteria (PERC) rule

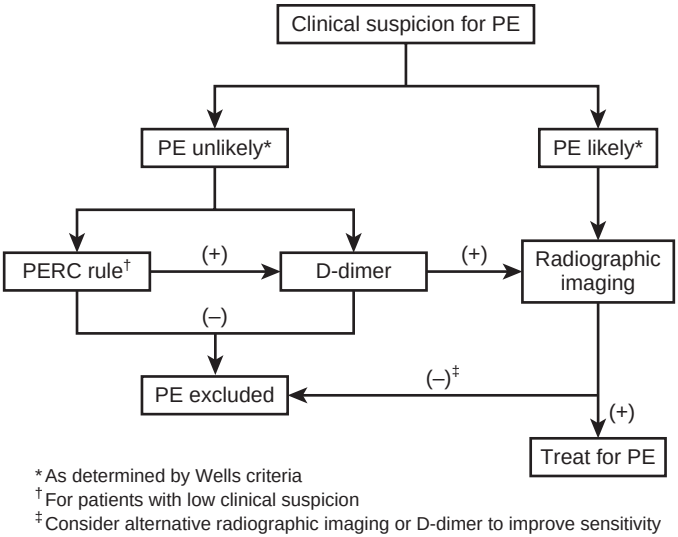

Figure 29-1. Sample algorithm for the clinical suspicion for PE. *PE,* Pulmonary embolism; *PERC,* pulmonary embolism rule-out criteria.

- Recent immobilization or surgery (+1 point)
- Tenderness along deep veins (+1 point)
- Swelling of entire leg (+1 point)
- 3-cm difference in calf circumference (+1 point)
- Pitting edema (+1 point)
- Collateral superficial veins (+1 point)
- Alternative diagnosis more likely than DVT (–2 points)

9. Once I have calculated a patient's Wells score for DVT, how do I interpret it?

Using the Wells criteria for suspected DVT, patients are considered to have low (<2 points), moderate (2 to 6 points), or high (>6 points) pretest probability for a DVT. This correlates to an incidence of DVT of 3%, 17%, and 75%, respectively. Patients with a Wells score for DVT of less than 2 points are considered to be *DVT unlikely* and good candidates for a screening D-dimer test.

10. What is the pulmonary embolism rule-out criteria (PERC) rule for PE?

PERC is a clinical decision rule that can be used to identify patients who do not require a laboratory or radiograph workup to exclude the diagnosis of PE. The criteria include:

- Age younger than 50 years
- Heart rate less than 100 beats per minute
- Initial room air oxygen saturation level (SaO_2) greater than 94% at sea level
- No unilateral leg swelling
- 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 workup 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 greater than 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?

Using the Wells criteria for suspected PE, patients can be considered to have low (<2 points), moderate (2 to 6 points), or high (>6 points) pretest probability for a PE. This correlates to an incidence of PE of 4%, 21%, and 67%, respectively. Patients with a Wells score for PE of 4 points or less are considered to be *PE unlikely* and good candidates for a screening D-dimer test.

14. What other clinical decision rules have been validated to stratify patients with suspected VTE for risk?

The Geneva score, Revised Geneva score, Charlotte criteria, and Pisa model are other validated risk-stratification scoring systems that can be used to determine pretest probability in patients with suspected PE.

15. What is a D-dimer test? How is it used?

D-dimer, a degradation product of cross-linked fibrin, is found in increased levels of the circulation of patients with acute VTE. The enzyme-linked immunosorbent assay (ELISA), rapid ELISA, turbidimetric assay, 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 proven useful at ruling in the diagnosis.

16. Which patients can have VTE excluded, based on a negative D-dimer?

Only patients considered to be DVT or PE unlikely (i.e., those with low to low-moderate pretest probability for disease) can have VTE excluded based on a D-dimer result. 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 higher pretest probability.

17. What are some clinical situations that cause a false-positive D-dimer test, lending to a decreased specificity?

Sepsis, disseminated intravascular coagulation (DIC), aortic dissection, pregnancy, recent surgery, and severe trauma

18. What are two clinical situations that might cause a false-negative D-dimer result?

Subacute thrombosis (>7 days) and recent anticoagulation

19. What noninvasive imaging methods are available for the diagnosis of DVT?

- Duplex ultrasound: Although often the test of choice, the sensitivity and specificity are operator dependent and related to patient symptomatology. Ultrasound can detect more than 95% of acute symptomatic proximal DVTs. However, its specificity for acute thrombosis decreases in the settings of chronic or recurrent VTE.
- Spiral multirow detector computed tomography venography (CTV): Although not often used in isolation, this modality has a sensitivity and specificity comparable to ultrasound. Venography is most often used in combination with a CT angiogram of the chest to increase the sensitivity of an evaluation for PE.
- Magnetic resonance imaging (MRI) venography: This modality can be useful, particularly for patients with inconclusive ultrasound studies or a contraindication to radiation or contrast dye (i.e., pregnant patients). It has proven accurate for both lower extremity and pelvic DVT.
- Radio-fibrinogen leg scanning: This modality is 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 of this test 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.

20. 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 test or a repeat duplex ultrasound in 5 to 7 days is indicated to definitively exclude the diagnosis.

21. Are there classic chest radiography findings in patients with PE?

No, the chest radiograph may be normal in up to 30% of patients. 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 sign) or a pleural-based wedge-shaped infiltrate suggestive of pulmonary infarct (Hampton hump) are relatively uncommon.

22. 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% of patients. 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 incidence in patient workup whether or not the patients are diagnosed with PE. Of note, precordial T-wave inversions should raise significant concern for right heart strain, and in the setting of PE, indicate worse prognosis.

23. What imaging studies can be used to evaluate PE?

- Spiral multirow detector CT angiography (CTA) scan. CTA is rapid and generally the test of choice. It is highly sensitive and specific in diagnosing central or segmental emboli and other intrathoracic pathology. However, it is not as sensitive in ruling out subsegmental clots. Outcomes data using newer-generation multirow detector CTAs show higher sensitivities. In patients likely to have PE (i.e., those with moderate to high pretest probability), the sensitivity of a negative CTA can be improved with the finding of a negative CT venogram, lower extremity duplex ultrasound, or a D-dimer assay.
- 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 less than 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 if the patient has underlying cardiopulmonary disease. A nondiagnostic scan should be followed up with further diagnostic workup. Limitations of the V/Q scan include technical support, availability, and interpretation variability.
- Pulmonary angiogram. This test has been the traditional gold standard for the diagnosis, even though its interrater 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). Studies have shown that 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.

24. 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

25. 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 patients with low pretest probability. 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. CTA (without concomitant CTV) has been shown to confer lower fetal

radiation doses than the V/Q scan. CTV should be avoided because of the high pelvic radiation doses. When available, MRA 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 obviate the need for thoracic testing.

26. 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%.

27. What is a massive PE?

A massive PE can be either anatomically described as the occlusion of greater than 50% of the pulmonary vasculature, or physiologically described 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.

29. What is a submassive PE?

A submassive PE is generally felt to be one that is significant enough to cause right heart strain or impaired right heart function without systemic hypotension. Controversy exists on how to optimally treat patients with a submassive PE.

29. What is the treatment for DVT?

Anticoagulation should be started in the ED. Studies suggest that patients with proximal DVTs and temporary risk factors can receive heparin as an anticoagulation agent (80 mg/kg loading dose followed by 18 mg/kg/h infusion), followed by warfarin for 3 months. Patients with calf DVTs need to be treated for only 6 weeks. Patients with permanent risk factors potentially need lifelong treatment but should take anticoagulant medication for at least 3 months.

30. What is the treatment for PE?

Initial treatment of acute PE is dependent on the patient's hemodynamic stability.

- Persistently unstable patients (e.g., massive PE) should be considered for systemic thrombolytic therapy with or without embolectomy.
- The treatment of hemodynamically stable patients who have evidence of significant right heart strain (i.e., submassive PE) with systemic or catheter-directed thrombolytic therapy is more controversial and should be considered on a case-by-case basis, weighing the risks and benefits to the patient.
- In hemodynamically stable patients with acute PE who are not receiving thrombolytics, early anticoagulant therapy is the primary treatment for PE and should be started in the ED. Unfractionated heparin can be initiated at 80 mg/kg loading dose, followed by 18 mg/kg/hr infusion, followed by warfarin for at least 3 months.

31. What is the role of low–molecular-weight heparin (LMWH) in the treatment of VTE?

- LMWH is 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.
- Recent studies indicate that LMWH may be acceptable for the initial treatment of PE in select patient populations. When considering outpatient therapy or early discharge from the hospital, the Pulmonary Embolism Severity Score (PESI) can be used to stratify patients at low risk for mortality. Patients should be considered for early home therapy on a case-by-case basis, weighing the risks and benefits to the patient.

32. Under what conditions can an inferior vena caval filter be considered in the treatment of VTE?

- Contraindication to anticoagulation
- Recurrent VTE despite adequate anticoagulation

KEY POINTS

1. Use a clinical decision rule (e.g., Wells criteria) to help stratify a patient's pretest probability of disease when interpreting diagnostic test results as evaluation for VTE in the ED.
2. Use the D-dimer assay only in patients who are classified as PE unlikely, as determined by Wells criteria, to rule out PE.
3. Use the PERC rule to determine which low-risk patients require no radiographic or laboratory evaluation in order to exclude PE.
4. Strongly consider additional diagnostic testing (e.g., CTV, lower extremity duplex ultrasound, or a D-dimer) to exclude PE in high-probability patients with a negative CT angiogram of the chest for PE.
5. Consider appropriate outpatient therapy for VTE when supported by the patient's clinical condition and careful consideration of the risk-to-benefit ratio.

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QUESTIONS

1. In a patient with a low gestalt suspicion for PE, which historical factor should prompt further workup for the disease based on the PERC rule?
   a. Pleuritic chest pain
   b. Obesity
   c. Tobacco use
   d. Oral hormone use
   The correct answer is *d.*
2. What would be the most appropriate next diagnostic step for evaluation of PE in a patient with a Wells score of 4 (i.e., a moderate pretest probability but in the *PE unlikely* category)?
   a. No further workup needed
   b. D-dimer assay
   c. Spiral multirow detector CTA scan
   d. Lower extremity ultrasound
   The correct answer is *b.*
3. The most appropriate therapy for a young, hemodynamically stable patient diagnosed with an acute PE but found to have isolated T-wave inversions in precordial leads on ECG would be?
   a. Embolectomy
   b. Thrombolytics
   c. Unfractionated heparin
   d. LMWH
   The correct answer is *c.*

CHAPTER 30

CONGESTIVE HEART FAILURE AND ACUTE PULMONARY EDEMA

Jeffrey Sankoff, MD

1. What is congestive heart failure (CHF)?

CHF is 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 ensues.

2. What causes CHF?

CHF results from any of the following four types of processes:

1. Restrictive (hemochromatosis, pericardial disease)
2. Ischemic (myocardial infarction)
3. Congestive (volume overload of the ventricle from valvular insufficiencies)
4. Hypertrophic (long-standing hypertension or valvular stenoses)

3. Describe the symptoms of CHF.

Common symptoms are dyspnea (the subjective feeling of difficulty breathing) and fatigue. Early in the course of CHF, the patient reports exertional dyspnea; the heart is able to supply enough cardiac output (CO) for sedentary activities but does not have the reserve to increase CO 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 a supine posture, venous return from the abdomen and lower extremities is improved, increasing right ventricular CO 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 main determinants of cardiac function in CHF.

CO = Stroke volume (SV) × Heart rate (HR)

SV is determined by:

- Preload
- Afterload
- Myocardial contractility

Abstract

This chapter summarizes the pathophysiology of congestive heart failure (CHF). The commonly presenting situations of subacute and acute failure are described, and the diagnosis and management of both conditions is covered. Finally, brief consideration is given to outpatient management of patients with stable CHF.

Keywords:

cardiac failure, pulmonary edema, noninvasive mask ventilation, congestive heart failure (CHF), preload, afterload, B-type natriuretic peptide (BNP)

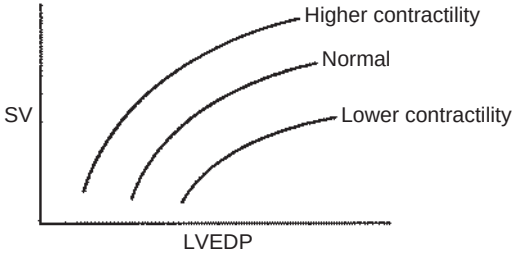

Figure 30-1. Frank-Starling curves. *LVEDP,* Left ventricular end-diastolic pressure; *SV,* stroke volume.

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 SV represents cardiac work (Fig. 30-1). (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 VEDPs, the increase in SV is less for a given increase in VEDP.

7. What are the effects of decreased contractility?

From Figure 30-1, 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. The difference in volume pumped 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 an adequate CO.

9. What about HR?

Low HR may cause low CO even in normal hearts, because $CO = SV \times HR$. But at excessively high HRs, there may be insufficient time to adequately fill the ventricle during diastole, leading to decreased left VEDP (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 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 (BNPs) 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. The single best instance when a BNP can be of value is in distinguishing the cause of dyspnea in a patient in whom both chronic obstructive pulmonary disease (COPD) and CHF are possibilities.

12. How do I interpret BNP levels?

- Less than 100 pg/mL is unlikely to be CHF.
- 100 to 500 pg/mL may be CHF.

- 500 pg/mL is most consistent with CHF. (There is difficulty interpreting elevated BNP when a patient has known severe CHF.)

13. How do patients with CHF appear upon arrival in the ED?

Patients with CHF come to the ED in one of two ways:

1. With subacute gradual worsening, with slow progression of symptoms and signs
2. With 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, whereas 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 experience symptoms?

Patients develop acute shortness of breath and generally are fighting for air. These patients sit upright to decrease venous return (preload) and to redistribute edema to the dependent parts of the lungs. They cough up frothy, red-tinged sputum. Auscultation of the lungs reveals wet rales throughout, and sometimes wheezes (resulting from 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 (CPAP/BiPAP) has decreased the need for intubation of airways in 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 reevaluate the patient to assess the need for 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, CPAP, 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; however, it also dilates coronary arteries, so it may be especially helpful in the setting of coronary artery disease. Diuretics should only be administered to 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 taking diuretic medication, 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 reevaluation of the patient with an eye toward increasingly aggressive measures is important.
3. Nitrates are first-line pharmacotherapy.
4. Diuretic drugs 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 spontaneously 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 μg/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 μg/kg/min are sufficient.

19. What about giving positive inotropic drugs?

Digoxin has traditionally been used in the treatment of chronic CHF, but has little role in the treatment of acute pulmonary edema. Increasingly, the effectiveness of digoxin has been questioned, even in outpatient settings. 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. All of these agents may increase myocardial oxygen demand, and this can be deleterious in patients who owe their exacerbation to the mechanism of cardiac ischemia. 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's condition 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 workup 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 networks, and attend follow-up appointments with their primary care physicians. 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 a trip to the ED. Causes of acute CHF exacerbations are principally cardiac and include acute myocardial infarction, dysrhythmias, 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, but they must be used cautiously because of their potential effects on cardiac contractility. Diuretics also are beneficial, especially in patients with volume overload. Digoxin has long been used as a means of improving the symptoms of chronic CHF, although it has been historically acknowledged that it does not have any impact on overall mortality. Recently though, the role of this medication has been increasingly questioned, with conflicting evidence. Some studies suggest that the drug may benefit patients in terms of decreased hospitalizations, and other studies suggest worse outcomes. Consequently, the popularity of digoxin may be waning in this setting. 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 and leads to increased afterload. Stimulation of aldosterone causes sodium retention, 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, such as in valvular heart disease. Patients with mild disease who 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.

Acknowledgment

The editors gratefully acknowledge the contributions of Rodney W. Smith, MD, author of this chapter in the previous edition.

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QUESTIONS

1. Which of the following may worsen left ventricular CO?
   a. Increased preload
   b. Increased afterload
   c. Increased contractility
   d. Decreasing HR from an excessively high rate
   The correct answer is *b.*
2. Which of the following medication classes is unlikely to be of benefit to patients with acute (flash) pulmonary edema?
   a. Nitrates
   b. Inotropic agents
   c. Diuretics
   d. Opiates
   The correct answer is *c.*
3. Of the following possible causes of acute CHF exacerbations, which is the least common?
   a. Acute, severe hypertension
   b. Medication noncompliance
   c. Acute myocardial ischemia
   d. Dysrhythmias
   The correct answer is *a.*

ISCHEMIC HEART DISEASE

Danya Khoujah, MBBS, and Amal Mattu, MD

CHAPTER 31

1. How is ischemic heart disease classified?

- Chronic stable ischemic heart disease
  - Asymptomatic
  - With angina
  - After myocardial infarction (MI), with or without angina
- Acute coronary syndrome (ACS)
  - Unstable angina
  - Acute non–ST-segment elevation MI (NSTEMI)
  - Acute ST-segment elevation MI (STEMI)

2. How do patients with acute ischemic heart disease experience symptoms?

The most common presenting symptom is chest pain. Typically, ischemic pain is described as heaviness, pressure, tightness, or squeezing, although less commonly it is described as sharp or aching. Patients may describe their pain as indigestion, which leads to the common misdiagnosis of reflux esophagitis. The discomfort also may be felt anywhere on the chest, the right or left shoulder or arm, the throat or jaw, or the upper abdomen. Chest pain often may radiate to one of these locations as well. Less commonly, patients may simply have pain in other more unusual locations (e.g., left ear or upper back). Associated symptoms include dyspnea, nausea, vomiting, sweats, lightheadedness or syncope, palpitations, or severe malaise. However, one third of patients may not experience pain and just present with these associated symptoms—in this case termed *angina equivalent* or *atypical angina.* This is more common in the elderly, women, and patients with diabetes, therefore requiring clinicians to be more vigilant in their assessment.

3. Which descriptors have the highest predictive value for true ACS?

Chest pain that radiates, chest pain accompanied by sweating or vomiting, and chest pain that is associated with exertion all have high predictive values for ACS.

4. To understand the discomfort better, what information should be obtained?

Use the *OLDCAAAR* mnemonic when obtaining the history of present illness:

Onset: When did the pain begin? Was it abrupt or gradual?
Location
Duration: If intermittent, how long do the episodes last?
Character: Is the pain sharp, dull, aching, pressure, or squeezing?
Alleviating and aggravating factors: What makes the pain better? What makes the pain worse?
Associated symptoms: Dyspnea, nausea, vomiting, lightheadedness, diaphoresis
Activity at onset
Radiation: Does the pain radiate to any other part of the body?

5. Describe the typical features of chest discomfort in stable angina.

Typically, patients with stable angina have discomfort during exertion that is relieved within minutes by rest or nitroglycerin (NTG). The degree of exertion bringing on the discomfort (and rest/NTG required for relief) is predictable and constant (or stable) over time. The electrocardiogram (ECG) and cardiac markers are unchanged.

6. How do patients with unstable angina experience symptoms?

Patients with unstable angina usually have similar pain to those with stable angina, but it occurs with progressively less exertion or at rest, or is new in onset. Patients with unstable angina who have pain at rest also typically have pain with exertion. The ECG may show some ischemic changes, but the cardiac markers are normal.

Abstract

Acute coronary syndrome (ACS) is one of the most common truly emergent conditions encountered by the emergency medicine physician. Its recognition and timely treatment will ensure the best possible outcome.

Keywords:

cardiac, ischemia, infarction, acute coronary syndrome (ACS), unstable angina, myocardial infarction (MI), ischemic heart disease, aspirin, β-blockers, antiplatelet, anticoagulation, electrocardiogram (ECG), electrocardiography

7. What is Prinzmetal angina?

Prinzmetal angina is caused by coronary artery spasm. Patients with Prinzmetal angina typically have pain at rest, usually in the early morning hours, and often do not have exertional discomfort. True vasospastic angina is uncommon.

8. How does the pain of MI differ from that of angina?

Patients with acute MI typically have pain that is more severe than any preceding angina. It may be described as crushing, or it may be atypical (see Question 2). It is caused by myocardial necrosis. The ECG may show ST elevation (STEMI), or the cardiac markers may be elevated (NSTEMI)

9. 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 often with unstable angina or acute MI and should raise concern, because it does not typically 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. The presence of diaphoresis or vomiting significantly increases the likelihood of true MI or unstable angina.

10. Is there anything different about evaluating elderly patients?

The older the patient, the more atypical the symptoms become. Over the age of 70 years, only half of all patients will feel chest pain when they experience cardiac ischemia or acute MI. On the other hand, dyspnea is very common. Other atypical presentations that become more common with advancing age include weakness, vomiting, and syncope.

11. Are there other groups at high risk for atypical presentation?

- Patients with diabetes
- The elderly
- Women

These groups tend to experience "anginal equivalents," such as dyspnea, vomiting, extreme fatigue, or lightheadedness rather than classic ischemic chest pain.

12. What are the risk factors associated with ischemic heart disease?

- Traditional risk factors: Male gender, age, smoking, hypertension, hyperlipidemia, diabetes mellitus, family history, menopause, and cocaine abuse
- Non-traditional risk factors: Antiphospholipid syndrome, rheumatoid arthritis, systemic lupus erythematosus (SLE), and HIV, among others

13. Should demographic features and the presence or absence of coronary risk factors change my mind about the diagnosis?

No. Risk factors are less important than the history of presenting illness or ECG changes. A young woman with no risk factors but with typical symptoms and 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, even though he may turn out to be less likely to actually have the disease.

14. List the key elements of the initial evaluation of a patient with a suspected ACS.

- The patient should be outfitted with a cardiac monitor and have reliable intravenous (IV) access established.
- Supplemental oxygen should be applied only if the patient is hypoxic (blood oxygen saturation [SpO_2] <94%) or complaining of dyspnea. Unnecessary oxygen may be harmful.
- The patient should have an ECG performed and read as soon as possible, ideally within 10 minutes of arrival to the ED.
- Vital signs are crucial. The presence of abnormal vital signs, especially hypotension, should be addressed 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, and also for assessing the presence of any complications of acute ischemia/infarction, such as acute heart failure or valvular dysfunction.
- Administer chewable aspirin (182 to 324 mg [two to four 81 mg chewable tablets]) unless contraindicated (see Question 34).

15. What is the significance of abnormal ST-segment changes on an ECG?

Abnormal ST-segment changes may or may not represent ischemic cardiac injury. ST elevation may represent infarction. The current of injury that accompanies STEMI is typically a convex-upward elevation of the ST segment (resembling a tombstone). However, the elevation may be horizontal or concave upward instead. Other causes of ST elevation are as follows:

- Left ventricular hypertrophy
- Left bundle branch block
- Benign early repolarization
- Acute pericarditis
- Hyperkalemia
- Hypercalcemia
- Hypothermia
- Left ventricular aneurysm
- Ventricular rhythm
- Brugada syndrome
- Acute cerebral hemorrhage

ST depression is typically caused by cardiac ischemia. A particularly high-risk condition is when a patient demonstrates ST depression in multiple leads in conjunction with ST elevation in lead aVR. This combination of ST findings is predictive of left main coronary artery occlusion, which just as dangerous as a true ST-elevation myocardial infarction (STEMI). Such patients should be treated aggressively. In addition to cardiac ischemia, ST depression may be caused by such things as ventricular hypertrophy, drugs (e.g., digoxin), and electrolyte abnormalities.

16. How do I differentiate ST elevation owing to ischemia from other causes of ST elevation?

ST elevation caused by infarction may display one or more of the following:

- Morphology: Convex upward ST ("tombstone") elevation
- Presence of reciprocal changes: Presence of ST depression in another area of the ECG, in the opposite side of the heart
- ST elevation caused by infarction is dynamic; serial ECGs will show gradual worsening over time

17. What is the typical course of ECG changes in ischemic cardiac injury?

The first ECG abnormality is often described as development of hyperacute T waves (Fig. 31-1). This consists of straightening of the initial portion (upward slope) of the T wave, as well as broadening of the base and an increase in the amplitude of the T wave. These T-wave changes may occur within the first minutes of ischemia. Next, the ST segment displays elevation (injury pattern; Fig. 31-2) or depression (ischemic pattern). ST-segment changes tend to occur in the first hour of ischemia, but they may be delayed by 1 or more hours in some cases. Therefore serial ECGs are recommended

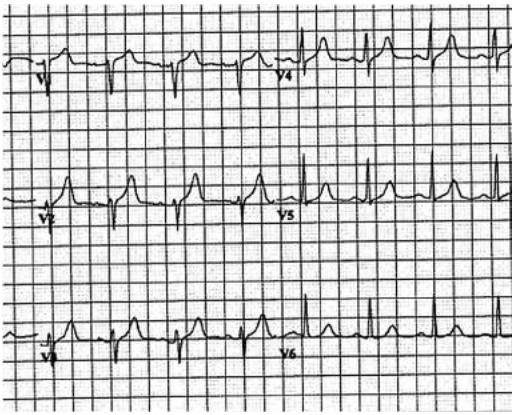

Figure 31-1. Hyperacute T-waves.

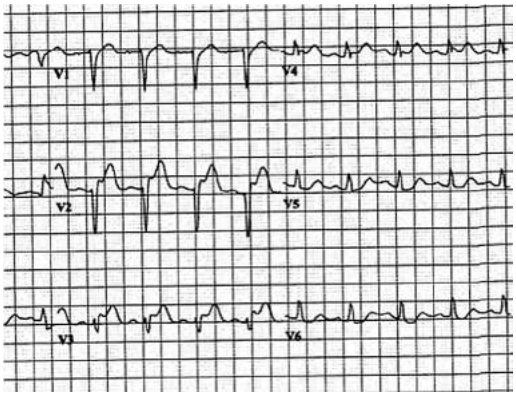

Figure 31-2. ST elevation in V2-V4.

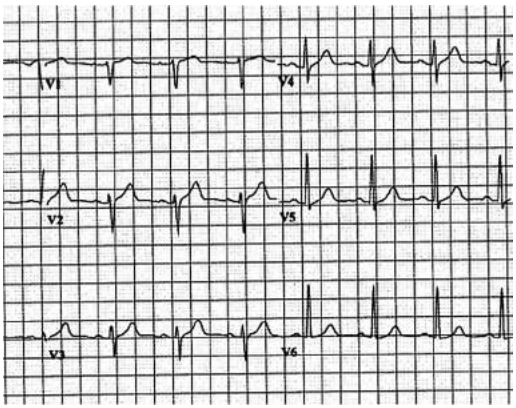

Figure 31-3. Normalization of ST elevation.

during the first few hours of symptoms if the initial ECGs are nondiagnostic. Q waves may develop within 2 to 3 hours of infarction. Significant Q waves should be at least 40 milli seconds in width and at least one third of the height of the R wave. 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.

18. Can the ECG be normal while a patient is having cardiac ischemia or an acute MI?

Yes. Although serial ECGs showing evolving changes are diagnostic for acute MI in more than 90% of patients, 20% to 50% of initial ECGs show nonspecific abnormalities, and up to 10% of ECGs may be completely normal (Fig. 31-3). The initial ECG may be diagnostic for acute MI in only half of all patients. Therefore serial ECGs are often helpful.

19. Are cardiac markers useful in the ED?

Maybe. Cardiac markers typically include troponin, creatine kinase MB (CK-MB), and myoglobin. Troponin is the most commonly used cardiac marker. The current generation assay doesn't demonstrate rising levels until 3 to 6 hours after the onset of infarction. A newer generation of "highly sensitive troponin," which is expected to be available in the United States by the end of 2015, will demonstrate elevations within 1 to 2 hours of infarction. It is critically important to

remember, however, that troponins are reliable markers only for infarction and are not reliable at detecting cardiac ischemia (unstable angina). Additionally, as the sensitivity of troponin assays has increased, the specificity has fallen.

20. Can troponin be elevated in other conditions?

Troponin elevations are noted in numerous noninfarction cardiac and noncardiac conditions.

- Acute and chronic heart failure
- Cardiomyopathy
- Pericarditis, myocarditis
- Left ventricular hypertrophy
- Severe hypertension
- Tachydysrhythmias such as rapid atrial fibrillation and supraventricular tachycardia
- Sepsis
- Stroke (hemorrhagic and ischemic)
- Strenuous endurance exercise activities

Troponin elevations may also be the result of chemotherapeutic agents, among other causes. Because of the lack of specificity of troponin testing, the World Health Organization recommends that troponins be used to diagnose acute MI only in patients in whom there is a reasonably high clinical suspicion, and that the diagnosis of acute MI using troponins should be based not on a single elevated level but rather on a rise in troponin levels of at least 20% on serial testing over the course of a few hours. In other words, single troponin determinations in isolation are generally not sufficiently sensitive or specific to make a diagnosis. You should not wait to see an elevated troponin level before making a decision to treat for ACS.

21. How can echocardiography be useful in ED patients with suspected ACS?

Its sensitivity in the acute setting is limited. The presence of cardiac wall motion abnormality is evidence that supports the diagnosis of ischemia, although it may be the result of an old rather than acute infarction. It may also provide information about complications, such as acute valvular dysfunction or acute heart failure with low ejection fraction. Echocardiography may also be helpful at distinguishing acute MI from pericarditis (looking for pericardial effusion) or large pulmonary embolus (looking for a hyperdynamic distended right ventricle).

A negative echocardiogram in the setting of a typical history and ECG does not rule out the diagnosis of an ACS. Waiting for the results of an echocardiogram may unnecessarily delay treatment.

22. What other diagnoses should be considered in a patient with chest pain?

The patient's history is paramount, and the following life-threatening conditions should be considered first:

- Acute aortic dissection
- Pulmonary embolism
- Tension pneumothorax
- Esophageal rupture

Additional diagnoses include the following possibilities:

- Gastrointestinal (GI): Esophagitis, esophageal spasm
- Pulmonary: Pleuritis, pericarditis, pneumonia
- Chest wall pain: Musculoskeletal, herpes zoster, or cervical or thoracic nerve root compression
- Psychiatric: Anxiety or depression

23. What are the indications for reperfusion therapy in acute MI?

Reperfusion therapy includes either percutaneous coronary intervention (PCI) or thrombolytics.

- The patient should have symptoms concerning for acute MI

PLUS any of the following ECG patterns that persist despite use of sublingual NTG:

- ST elevation in leads V2-V3 greater than 2 mm for men older than 40 years of age, or greater than 2.5 mm in men younger than 40 years of age, or greater than 1.5 mm in women; or ST elevation greater than 1 mm in any other two anatomically contiguous leads

OR

- Evidence of acute posterior MI (diagnosed by noting ST-segment depression with tall R waves and upright T waves in leads V1-V3, or by ST-segment elevation in posteriorly placed leads)

OR

- A left bundle branch block pattern with evidence of concordant Sgarbossa criteria (concordant ST-segment elevation in any lead, or concordant ST-segment depression in any of leads V1, V2, or V3)

Of note, a new left bundle branch block is no longer an indication for acute reperfusion.

24. What if persistent ST elevation is not present?

Acute reperfusion therapy (PCI or thrombolytics) is not indicated if the patient does not have the ECG criteria noted in Question 23. If those ECG findings resolve, the patient's case should still be managed aggressively with the therapies noted in Question 34 (except for the reperfusion therapies), as if he or she has unstable angina or NSTEMI.

25. What is the preferred method of reperfusion therapy in acute MI with STEMI—thrombolytic therapy or PCI?

PCI is preferred over thrombolytic medication if done within 90 minutes of arrival. PCI shows a greater reduction in mortality, lower rates of reocclusion over the subsequent few days, and lower risks of intracranial bleeding. If PCI cannot be accomplished in such a timely manner, thrombolytic drugs should be used. There are notable exceptions to this rule, however. Thrombolytics are somewhat ineffective for patients with cardiogenic shock (see Question 28) and also for patients whose symptoms have been ongoing for more than 6 hours. For these patients, the time window of benefit for PCI over thrombolytics can be extended. Management decisions for these patients should be made in conjunction with the interventional cardiologist.

26. What if cardiac intervention is not available on site?

If transfer from one hospital to a facility capable of intervention is needed for treatment of STEMI, the permissible time window for transfer and intervention is 120 minutes. Note that in this scenario, balloon inflation and not just the transfer should occur within 120 minutes. If intervention cannot be accomplished within this estimated window, thrombolytics should instead be given as soon as possible.

27. How do you choose which thrombolytic agent to use?

The choice of which thrombolytic to administer is of little importance. Streptokinase, alteplase, reteplase, and tenecteplase are all used worldwide and have similar efficacy. Typically hospitals will tend to stock one of the forms of thrombolytic based on cost, and physicians will simply choose the drug that they have on their formulary. Streptokinase has the advantage of being the least expensive, but has a very slightly higher rate of intracranial hemorrhage compared with the others. Reteplase and tenecteplase have become more popular than alteplase, because they can be administered in bolus form rather than an infusion and with a varying dose schedule. Regardless of which agent is chosen, it should be given within 30 minutes of arrival. Of note, exposure to streptokinase in the previous 6 months or streptococcal infection in the previous 6 months are reasons to use another agent.

28. What is the preferred therapy for cardiogenic shock?

The only therapy shown to decrease the historically high mortality associated with this syndrome is invasive therapy (PCI or, in some cases, bypass surgery). PCI should be performed without delay. The added benefit of immediate transfer to a catheterization laboratory is that this facilitates insertion of an intraaortic balloon pump (IABP). An IABP is thought to be superior to pharmacologic therapy for supporting blood pressure, decreasing afterload, and augmenting cardiac output. Vasopressors (such as norepinephrine or dopamine) should be used while these measures are being arranged.

Of note, hypotension caused by right ventricular infarction should be considered in patients with an acute MI and hypotension. This is not a true form of cardiogenic shock, but clinicians often confuse the two conditions. Right ventricular infarction should be suspected whenever hypotension accompanies an acute inferior MI. The presence of jugular venous distention and clear lungs are important clues to this diagnosis. These patients do not need vasopressors or inotropic support. Instead, their cases can be managed successfully with aggressive volume replacement with IV fluids.

29. List the contraindications to thrombolytic therapy.

Absolute contraindications

- PCI immediately available
- Active bleeding
- Suspected aortic dissection

- Ischemic stroke within the past 6 months
- History of hemorrhagic stroke
- Intracranial or intraspinal surgery or trauma in the past 8 weeks
- Intracranial or intraspinal neoplasm, aneurysm, or arteriovenous (AV) malformation

Relative contraindications
- History of GI bleed
- Surgical or invasive procedure within the past 3 weeks
- Significant trauma within the past 4 weeks
- Bleeding diathesis, thrombocytopenia (<100,000/mm^3)
- Pregnancy or 10 or fewer days postpartum
- Prolonged (>10 minutes) or traumatic cardiopulmonary resuscitation (CPR)
- Hemorrhagic ophthalmic condition, especially diabetic retinopathy
- Active cavitary lung disease
- Known allergy to the thrombolytic agent

30. 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 evaluated with a careful history, examination of peripheral pulses, and chest radiography. If significant concern for aortic dissection still exists, a chest computed tomography (CT) scan with IV contrast should be obtained to exclude the diagnosis before thrombolytics are given. Pericarditis can be excluded by carefully listening for a rub and examining the ECG for widespread, concave upward ST elevation.

31. What is the risk for fatal complications of thrombolytic therapy for acute MI?

Intracranial hemorrhage occurs in 2% of patients and is fatal in 0.5% of treated patients. Angioedema is rare, but can be fatal.

32. What is the role of NTG?

NTG is commonly used in patients with ongoing ischemic chest pain. NTG produces a reduction in preload and coronary vasodilation, and might also improve coronary perfusion. NTG produces effective improvement in ischemic chest pain in most patients. In addition, NTG can be used to help control severe blood pressure in these patients. NTG can be administered sublingually at a dose of 400 μg every 3 to 5 minutes or can be given as an IV infusion. NTG should be used cautiously, if at all, in patients with borderline or low blood pressure, and it should be withheld in patients with right ventricular infarction because it can produce or worsen hypotension. In addition, NTG has been found to produce precipitous decreases in blood pressure when given to patients that have recently taken sildenafil or other similar medications for erectile dysfunction.

33. Is there any use for morphine in patients with acute MI?

Morphine has been used for decades to decrease pain in patients with acute MI. Pain produces a release of catecholamines, which can be detrimental to patients who are ischemic. However, a large nonrandomized study demonstrated an association between the use of morphine and worse outcome in patients with acute MI. The analgesic effect of morphine might also mask underlying ongoing ischemia. Therefore morphine should be used with caution. Ideally, ischemic pain should be treated with NTG. However, if the ischemic pain is intractable, IV morphine sulfate can be added in increments of 2 to 4 mg to alleviate these symptoms. Other aggressive measures, including PCI if needed, should also be taken to relieve ischemia.

34. What other medications are useful adjuvants to reperfusion therapy?

- Aspirin: Unless the patient has had a life-threatening allergic reaction to aspirin, it should be given immediately, because it reduces mortality independent of other therapies via its irreversible antiplatelet effects. The dosage is 162 to 324 mg of chewable aspirin; enteric-coated forms should be avoided because they have very slow absorption rates.
- Other antiplatelet agents: Administer other agents after consultation with a cardiologist, because a great deal of debate exists regarding their use. The two major groups of additional antiplatelet agents are the thienopyridines (clopidogrel, ticagrelor, and prasugrel) and the glycoprotein IIb-IIIa receptor antagonists (G2b3aRAs), also referred to as *glycoprotein inhibitors (GPIs)*. Thienopyridines are most commonly used, because they have lower bleeding complications and are easier to administer than the G2b3aRAs. Clopidogrel is the most commonly used and oldest of the

thienopyridines, although ticagrelor has gained popularity. Either of these medications can be given before or at the time of reperfusion therapy. Prasugrel is best reserved for use in the cardiac catheterization laboratory, and should not be given to patients with a history of stroke or transient ischemic attack. The G2b3aRAs were more commonly used before the thienopyridines gained popularity; however, some cardiologists still combine them with the thienopyridines in patients who are to receive PCI. This combination use may produce an increase in coronary artery potency at the expense of increased bleeding complications. The G2b3aRAs (abciximab, eptifibatide, and tirofiban) are generally only indicated in patients who will be having PCI.

- Some cardiologists will combine both groups (thienopyridines and G2b3aRAs) in patients receiving PCI; however, it may produce an increase in coronary artery potency at the expense of increased bleeding complications.
- Heparin and other anticoagulants: Anticoagulant medications are used in patients whether they undergo PCI or thrombolytics. With alteplase, initiation of heparin is imperative at least 1 hour before the completion of the thrombolytic infusion. With streptokinase, administration of heparin should be delayed 4 to 6 hours. Anticoagulants should be continued in the hospital for at least 48 hours after a patient has had thrombolytics or PCI. In addition to unfractionated heparin, fondaparinux, low–molecular-weight heparin, and enoxaparin have been shown to be acceptable alternatives. Bivalirudin is another option for anticoagulation in patients receiving PCI but is less commonly used.

35. When should β-blockers be given?

IV β-blockers were traditionally used within the first hours of acute MI to decrease heart rate, decrease myocardial oxygen demand, and decrease in-hospital sudden death. It was thought that they would decrease infarct size and mortality. However, large studies have suggested that the routine early use of IV β-blockers is actually associated with a slightly increased risk of the development of cardiogenic shock. Current guidelines recommend that β-blockers should be given orally within the first 24 hours of admission in order to obtain the benefits of β-blockade without the risk of development of cardiogenic shock. IV β-blockers should be reserved for patients with severe hypertension or tachydysrhythmias, such as rapid atrial fibrillation. The most common β-blockers used in the setting of acute MI are metoprolol and atenolol. Contraindications include heart failure, bradycardia (heart rate <55 beats per minute), advanced atrioventricular blocks, and bronchospasm.

36. What other dysrhythmias occur with acute MI?

- Ventricular irritability: Secondary causes such as drugs, electrolyte imbalance, and hypoxia should always be sought and treated.
  - Isolated PVCs and nonsustained ventricular tachycardia do not need treatment.
  - Sustained ventricular tachycardia (lasting 30 seconds or more) should be treated with lidocaine or amiodarone.
  - Ventricular fibrillation is a common cause of death after MI.
  - Accelerated idioventricular rhythm (heart rate 60 to 120 beats per minute) should simply be observed; it tends to resolve on its own within seconds to minutes. Treatment with ventricular antiarrhythmics can cause asystole.
- Bradydysrhythmias, such as second- or third-degree heart block
  - If accompanying inferior MI, bradydysrhythmias are usually transient and do not require a temporary pacemaker.
  - If accompanying anterior MI, bradydysrhythmias usually require a temporary pacemaker.
  - A prophylactic temporary pacemaker should be considered when severe conductive system disease (bifascicular block or left bundle-branch block plus first-degree block) accompanies an anterior acute MI.

37. Which patients with unstable angina are at highest risk for MI and benefit from more aggressive treatment?

Patients with the following conditions are thought to benefit more from more aggressive medical treatment and early catheterization:

- ECG changes: Transient or fixed ST-segment depression or T-wave inversion, especially when these changes are in anterior leads
- 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 previous 24 hours

38. What is the management of unstable angina and NSTEMI?

The management is similar to that for STEMI, except for reperfusion therapy and G2b3aRAs. This means that all patients with unstable angina and NSTEMI get aspirin (clopidogrel may be given after consultation with a cardiologist), NTG for pain and severe hypertension, anticoagulation if there is no contraindication, and β-blockers within 24 hours.

39. Which is better, low–molecular-weight heparin or unfractionated heparin?

Unfractionated heparin and low–molecular-weight heparin, enoxaparin, are equally effective and have a similar class rating. The real difference is in the practical considerations and the consulting cardiologist's preference. The advantage of low–molecular-weight heparin is that it can be given in the ED as a single bolus and does not require an infusion pump. The major disadvantage is that it is difficult to reverse if there is a bleeding complication or an invasive strategy is planned. Therefore some cardiologists prefer unfractionated heparin, because it enables them to measure levels of anticoagulation (international normalized ration [INR] levels).

KEY POINTS

1. Most predictive features of true cardiac ischemia
   - Pain that radiates
   - Pain associated with vomiting
   - Pain associated with diaphoresis
   - Pain that predictably occurs or worsens with exertion
2. Key points in the evaluation of patients with possible acute cardiac ischemia or MI
   - A good history of present illness is absolutely essential.
   - Chest pain is not universally present. Dyspnea is common in patients who do not have chest pain.
   - Three groups of patients are at high risk for atypical presentations of cardiac ischemia: elderly patients, patients with diabetes, and women.
3. ECG features most consistent with STEMI (versus a mimic)
   - ST elevation that is convex upwards (like a tombstone)
   - ST elevation that is associated with reciprocal ST-segment depression
   - Evolving changes of the ST segments and T waves
4. PCI is preferable to thrombolytics for emergent management of STEMI unless there is an anticipated delay of more than 90 to 120 minutes to balloon inflation.
5. The most important medication to be given to virtually all patients with suspected cardiac ischemia or MI is aspirin.

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QUESTIONS

1. Which of the following patient groups are at high risk for an atypical presentation of cardiac ischemia?
   a. Young men
   b. African Americans
   c. Patients with asthma
   d. Women
   The correct answer is *d.*
2. Which of the following factors is most highly associated with true cardiac ischemia?
   a. Chest pain associated with diaphoresis
   b. Chest pain that is described as pressure
   c. Chest pain associated with dyspnea
   d. Chest pain associated with lightheadedness
   The correct answer is *a.*
3. Which of the following medications is the most important medication that should be given to virtually all patients with cardiac ischemia or MI?
   a. β-Blockers
   b. Heparin
   c. Aspirin
   d. Thrombolytics
   The correct answer is *c.*
4. A patient comes to the ED with chest pain and diaphoresis. An ECG demonstrates 1 mm of ST depression in inferior and anterolateral leads, and 2 mm of ST elevation in lead aVR. What is the most likely diagnosis?
   a. Acute pericarditis
   b. Massive pulmonary embolism
   c. Occlusion of the right coronary artery
   d. Occlusion of the left main coronary artery
   The correct answer is *d.*

CARDIAC DYSRHYTHMIAS, PACEMAKERS, AND IMPLANTABLE DEFIBRILLATORS

CHAPTER 32

Christopher B. Colwell, MD, and Karl Marzec, 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 is to provide electricity (perform electrical cardioversion) if the heart rate is fast, and if the heart rate is slow, pace the patient with a pacemaker.

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 bradydysrhythmias?

Do not treat bradycardia if the patient is hemodynamically stable and asymptomatic. Always treat the patient, not a numeric test value. 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; 0.02 mg/kg in a neonate) 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 tachydysrhythmias?

Any unstable patient with a tachydysrhythmia 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 tachydysrhythmias 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 he or she has narrow- or wide-complex tachyarrhythmia.

Abstract

This chapter describes the presentation and management of common cardiac rhythm abnormalities, as well as pacemaker use and malfunction.

Keywords:

bradydysrhythmias, tachydysrhythmias, ventricular tachycardia (VT), supraventricular tachycardia (SVT), pacemaker, external transcutaneous pacing, transvenous pacing, automatic implantable cardioverter defibrillator (AICD), narrow-complex tachycardia, wide-complex tachycardia, aberrancy, synchronized cardioversion, monophasic defibrillation, biphasic defibrillation, twiddler's syndrome

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 at 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, or 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 dysrhythmia 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 regular 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 to 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 to 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 upon receiving 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 should not use adenosine or a calcium channel blocker for a narrow-complex tachycardia?

The one situation in which it would be potentially dangerous to use these agents is AF in the setting of Wolff-Parkinson-White (WPW) syndrome. 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 syndrome can present as narrow- or wide-complex tachycardia. It is difficult to determine on the ECG whether someone has WPW syndrome if the rhythm is very fast, but if the patient has a known history of the disorder, do not give adenosine or a calcium channel blocker. Procainamide or synchronized cardioversion 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 AV node, it does not use the Purkinje "superhighway" of conduction and therefore takes longer to activate the ventricular mass—longer than 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 coming to the ED with a wide-complex tachycardia, 70% to 90% have VT, and only 10% to 30% have SVTs 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.

Treatment of torsades de pointes is unsynchronized shock if the patient is unstable. If the patient is hemodynamically stable, treat torsades de pointes with magnesium (1- to 2-g bolus over 5 to 10 minutes); if there is a prolonged QT interval consider isoproterenol or ventricular pacing.

15. Does VT always cause a patient to be hemodynamically unstable?

No, hemodynamic status should not be used to determine the nature of a wide QRS tachycardia. Do not assume that a wide-complex tachycardia cannot be 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 common, an impulse of supraventricular origin that travels through the ventricle in an aberrant fashion also can be wide and is called a *supraventricular rhythm with aberrancy*. One example, as discussed in Question 11, is AF in the setting of WPW; this is a supraventricular dysrhythmia 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 that the rhythm is 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 32-1.

19. What does amiodarone do?

Amiodarone is a class III antidysrhythmic drug 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?

Synchronized cardioversion is synchronization of delivered energy to match the timing of the QRS complex. This reduces the chance that a shock will induce VF, which can occur when electrical

Table 32-1. Treatment of Wide-Complex Tachycardia

CLINICAL SITUATION	TREATMENT
Unstable patient	Cardioversion
Wide-complex tachycardia known to be SVT with aberrancy	Adenosine (6 mg IV push followed by 12-mg IV 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 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

Modified 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.
IV, Intravenously; *SVT,* supraventricular tachycardia.

energy impinges on the relative refractory portion of the cardiac electrical activity (downward 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 synchronization control button, and look for markers on the R waves indicating the synchronization mode is functioning and capturing the QRS complex and not the T wave.
- You may need to adjust the R wave again until the synchronization 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 lb of downward pressure.)

23. Does it make sense to use cardioversion with asystole?

Strictly speaking, no, it does not. 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 no reason for cardioversion. Although the American Heart Association currently does not recommend routine shocking during 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 give anticoagulants to a patient with AF before 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's blood is fully anticoagulated.

25. Should I 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 result in any statistically significant difference in overall survival. A 2006 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?

A pacemaker is 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. Long-term therapy requires the placement of a surgically implanted device. It is usually 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 bradydysrhythmias and prevention of bradycardia-dependent malignant dysrhythmias. In symptomatic or unstable patients who do not 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 greater than 3 seconds
- AV nodal block
- Second-degree AV block (Mobitz type I block)
- Complete heart block

Infranodal block
- New bifascicular block associated with acute myocardial infarction (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 cardiac capture is achieved.

29. State the limiting factors in the use of external pacemakers.

Skeletal muscle contraction can be quite uncomfortable for the patient, and often limits use of external pacemakers. Placing electrodes over areas with the 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 the electrical 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 V1, with another clip attached to the pacing wire. When a current of injury (ST elevation) is seen on the monitor, indicating contact with the heart, 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 performing defibrillation or cardioversion in 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. As of 2014, 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 (Table 32-2). 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. Fourth and fifth letters may be added to describe whether the pacemaker is programmable and whether

Table 32-2. 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)

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., it 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.

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?

The most common cause of malfunction is lead dislodgement. Most pacemaker failures are the result of problems with the electrodes or the wires, and not the battery or the pulse generator. Because of greater technologic sophistication, patients with pacemaker problems come the ED much less commonly now than in the past.

39. What is the most reliable indicator of pacer malfunction?

A good indication of malfunction is a rate that are usually inappropriate for the patient's paced heart. 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:

1. AOO (atrium paced)
2. VOO (ventricle paced)
3. 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 after a native QRS complex, it may be because the pacer is undersensing. See Table 32-3 for a description of common pacemaker malfunctions.

42. What is pacemaker syndrome?

Pacemaker syndrome is 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 32-3. Malfunctions of Permanent Pacemakers

COMPLICATION	DESCRIPTION
Oversensing	Occurs when a pacer incorrectly senses electrical activity and is inhibited from correctly pacing. This may be caused by muscular activity, electromagnetic interference, or lead insulation breakage.
Undersensing	Occurs when a pacer incorrectly misses intrinsic depolarization and paces despite intrinsic activity. This can the result of 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 caused by 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?

Twiddler's syndrome is the most common cause of late lead dislodgement. It occurs when the patient twists, or twiddles, the pulse generator within 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 tachydysrhythmia. 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 tachycardia secondary to rapid ventricular pacing is known as a *runaway pacemaker.* 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.

Table 32-4. Malfunctions Associated with an Automatic Implantable Cardioverter Defibrillator

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 has 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

Modified from Higgins GL 3rd: The automatic implantable cardioverter-defibrillator: management issues relevant to the emergency care provider. *Am J Emerg Med* 8:342–347, 1990.
AICD, Automatic implantable cardioverter defibrillator; *MRI*, magnetic resonance imaging.

47. Can a patient with a permanent pacemaker undergo defibrillation?

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 AICD is a specialized device designed to treat a cardiac tachydysrhythmia. If the device senses a ventricular rate that exceeds the programmed cut-off 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 32-4.

50. Name the most common 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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QUESTIONS

1. Which patient requires electrical cardioversion?
   a. A 50-year-old male with 1 hour of chest pain, bradycardia with a heart rate in the 40s, and ST elevations in the inferior leads on his ECG?
   b. A 75-year-old female with a history of hypertension who comes to the ED with palpitations. The monitor shows an irregular narrow-complex tachycardia with a heart rate of 120 beats per minute.
   c. A 60-year-old male with a history of a myocardial infarction who has shortness of breath, blood pressure of 88/50, and a wide-complex tachycardia.
   d. A 35-year-old female who admits to drinking several energy drinks before arrival in the ED, is complaining of shortness of breath, has a blood pressure of 110/90, and has a narrow-complex tachycardia with a heart rate of 150 beats per minute.

   The correct answer is *c.*
2. Which patient condition and treatment plan are not appropriate?
   a. Stable VT and amiodarone
   b. AF with WPW syndrome and diltiazem
   c. SVT and adenosine
   d. Pulseless VF and electrical defibrillation

   The correct answer is *b.*
3. How does placing a magnet on a patient's chest with a pacemaker affect the pacemaker function?
   a. The magnet temporarily turns on the defibrillation action.
   b. The pacing function is disabled.
   c. Magnets only affect defibrillators and not pacemakers.
   d. The pacemaker reverts to a fixed rate and mode.

   The correct answer is *d.*

HYPERTENSION, HYPERTENSIVE CRISIS, AORTIC DISSECTION, AND AORTIC ANEURYSMS

CHAPTER 33

Madonna Fernández-Frackelton, MD, FACEP

1. What is the description of hypertension (HTN) according to the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) report?

- Normal blood pressure (BP): Lower than 120/80 mm Hg
- Prehypertension: Systolic BP (SBP) 120 to 139 mm Hg or diastolic BP (DBP) 80 to 89 mm Hg
- Stage 1 HTN: SBP 140 to 159 mm Hg or DBP 90 to 99 mm Hg
- Stage 2 HTN: SBP greater than 160 mm Hg or DBP greater than 100 mm Hg

2. How does the JNC8 report differ from the JNC7 report?

- JNC8 does not address definitions, but rather thresholds for treatment.
- There is strong evidence to treat patients older than 60 years with a goal BP of less than 150/90 mm Hg.
- There is strong evidence to treat patients age 30 to 59 years with a DBP goal of less than 90 mm Hg.
- There is insufficient evidence for a goal SBP in patients younger than 60 years or for a goal DBP in patients younger than 30 years. Expert recommendation in these age groups is less than 140/90 mm Hg.

3. What is the difference between primary and secondary HTN?

- Primary, or essential, HTN accounts for more than 90% of patients with HTN. Its 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 that increase intracranial pressure (ICP), such as ischemic or hemorrhagic stroke, mass, or cerebral edema
  - Renal disorders (glomerulonephritis, polycystic kidney disease, chronic pyelonephritis, hemolytic uremic syndrome)
  - Vascular disorders (coarctation of the aorta, renal artery stenosis, fibromuscular dysplasia, Takayasu arteritis, polyarteritis nodosa)
  - Endocrine disorders (Cushing syndrome [increased cortisol], Conn syndrome [increased aldosterone], pheochromocytoma [increased catecholamines], thyroid disorders, renin secreting tumor)
  - Pregnancy-induced HTN (preeclampsia and eclampsia)
  - Sleep apnea

4. What else might cause HTN, often transient, in the ED?

- Anxiety or 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
- Lead intoxication
- Anti-HTN medication withdrawal
- Certain foods containing large amounts of tyramine

Abstract

Hypertensive emergencies are seen commonly in the ED and must be evaluated and treated promptly to decrease morbidity and mortality. Treatment recommendations differ, depending on the end organ affected. Additionally, the treatment of asymptomatic hypertension in the ED carries its own risks.

Keywords:

hypertensive emergency, hypertensive crisis, aortic dissection, aortic aneurysm, pseudoaneurysm

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 HTN.

5. How do I explain to patients the importance of treating HTN?

Treating HTN has a significant impact on morbidity and mortality, and is 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 achieving a SBP reduction of 12 mm Hg for 10 years in patients with stage I HTN and additional cardiovascular risk factors will prevent one death for every 11 patients treated.

6. Is diagnostic testing necessary in a patient with elevated BP and no symptoms in the ED?

- Testing is generally not necessary, because these patients should receive prompt follow-up care with a primary care physician who can confirm the diagnosis and perform diagnostic studies.
- In select patients for whom follow-up care is poor, or if outpatient treatment is to be started by the emergency physician, a basic metabolic panel and creatinine are recommended (level C), because the results might affect disposition or drug selection.
- In a study of 109 patients with BP greater than 180/110 mm Hg, 6% had laboratory abnormalities.
- Electrocardiograms (ECGs) and chest radiography may show abnormalities related to chronic HTN, but are unlikely to affect care.

7. Should treatment be initiated in the ED in asymptomatic patients with elevated BP?

- Generally, no, because it has been found that a significant number of patients, even with SBP greater than 160 mm Hg in the ED, will not have HTN on follow-up visits.
- If no follow-up visit 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 higher than 180 mm Hg or DBP above 110 mm Hg, consideration should be given to starting an antihypertensive agent. Patients with SBP greater than 200 mm Hg or DBP higher than 120 mm Hg should be started on an antihypertensive agent at discharge. Acute treatment to lower BP in the ED is not necessary.
- Grassi and co-workers showed that 32% of 549 patients in the ED with BP above 180/110 mm Hg had spontaneous decreases of more than 20 mm Hg in SPB and 10 mm Hg in DBP within 30 minutes. They also showed that the outcome at 72 hours was the same for those who received antihypertensive treatment in the ED and those who did not.

8. What is a hypertensive emergency, or crisis?

- A hypertensive emergency is characterized as severely elevated BP (typically >220/120 mm Hg), with acute end-organ (brain, heart, kidneys) damage. The absolute BP is not a criterion. The terms *malignant HTN* and *accelerated HTN* are used in the *International Classification of Diseases (ICD),* tenth edition.
- Examples include:
  - Hypertensive encephalopathy
  - Ischemic and hemorrhagic stroke
  - Subarachnoid hemorrhage (SAH)
  - Acute MI (AMI)
  - Congestive heart failure (CHF)
  - Aortic dissection
  - Acute kidney injury (AKI)
  - Preeclampsia/eclampsia

9. What is hypertensive urgency?

- *Hypertensive urgency* is a term commonly used to describe asymptomatic HTN. The patient has very high BP (>220/120 mm Hg) but no evidence of acute end-organ damage. There may be a history of chronic HTN and chronic end-organ damage.
- The treatment should be to control the BP with oral medications over the next 48 hours, which can be done on an outpatient basis with appropriate follow-up care arranged.

- Of note, there is no *ICD-10* code for hypertensive urgency, and the term seems to more refer to the feeling the provider has about the HTN rather than anything relevant to the treatment of the patient.

10. What are the symptoms of patients with hypertensive emergency when they arrive in the ED?

- The signs and symptoms of hypertensive crisis are manifestations from the organ systems involved.
  - Central nervous system involvement may cause severe headache, lethargy, dizziness, confusion, focal neurologic deficits, paresthesias, or vision changes. If left untreated, this can progress to seizures, blindness, and coma. Headache alone is not a hypertensive emergency.
  - Chest pain, back pain, shortness of breath, and lower extremity swelling may be presenting signs and symptoms of CHF, MI, or aortic dissection.
  - Decreased urine output, nausea, and generalized malaise and weakness may suggest AKI.

11. What signs support the diagnosis of hypertensive crisis?

- Central nervous system: Confusion, altered level of consciousness, and focal neurologic findings
- Funduscopic examination: Arteriovenous nicking, copper-wiring, flame-shaped hemorrhages, exudates, and papilledema
- Cardiopulmonary: Rales, hepatomegaly, and lower extremity edema may be present, as well as a gallop, jugular venous distention, and a displaced point of maximal impulse
- Vascular: A pulsatile mass in the abdomen, or unequal pulses may indicate an aortic aneurysm or dissection respectively, but their absence does not rule out these diseases.

12. What diagnostic studies should be considered in a patient with a hypertensive emergency?

- If neurologic symptoms or physical findings are present, a computed tomography (CT) of the head should be performed to evaluate for hemorrhagic or ischemic stroke, hypertensive encephalopathy, or SAH.
- An ECG should be performed in patients with chest pain or shortness of breath to evaluate for ischemia or infarction.
- Chest radiography should be performed in patients with chest pain or shortness of breath to evaluate for pulmonary edema or evidence of aortic dissection or aneurysm.
- Troponin and a hemoglobin tests 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 aortic dissection or aneurysm, a CT angiogram should be obtained in the ED. These patients should also have a type and screen, as well as any other standard hospital protocol panels needed to prepare for surgical management, including a complete blood count (CBC), metabolic panel, coagulation profile, and ECG.
- A chemistry panel should be considered to screen for renal insufficiency, and a urine sample can be obtained to check for protein, blood, and glucose. The results of the creatinine test should not delay the decision to perform a CT angiogram in patients with a high suspicion for aortic dissection.

13. 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.
- Other causes of AMS should be evaluated, including CVA, intoxication, renal insufficiency, and microangiopathic hemolytic anemia.

14. What is the pathophysiology of hypertensive encephalopathy?

Acute, severe elevations in BP cause cerebral autoregulation to fail. 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.

Cerebral autoregulation works only within a certain range of mean arterial pressure (MAP), above or below which the 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 minus venous pressure. Under normal conditions, cerebral venous pressure is governed by ICP.

$$MAP = [(2 \times DBP) + SBP] \div 3$$

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 raised. For both hypertensive and normotensive patients, the lower limit of autoregulation has been found to be approximately 25% below the resting MAP.

15. How do I treat hypertensive encephalopathy?

- The goal of treatment is to carefully decrease the MAP by approximately 25% over the first hour.
- Medication selection should be based on patient parameters, physician experience, and hospital protocol.
- Each medication works by different mechanisms but should have three important properties in common.
  - Intravenous (IV) route for easy titration
  - Rapid onset
  - Short duration of action
- IV nicardipine, labetalol, clevidipine, or esmolol are the currently recommended medications.

16. What is the treatment threshold for HTN in ischemic stroke?

- 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 only if the SBP is greater than 220 mm Hg or DBP is greater than 120 mm Hg. The BP should be lowered by 15% over the first 24 hours.
- If the patient could have thrombolysis, it is recommended to lower the BP to less than 185/110 mm Hg. The decision making regarding BP management should be done in close consultation with a neurologist or neurosurgeon.
- It is important not to lower the BP too rapidly in these patients, because it may drop the CPP and lead to further ischemia.

17. What are the recommendations regarding treatment of HTN in hemorrhagic stroke?

See Chapter 25.

18. How do I treat HTN if it is associated with SAH?

- There are no definitive data on what BP is beneficial to the patient. The 2012 American Stroke Associate guidelines suggest that achieving an SBP of less than160 mm Hg is reasonable. The medications used are labetalol, esmolol, and nicardipine.
- Nitroprusside and nitroglycerin should be avoided, because they can increase CBF and thus increase ICP.
- Pain should be controlled with narcotic analgesics.

19. How do I treat a patient with severe HTN and evidence of pulmonary edema?

Patients with pulmonary edema and severe HTN should be treated with a focus on afterload reduction, but also with other supportive care.

- Sit the patient upright and provide oxygen and bilevel positive airway pressure (BiPAP) as needed.
- Administer IV nitroglycerin with or without sodium nitroprusside for both preload and afterload reduction.
- Provide angiotensin-converting enzyme (ACE) inhibitors, such as enalaprilat, for afterload reduction.
- Loop diuretics, such as furosemide, may decrease the need for other antihypertensive agents.

20. How do I treat a patient with severe HTN and chest pain caused by ischemia?

- Reduction of BP in the setting of angina or AMI is crucial to decrease the work of the myocardium and prevent ongoing ischemia. First-line treatment is IV nitroglycerin in combination with a β-blocker.
- Morphine at low dosages can be used as an adjunct for pain control.
- If this fails to control BP, nicardipine or fenoldopam can be added.

- Sodium nitroprusside should be avoided, because it can cause a coronary steal phenomenon in patients with coronary artery disease, causing increased mortality in the presence of an AMI.
- Hydralazine should be avoided because it can cause reflex tachycardia, thus increasing oxygen demand.

21. What agents should I use to treat a patient with severe HTN and AKI?

- IV fenoldopam is a short-acting dopamine-1 receptor agonist that increases renal perfusion, creatinine clearance, sodium excretion, and diuresis. It is as effective as nitroprusside at lowering the BP without the risk of cyanide toxicity, but is more costly.
- Other reasonable alternatives include nicardipine and labetalol.
- ACE inhibitors should be avoided if bilateral renal artery stenosis has not yet been ruled out.

22. What should I always think about in a pregnant or postpartum woman with HTN?

Preeclampsia; see Chapter 80.

23. What antihypertensive medications, if stopped abruptly, can cause rebound HTN?

Short-acting sympathetic blockers, such as clonidine, and β-blockers

24. How do I treat catecholamine-induced hypertensive emergency?

- Benzodiazepines are the first-line treatment of hyperadrenergic or catecholamine-induced HTN.
- Antihypertensive agents that can be used for treatment of a catecholamine-induced hypertensive emergency include nicardipine, fenoldopam, phentolamine, and nitroprusside.
- β-Blockers should be avoided, because they can cause unopposed α-adrenergic vasoconstriction and elevate BP further. In patients with cocaine ingestion, β-blockers fail to decrease heart rate and enhance coronary artery vasoconstriction, increase BP, decrease the seizure threshold, and increase mortality.
- Labetalol, an α- and β-blocker, theoretically avoids the problem of unopposed α-adrenergic vasoconstriction, but some sources say it still may cause harm in patients with cocaine ingestion or pheochromocytoma.

25. What are the common parenteral antihypertensive medications and their indications and contraindications?

See Table 33-1.

26. Can I use oral agents to treat hypertensive emergencies?

There is no place for the use of oral agents in a true hypertensive emergency. The therapeutic response is unpredictable and cannot be titrated. Oral agents are appropriate for use in patients with hypertensive urgency (also called *asymptomatic HTN*) if an antihypertensive medication is to be initiated.

KEY POINTS: SPECIAL CONSIDERATIONS WITH HYPERTENSIVE EMERGENCIES

1. Avoid precipitous or excessive drops in BP with cerebrovascular emergencies.
2. Avoid the urge to treat hypertensive urgency in any other way than as if you were treating asymptotic HTN. They are identical and should be treated with oral medication on an outpatient basis.
3. Avoid pure β-blockers for catecholamine-induced hypertensive emergencies. Treat with benzodiazepines first.
4. Understand that pain, anxiety, and just being in the ED might cause transient HTN.

AORTIC DISSECTION

27. How do aneurysms, pseudoaneurysm, and dissection differ?

- An aneurysm involves dilation of all three layers of the arterial wall: the intima, media, and adventitia.

Table 33-1. Parenteral Antihypertensive Medications

DRUG	DOSAGE	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 and AKI
Nitroglycerin	10-200 μg/min IV	2-5 min	3-5 min	AMI, CHF	CVA
Nicardipine	5-15 mg/h IV	15 min	6 hr	AMI, AKI, eclampsia, hypertensive encephalopathy, catecholamine excess	CHF, second- or third-degree AVB
Fenoldopam	0.1-1.6 μg/kg/min IV	5-15 min	1-4 hr	AMI, CHF, AKI, aortic dissection, hypertensive encephalopathy, catecholamine excess	Glaucoma (can cause increased IOP)
Hydralazine	10-20 mg IV bolus; repeat every 2-4 hours prn (max 40 mg)	10-20 min	3-8 hr	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 hr	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

AKI, Acute kidney injury; *AMI*, acute myocardial infarction; *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.

- A pseudoaneurysm, or false aneurysm, is typically caused by arterial injury, resulting in a tear in the arterial wall and consequent blood leak. The hematoma is contained within the outer tissue layer, but still communicates with the arterial lumen.
- Dissection is a distinctly different disease from an aneurysm, and involves a tear in the intima, resulting in a false lumen within the media. Blood can dissect in the wall either proximally or distally.
- The term *dissecting aneurysm* is not an accurate use of the terms. Although it is possible for an aneurysm to dissect, dissection typically exists without an aneurysm present.

28. Other than cardiac ischemia and aortic dissection, what causes chest pain in the hypertensive patient?

- Other life-threatening causes of chest pain include pulmonary embolism, tension pneumothorax, pericardial tamponade, and esophageal rupture.
- In the setting of severe HTN, aortic dissection should be strongly considered. Dissection can be rapidly fatal.

29. What are risk factors associated with aortic aneurysms?

- 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), blunt chest trauma (usually resulting in pseudoaneurysms), patients with connective tissue diseases (such as Marfan syndrome and Ehlers-Danlos syndrome), and arteritis.
- Although true aneurysms can develop anywhere along the aorta, 75% are abdominal aortic aneurysms (AAAs).

30. What are the risk factors for aortic dissection?

- HTN is present in 70% of patients (most common)
- Men are at higher risk than women
- Age older than 60 years
  - The peak age for proximal dissection is 50 to 55 years, and for distal dissection is 60 to 70 years.
- Bicuspid aortic valves
- History of aortic valve replacement or cardiac catheterization
- Cocaine or amphetamine use
- Genetic conditions (Marfan, Ehlers-Danlos, Loeys-Dietz, Noonan, and Turner syndromes)
- A family or personal history of dissection
- Known thoracic aortic aneurysm
- Pregnancy
- Weight lifting

31. What symptoms may be present in a patient with thoracic aortic dissection?

- The patient experiences a sudden onset (85%) of severe chest pain with radiation to the jaw, neck, or back in the intrascapular region
- Pain is most severe at onset and is often described as sharp, ripping, or tearing in quality.
- Pain starts in one region and moves to another (abdomen to chest or chest to abdomen).
- The patient has nausea, vomiting, diaphoresis, lightheadedness, and apprehension or a sense of impending doom.
- Syncope can also be the presenting complaint, and in some cases may be the only symptom.
- Proximal dissections may cause aortic regurgitation and pericardial effusion with tamponade (16%).
- Occlusion of aortic branches may cause AMI (coronary artery involvement); stroke (carotid or vertebral artery involvement); or paresthesias and arm pain (subclavian artery involvement), which may be suggested by unequal BPs or pulses.
- Spinal artery occlusion can cause neurologic compromise.
- Hoarseness may result from recurrent laryngeal nerve compression.
- Chest pain unrelieved by large doses of narcotic analgesics should raise the concern for this diagnosis.

32. What physical examination findings may be present in a patient with thoracic aortic dissection?

- The patient often has high BP on arrival, but hypotension may be present if the patient develops tamponade, aortic regurgitation, MI, or rupture.
- A new diastolic murmur of aortic regurgitation suggests dissection into the aortic root.
- Unequal upper extremity BPs occur less than one third of the time, but, if present, are highly suggestive of proximal aortic dissection.
- Pulses should be checked in all four extremities, because dissection can extend the entire length of the aorta and into the iliac arteries.

33. What diagnostic imaging should be performed when thoracic aortic dissection is suspected?

- Chest radiographs are abnormal in about 80% of cases, but the abnormalities are nonspecific. The study may rule out other causes of chest pain, such as pneumothorax, but further imaging is usually required if dissection is suspected.
- CT angiogram is the study of choice in the ED, because it is quick, accurate, and readily available in most practice settings.
- MRI is sensitive and specific, but scan times are long and place the patient in a position inadequate for resuscitation if needed.
- Transesophageal echocardiogram (TEE) is excellent for determining involvement of the aortic valve and coronary arteries and can detect the presence of pericardial effusion or tamponade, but the study requires sedation and an experienced cardiologist or technician.
- Conventional contrast angiography, once the gold standard, is rarely used 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 MI.

34. What might I see on the chest radiograph of a patient with a thoracic aortic dissection?

- Widened mediastinum
- Loss of the aortic knob
- Left pleural effusion
- Tracheal (or nasogastric tube) deviation to the right
- Apical pleural capping
- Calcium sign (displacement of the intimal calcium layer > 10 mm in the aorta)

  Of note, chest radiographs are normal in up to 20% of patients with dissection.

35. What other tests should I perform?

- The patient should be prepared for possible surgical intervention with laboratory testing, including a CBC; metabolic panel; blood, urea, nitrogen (BUN)/creatinine test; blood type and crossmatch; and prothrombin time (PT)/partial thromboplastin time (PTT).
- D-dimer is elevated in 97% of aortic dissections. A D-dimer level less than 500 ng/mL has a negative predictive value of 95% for aortic dissection. It has been suggested that this could be used to rule out dissection in patients with a low pretest probability, but it has not been prospectively studied.

36. What is the Stanford classification for aortic dissection?

- The Stanford classification describes the location of the dissection related to the recommended treatment modalities.
  - Type A dissections (67%) involve the ascending aorta proximal to the ligamentum arteriosum, and require emergent surgical repair. If the patient survives surgery, the in-hospital mortality is 30%.
  - Type B dissections (33%) affect the descending aorta distal to the ligamentum arteriosum and are usually managed medically, but about one third will eventually require surgical or endovascular repair. Overall, in-hospital mortality is 10%, but it is only 8% if dissections are managed medically and 19% if vascular intervention is required.

37. How do I treat a patient with aortic dissection?

- Treatment should be initiated before imaging in patients with a high suspicion of aortic dissection.
- Opiate analgesics are appropriate to provide adequate pain control.

- IV antihypertensive medication should be initiated if the patient is hypertensive.
  - Rate control should start first with an IV β-blocker, such as esmolol, before the initiation of an antihypertensive to prevent reflex tachycardia and increased shear forces.
  - This should be followed by an antihypertensive agent such as nicardipine, fenoldopam, or nitroprusside.
  - An alternative treatment regimen is IV labetalol used as a single agent.
  - Rapid reduction of SBP to a range of 100 to 110 mm Hg and a heart rate or 60 bpm is indicated.
- A cardiothoracic surgery specialist should be consulted emergently.
- If the patient is hypotensive, a bedside ultrasound should be performed to evaluate for pericardial tamponade.
  - Pericardiocentesis can be a life-saving, temporizing intervention until the patient reaches the operating room.

ABDOMINAL AORTIC ANEURYSM

38. What are common presenting signs and symptoms of an AAA?

- Most patients with AAAs are asymptomatic, and their aneurysm is found incidentally on physical examination or on diagnostic studies performed for other reasons.
- Patients with symptoms or acute expansion of an AAA may have gradually increasing abdominal pain, or low back pain or flank pain radiating to the groin, that usually is unaffected by movement. It is often described as dull, throbbing, or colicky.
- Two percent to 3% of men older than 50 years have an occult AAA.
- Approximately 75% of aneurysms greater than 5 cm can be palpated.
- Only 5% to 10% of patients with an AAA have an abdominal bruit.

39. What common diseases may mimic ruptured AAA?

Many disease processes can present in similar ways to AAA, including renal colic, pancreatitis, perforated peptic ulcer, AMI, gallbladder pathology, diverticulitis, appendicitis, perforated viscus, bowel obstruction, musculoskeletal back pain, and intestinal ischemia. The diagnosis should be considered in patients older than 50 years with any one of the symptoms in the classic triad: pain, hypotension, and pulsatile mass.

40. What are the risks of rupture in AAA?

- The risk of rupture is minimal for an AAA measuring less than 4 cm.
- The risk increases dramatically at diameters larger than 6 cm. The annual rupture risk for AAAs from 6 to 7 cm is 10% to 20%, from 7 to 8 cm 20% to 40%, and, if greater than 8 cm, the annual rupture risk is increased to 30% to 50%.
- Rapid expansion is the greatest predictor of impending rupture, and routine screening of patients with known AAAs is important, because it significantly affects mortality. All patients with an AAA 5 cm or greater in diameter should have a follow-up consultation with a vascular surgeon.
- Expansion of more than 0.7 cm over 6 months or more than 1 cm per 1 year are risk factors for rupture, regardless of aneurysm size.

41. What is the presentation of a ruptured AAA?

- The classic 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.
- Hypotension, syncope, or low hematocrit may signify significant blood loss.
- Rarely AAAs can rupture into the intestines and present as a massive gastrointestinal (GI) bleed. GI 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 have a massive effect on surrounding structures, resulting in a bowel or ureteral obstruction.
- Radicular pain may occur if the bleeding is retroperitoneal. Leg ischemia may occur because of peripheral embolization of mural plaques.

42. How do I treat a patient with a suspected ruptured AAA?

- Place two large-bore IVs; test blood type and cross-match for at least six units of packed red blood cells.

- Call a vascular surgeon to get the patient to the operating room as soon as possible. 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 appropriate in hemodynamically stable patients, and 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 for 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.

43. What are the dilemmas of aggressive fluid resuscitation in a hypertensive patient with ruptured AAA?

There are no prospective studies to guide optimal fluid resuscitation in ruptured AAA. The goal is to achieve intravascular volume replacement adequate to maintain end-organ perfusion without generating excessive BP. 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 BP and a dilutional coagulopathy, further increasing bleeding. The amount and type of resuscitation fluids must take into consideration the patient's comorbid conditions. Warm saline and blood products should be used to maintain a MAP of 60 to 65 mm Hg.

44. When should a symptomatic unruptured AAA be repaired?

- The mortality rate associated with emergent repair of a symptomatic AAA is about 25%, as opposed to a 5% mortality rate associated with semielective repair.
- Aneurysms 5.5 cm or larger should be repaired.
- There is debate over when to operate on patients with aneurysms between 4 and 5.4 cm. A threshold of 5 cm for women has been suggested, because the rupture rate is higher in women.
- Expansion of more than 0.5 cm over 6 months or more than 1 cm per 1 year is an indication for repair.

45. How are AAAs surgically repaired?

AAAs can be repaired with open surgery or endovascular stenting. Open surgery involves laparotomy and cross-clamping the aorta. The acute risks are higher and the hospital stay is longer, but it corrects the problem. Stenting involves the insertion of a graft though a small incision in the groin and positioning of the stent with a balloon. The acute risks are lower, and the recover period is shortened.

46. What are the complications of endovascular aortic repair (EVAR)?

The short-term outcomes seem equal or favorable to open repair. However, the long-term mortality rates 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 incidence with the evolution of the technique and advance in materials used. Complications include:

- Graft infection, which can lead to aortoenteric fistula formation, commonly presenting as upper GI bleed
- Limb ischemia caused by arterial occlusion
- Stent migration
- Continued aneurysmal sac expansion (endotension)
- Endoleak is the most common 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 the surgery department.
3. Bedside ultrasound is an excellent screening tool for AAA.
4. CT is the gold standard for making the diagnosis of a ruptured AAA.

Acknowledgement

The editors and author of this chapter would like to acknowledge and thank Dr. Edward P. Havranck for his previous contributions to this chapter.

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QUESTIONS

1. A 65-year-old man comes to the ED with sudden onset of left flank pain and syncope. His wife says that has had intermittent flank pain for the past few months. His BP is 84/30, heart rate 150 bpm, temperature 98.5°F, and respiratory rate 20. A beside ultrasound shows an AAA measuring 7 cm. Which of the following is the most appropriate action?
   a. Begin vasopressors.
   b. Call the vascular surgeon for emergency repair.
   c. Perform a CT angiogram of the aorta.
   d. Perform a conventional angiogram.
   The correct answer is *b.* This patient is unstable with a ruptured AAA, and a bedside ultrasound demonstrating an AAA. It is unsafe to send this patient to the CT scanner or to a radiologist for an additional study. He requires emergency surgery. Some centers may have the option of performing endovascular stenting as well.
2. A 60-year-old woman is sent to the ED from an outpatient surgery center where she was scheduled to have her cataract removed. She has a history of HTN and normally takes labetalol, atorvastatin (Lipitor), and clonidine. The surgery center was concerned about her BP. She has no complaint other than hunger, as she has been fasting for surgery. She is afebrile, BP is 225/128, pulse 89, respirations 20, and oxygen saturation is 98% on room air. What is the most appropriate management?
   a. Begin IV nitroprusside.
   b. Check a metabolic panel and BUN/creatinine levels.
   c. Give her usual medications orally.
   d. Perform an ECG to evaluate for cardiac ischemia.
   The correct answer is *c.* This patient has rebound HTN from discontinuing her usual medication before surgery. This phenomenon is commonly seen with clonidine and β-blockers. Although her BP is high, she has no symptoms and has recently had a preoperative evaluation, so no blood work is indicated. She should resume her medication and be informed that she can take the medication before her next scheduled surgery appointment.
3. A 45-year-old male has had tearing chest pain radiating to the back for 90 minutes. His initial BP is 190/115, heart rate 110, temperature 97.4°F, and saturation level 96% on room air. A CT reveals a proximal, type A aortic dissection, and after being started on labetalol, he has good control of his symptoms. While the vascular surgeon is en route, the nurse calls you because the patient has lost consciousness and has a BP of 60/28 with a heart rate of 70. All of the following should be done immediately, except:
   a. Discontinue labetalol.
   b. Start vasopressors.
   c. Bedside ultrasound.
   d. Administer IV fluids.
   The correct answer is *b.* There are several possible causes for this patient to decompensate, and they include:
   - Tamponade from dissection into the pericardium: If this is the case, a pericardiocentesis may buy the patient some time before the surgeon arrives. IV fluids will also help with this.
   - Aortic regurgitation: IV hydration and discontinuing the labetalol may help with forward flow.
   - Overmedication: Discontinuing the labetalol alone may help if the bedside ultrasound does not reveal a pericardial effusion.
   - Dissection through the adventitial layer with free rupture: If this is the cause, the patient is dead. There is no roll for vasopressors in the setting of aortic dissection.

CHAPTER 34

PERICARDITIS AND MYOCARDITIS

Christopher B. Colwell, MD

PERICARDITIS

1. Describe a normal pericardium.

The pericardium is 1 to 2 mm thick, is relatively inelastic, 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 (Table 34-1). This postviral pericarditis, termed *idiopathic* because a viral source has 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. The discomfort 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. What are the electrocardiograph (ECG) findings in pericarditis?

The ECG typically evolves through the following four stages:

1. 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 V1, in which reciprocal changes occur. The ST-segment displacement is attributed to the associated subepicardial myocarditis, whereas the PR segment depression is attributed to subepicardial atrial inflammation.
2. In stage 2, the ST and PR segments normalize, and the T waves flatten.
3. In stage 3, deep T-wave inversion occurs.
4. 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.

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

Abstract

This chapter presents the common findings and management of pericarditis and myocarditis.

Keywords:

pericarditis, pericardial effusion, cardiac tamponade, myocarditis, dilated cardiomyopathy, pericardial friction rub, pulsus paradoxus

Table 34-1. 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

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. Besides pericardial effusion, can acute pericarditis cause an MI?
No, acute MI is not a known complication of acute pericarditis.

11. 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 2000 mL or more without hemodynamic compromise.

12. 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.

13. 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.

14. 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 the following:

- Tachycardia
- Hypotension
- Cyanosis
- Dyspnea
- Jugular venous distention
- Pulsus paradoxus
- 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.

15. What is pulsus paradoxus?

Pulsus paradoxus is an abnormally large (>10 mm Hg) drop in the systolic blood pressure with inspiration. Kussmaul termed this phenomenon *paradoxic* because of the disappearance of the pulse during inspiration when the heart was obviously beating. Pulsus paradoxus is a pulse—not 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.

16. What is the appropriate ED management of pericarditis?

Antiinflammatory agents, such as ibuprophen 600 mg four times a day for 1 week or indomethacin (Indocin) 25 mg three times a day for 1 week, should be administered. The use of corticosteroids is controversial. Although corticosteroids are effective antiinflammatory 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 pericardial pressure. Intravenous fluids should be infused rapidly to increase arterial pressure and cardiac output.

17. 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 antiinflammatory 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.

18. Do pediatric patients get pericarditis?

Yes, in fact, pericarditis accounts for about 5% of all children who come to the pediatric ED with chest pain. Children with pericarditis usually have sharp, stabbing, retrosternal chest pain; fever; and shortness of breath. Like in adults, the chest pain is typically worse with inspiration and relieved by sitting up and leaning forward. Referral to a cardiologist for echocardiography is recommended. The reported recurrence rate is as high as 36%.

MYOCARDITIS

19. What is myocarditis?

An inflammation of the myocardium in the absence of ischemia

20. 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:

1. Direct invasion of the myocardium
2. Production of a myocardial toxin (e.g., diphtheria)
3. 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*
- *Corynebacterium diphtheriae*

21. When should a diagnosis of myocarditis be considered in the ED?

Diagnosing myocarditis in the ED can be a challenge. 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 have 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.

22. 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 dysrhythmias and left ventricular aneurysms.

23. 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 the result of 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 an acute MI pattern also may occur. Atrial dysrhythmias have been described.

24. 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 commonly are elevated in patients with myocarditis. The 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.

25. 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.

26. 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 the result of an abnormal autoimmune reaction, opportunistic infections, or HIV itself.

27. 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.

28. 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 γ-globulin has been studied and may be associated with improved left ventricular function and better survival during the first year after initial presentation.

29. 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.

30. Does myocarditis present differently in children?

Pediatric myocarditis rarely presents with specific cardiac symptoms and should be considered in children with nonspecific clinical presentations, particularly those with symptoms and signs of hypoperfusion, especially syncope or seizure.

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 patients with AIDS, with rates at autopsy as high as 52%.

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QUESTIONS

1. All of the following are ECG findings that are more consistent with acute pericarditis than an acute MI except:
   a. PR-segment depression
   b. Simultaneous ST elevations and T-wave inversions
   c. Diffuse ST elevations not isolated to a particular anatomic distribution
   d. ST elevations that are upwardly concave rather than convex

   The correct answer is *b.* All of the others are more consistent with acute pericarditis than acute MI. In acute pericarditis, the T-wave inversions tend to occur after the ST-segment elevations have returned to normal.
2. All of the following are true about acute pericarditis except:
   a. Appropriate treatment includes antiinflammatory agents and intravenous fluids
   b. Pericardial effusions and cardiac tamponade are unusual but potential complications of acute pericarditis.
   c. Patients with acute pericarditis are at high risk for developing acute MIs as a complication of the acute pericarditis.
   d. Pericarditis can be distinguished from musculoskeletal chest pain, because it can be relieved by sitting up, can have a characteristic friction rub, and is associated with ECG abnormalities.

   The correct answer is *c.* Acute MI is not a known complication of acute pericarditis.
3. Which of the following is true regarding myocarditis?
   a. Bacteria are the most common cause of cases in the United States.
   b. The diagnosis of myocarditis can be made based on ECG and chest radiography findings.
   c. Myocarditis is easily distinguished from acute MI in the ED.
   d. Tachycardia that is out of proportion to temperature or apparent toxicity may be the only clue the myocarditis exists.

   The correct answer is *d.* Viruses are the most common cause of myocarditis in the United States. The diagnosis of myocarditis can be difficult to make in the ED and can be difficult to distinguish from MI. It is not based on any particular ECG or chest radiography findings. Tachycardia out of proportion to other findings of the examination may be the only clue that myocarditis exists.

ESOPHAGUS AND STOMACH DISORDERS

Rakesh Talati, MD, MBA, and Gillian McCafferty, MD

1. How are gastrointestinal (GI) problems differentiated from acute myocardial infarction?

Esophageal or gastric pain can present with visceral 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 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 unsuspecting physician.

2. What is a GI cocktail?

The two most commonly used GI cocktails contain antacids (30 mL), viscous lidocaine (10 mL), and either a preparation of atropine, hyoscyamine, phenobarbital, and scopolamine (Donnatal; 10 mL) or dicyclomine (Bentyl; 20 mg). These cocktails may provide temporary symptomatic relief of minor esophageal and gastric irritation.

Note: It has been concluded that the addition of atropine, hyoscyamine, phenobarbital, and scopolamine (Donnatal), and/or lidocaine did not provide more relief than an antacid alone.

3. What is heartburn?

Heartburn is a 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 caused by heightened mucosal sensitivity to acid.

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 (PPIs; 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. It can be traced 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, and pill-induced esophagitis, as well as esophagitis related certain systemic diseases (e.g., Behçet syndrome, Crohn disease, pemphigus vulgaris, Stevens-Johnson syndrome). Odynophagia is unusual in reflux esophagitis but may occur with a peptic ulcer of the esophagus (Barrett 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

Abstract

This chapter covers esophageal and stomach disorders ranging from esophageal foreign body and perforation to peptic ulcer disease (PUD) and gastrointestinal (GI) Bleeding.

Keywords:

esophagus, stomach, gastrointestinal (GI) bleeding, peptic ulcer disease (PUD), heartburn, Helicobacter pylori*, peptic ulcer, perforated peptic ulcer*

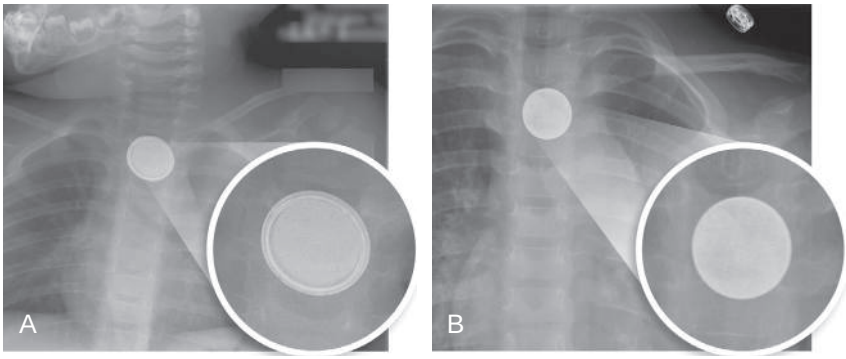

Figure 35-1. Anterior-posterior radiographs. A, Double ring, or halo sign, of a button battery in the esophagus of a child. B, Homogenous appearance of a coin in the esophagus of a child. *(From Jatana KR: Button battery injuries in children: a growing risk.* Everything Matters in Patient Care *26:9–10. Columbus, OH: Nationwide Children's Hospital, 2013.)*

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. The most dangerous esophageal foreign body is a disc (button) battery (Fig. 35-1), which can cause a chemically induced perforation in as few as 4 hours.

7. How is esophageal obstruction treated?

Foreign bodies, especially those that are sharp, or impacted food are best removed by endoscopy. Meat tenderizer should not be used to facilitate passage of obstructed meat. Glucagon 0.5 to 2 mg intravenously (IV), has historically been given to allow foreign bodies to pass by attempting to relax smooth muscle of the lower esophagus. However, no studies have shown that it decreases the need for endoscopy for ED patients. In addition, it often causes vomiting, which may increase risk of aspiration or esophageal perforation.

8. 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 experience upper GI bleeding. The tear usually heals within days without further complications. In rare circumstances, surgery is needed.

9. 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 syndrome), or diseases of the esophagus (e.g., corrosive esophagitis, ulceration, neoplasm). It often presents with mild, nonspecific symptoms. More than half of all patients are initially misdiagnosed. Symptoms can quickly progress to chest pain that becomes severe and may be worsened by swallowing or breathing. Chest radiograph may reveal pleural effusion or air within the mediastinum, pericardium, pleural space (pneumothorax), or subcutaneous tissue, or it may appear normal. Esophageal perforation may lead to leakage of gastric contents into the mediastinum and secondary infection (i.e., mediastinitis), rapidly progressing to sepsis. Esophageal perforation is confirmed radiographically by having the patient swallow diatrizoic acid (Gastrographin) and observing for leakage of radiopaque contrast material. Treatment includes broad-spectrum antibiotics, gastric suction, and surgical repair and drainage as soon as possible.

10. 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 caused by 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 (Fig. 35-2). Perforated PUD and gastric volvulus are the two most serious conditions requiring immediate diagnosis and treatment.

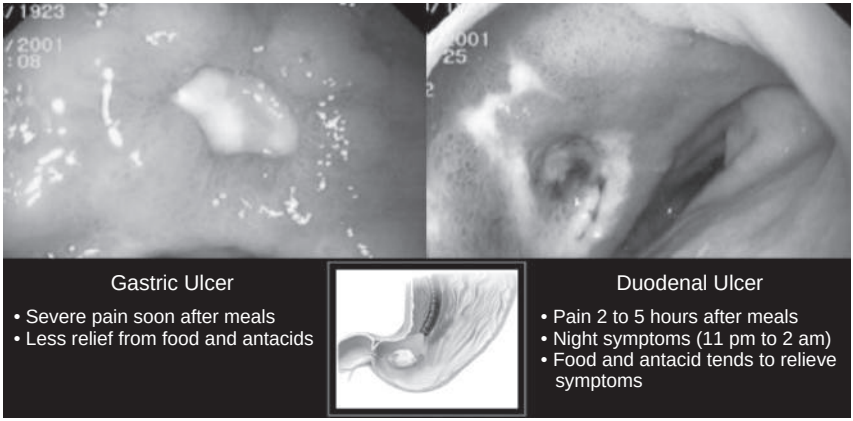

Figure 35-2. Clinical comparison of gastric ulcer and duodenal ulcer. *(Reproduced with permission from RoshReview.)*

11. What are the common causes of gastritis and PUD?

Gastritis is associated with alcohol, salicylates, nonsteroidal antiinflammatory drugs (NSAIDs), and hiatal hernia. Almost all non–NSAID-related ulcers are caused by *Helicobacter pylori*. It is the only bacterium to be classified as a class I carcinogen by the World Health Organization (WHO), because it is a precursor to gastric carcinoma. First-line treatment for patients with *H. pylori* is the combination of a PPI, clarithromycin, and amoxicillin. NSAIDs are the second most common cause of PUD. They suppress protective prostaglandins in the stomach. Up to 25% of chronic NSAID users develop ulcer disease.

12. How does perforated PUD present?

Sudden onset of abdominal pain that is not often related to eating is a common presentation for both gastric volvulus and perforated PUD. Pain typically radiates to the back, but also may radiate to the chest or upper abdomen. The pain is usually steady and refractory to antacids. Free air may cause referred pain to either or both shoulders. Vomiting is present in approximately 50% of cases. On physical examination, patients appear to be 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. Free air is present on upper right chest radiograph or the abdominal left lateral decubitus view in more than 70% of patients. Prompt surgical consultation must be obtained. Broad-spectrum antibiotics should be given, and the patient should be prepared for emergent laparotomy.

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. Bloody or coffee-ground vomit, known as *hematemesis*, or dark, tarry stools, known as *melena,* most often represents upper GI bleeding. Lower GI bleeding most often produces bright red or maroon blood, known as *hematochezia.* Although generally reliable, false-negative and false-positive results may occur when using Hemoccult and Gastroccult cards.

14. Do all patients with only lower GI bleeding require nasogastric (NG) tube placement?

The routine use of NG aspiration was once advocated to rule out occult upper GI bleeding. However, NG aspiration, with or without lavage, has a low sensitivity and poor negative likelihood ratio, limiting its role in ruling out an upper GI source of bleeding in patients with melena or hematochezia without hematemesis. NG tube placement is a painful procedure with potential complications, including aspiration and perforation.

15. How is a patient classified as low risk for having occult upper GI bleeding?

The Glasgow-Blatchford score (GBS) is a risk stratification tool applied to patients with acute nonvariceal upper GI bleeding. The GBS is calculated by totaling points assigned for laboratory and

Table 35-1. Glasgow-Blatchford Score

ADMISSION RISK MARKER	SCORE
Blood Urea Nitrogen (mg/dL)	
6.5-7.9	2
8-9.9	3
10-25.0	4
≥25	6
Hemoglobin (g/dL) for Men	
12-13	1
10-11.9	3
<10	6
Hemoglobin (g/dL) for Women	
10-11.9	1
<10	6
Systolic Blood Pressure (mm Hg)	
100-109	1
90-99	2
<90	3
Other Markers	
Pulse ≥ 100 (beats/min)	1
Presentation with melena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

clinical risk markers (Table 35-1). A score higher than 0 is 99.6% sensitive in predicting high-risk patients who will require clinical interventions, including blood transfusion, endoscopy, or surgery. Low-risk patients with a score of 0 are candidates for outpatient management.

16. What are 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.

Begin by rapidly assessing and managing the patient's airway, breathing, and cardiovascular status. Patients should be undressed, connected to cardiac and arterial oxygen saturation (SaO_2) monitors, and given supplemental oxygen if SaO_2 is less than 93%. A large-bore, peripheral intravenous catheter with infusion of normal saline should be started. 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 are given rapid infusion of crystalloid. Blood should be drawn for type and cross-matching; hemoglobin and hematocrit tests; platelet count; prothrombin time; and obtaining electrolyte, BUN, and creatinine levels. Stool should be tested for occult or gross blood. 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 pneumoperitoneum or pulmonary aspiration.

18. What medications improve GI bleeding outcomes?

Infusion of high-dose PPIs before endoscopy accelerated the resolution of signs of bleeding in ulcers and reduced the need for endoscopic therapy. Patients with upper GI bleeding from suspected

esophageal varices (severe liver disease, previous variceal bleed, history of alcoholism, or highly abnormal liver function tests) should receive an infusion of octreotide (50-μg IV bolus, followed by 50-μg/h infusion). Antibiotics should also be considered in these patients as well.

19. How should a patient with continued GI bleeding 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 emergently. 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 with endoscopy, but emergency operative repair may be required in patients with persistent GI bleeding.

20. Is placement of a NG 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.

21. Should most patients with upper GI bleeding undergo endoscopy?

Yes, because 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.

22. What is the disposition for patients with GI bleeding?

GI bleeding usually stops spontaneously, and no further ED management is necessary. Low-risk patients with upper GI bleeding can often be discharged home with clear instructions, including signs and symptoms of worsening GI bleeding, and a plan for urgent outpatient follow-up care. Lower GI bleeding with definitive source from hemorrhoids, fissure, or proctitis can also be managed on an outpatient basis. All other patients with upper and lower GI bleeding are admitted for further evaluation and intervention.

23. What are the criteria that allow a patient with low-risk upper 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 levels
- Proper understanding of signs and symptoms of significant bleeding
- Good home support
- Follow-up consultation arranged within 24 hours
- Immediate access to emergent care, if needed

KEY POINTS: ESOPHAGUS AND STOMACH DISORDERS

1. Epigastric pain may be caused by 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 the most common cause of PUD, and the only bacteria classified as a class I carcinogen by the WHO.
4. Esophageal perforation can present with vague nonspecific symptoms and rapidly progress to sepsis and death.
5. Patients with upper GI bleeding who are hemodynamically unstable should receive rapid intravenous crystalloid infusion, urgent surgery, and gastroenterology consultation.

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QUESTIONS

1. The most common cause of PUD is
   a. Alcohol
   b. NSAIDs
   c. *H. pylori*
   d. Salicylates
   The correct answer is *c*.
2. Which of the following esophageal foreign bodies is most dangerous?
   a. Meat bolus
   b. Button battery
   c. Chicken bone
   d. Coin
   The correct answer is *b*.
3. Which of the following signs and symptoms most likely distinguishes GI problems from acute myocardial infarction?
   a. Resolution of symptoms from nitroglycerin
   b. Resolution of symptoms from GI cocktail
   c. Description of the pain
   d. Hematemesis
   The correct answer is *d*.

BOWEL DISORDERS

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CHAPTER 36

1. When do I consider evaluating a patient for appendicitis?

Consider appendicitis in anyone who has abdominal pain. It can occur at any age, but is most prevalent in the teens and 20s. With the high incidence of appendicitis in the population, atypical presentations are common. Appendicitis is one of the most commonly missed diagnoses in emergency medicine, and it is the most common nonobstetric 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 rebound on physical examination. Involuntary guarding and fever may occur later.

3. How does appendicitis present clinically?

The classic presentation of appendicitis 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 abnormally 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 cholelithiasis or cholecystitis. Other diagnoses of RLQ pain should also be considered (Table 36-1). Prediction rules for pediatric appendicitis such as the Pediatric Appendicitis score and the Alvarado Score are available but have not been proven to be superior over clinical judgment.

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 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. Commonly, 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: More than 10,000/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

6. What radiologic study is best at imaging the appendix?

Abdominal and pelvic CT is the imaging modality of choice for appendicitis. The scan is routinely done with intravenous contrast alone, but oral or rectal contrast enhancement can be added. 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.

Abstract

Abdominal pain secondary to bowel disorders is a common cause of ED visits. This chapter discusses the ED approach and management of these disorders.

Keywords:

appendicitis, diverticulitis, inflammatory bowel disease (IBD), anal fissure, bowel obstruction

Table 36-1. 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 tuboovarian pathologic abnormality (e.g., tuboovarian abscess, ovarian torsion, ovarian cysts)
Incarcerated inguinal hernia	Ectopic pregnancy
Testicular torsion or epididymitis	Mittelschmerz
Mesenteric adenitis	Pyelonephritis, symptomatic nephrolithiasis

Consider ultrasound imaging in children, pregnant patients, and thin patients. The sensitivity is 88% to 94%, but the sensitivity is variable and 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. An appendiceal diameter greater than 6 mm is the most accurate finding. In some institutions ultrasound is initial diagnostic study in pediatric patients. Obtain abdominal and pelvic CT imaging if the ultrasound is indeterminate, or if it is normal despite a clinical setting that is still concerning for appendicitis. Magnetic resonance imaging (MRI) may also be used. A fluid-filled appendix greater than 7 mm is considered abnormal. MRI is difficult to obtain after hours, requires an extended period of examination, and is contradicted in some patients with renal disease or specific medical devices.

7. What is the treatment for appendicitis?

Appendectomy is the definitive treatment. Once appendicitis has been diagnosed, or is highly suspected, a surgical consultation 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 (Table 36-2).

9. How do patients with mesenteric ischemia experience symptoms?

Patients complain of a diffusely painful abdomen. In the early stage, patients complain of severe pain but have minimal physical findings; i.e., 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 is difficult. The combination of clinical suspicion, radiographic imaging, and laboratory findings can help make the diagnosis. Direct surgical visualization of the bowel is 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 have poor 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

Table 36-2. Risk Factors for Mesenteric Ischemia

Age older 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

Table 36-3. Common Features for Inflammatory Bowel Disease

CLINICAL FEATURE	CROHN 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

Modified from Podolsky DK: Inflammatory bowel disease. *N Engl J Med* 347:417–429, 2002.

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 64), but it can occur in adults. Typical pathologic lesions include tumors, Meckel diverticulum, and inflammatory lesions. The high incidence of mass lesions in adults mandates surgical exploration.

13. What is inflammatory bowel disease (IBD)?

IBD is an idiopathic, chronic inflammatory disease of the intestine. IBD includes two main groups:

1. Crohn disease (CD), also known as *regional enteritis* or *granulomatous ileocolitis*
2. Ulcerative colitis (UC)

CD and UC are rising in incidence. Common clinical features are summarized in Table 36-3.

Table 36-4. 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

14. How do CD and UC present?

Although they are pathologically distinct diseases, CD and UC can appear similar and affect all age groups (see Table 36-3). 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 observation. 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 a patient to toxic megacolon. Metronidazole may help treat the chronic perirectal complications of CD. Patients should be admitted if they have severe pain, heavy bleeding, signs of hemorrhagic shock, peritonitis, or any life-threatening complications. Extraintestinal manifestations of IBD can also occur (Table 36-4).

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 adynamic. Mechanical obstruction from adhesions or tumors commonly 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
- Bezoars

18. What are the clinical features of SBO?

Patients have diffuse abdominal pain, distention, and occasionally, vomiting. Early on, the pain is mild, crampy, and colicky. 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 electrolyte replacement, decompression with a nasogastric tube, and intravenous fluid resuscitation. 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 have symptoms of 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. Limit administration of medications, such as opioids, that slow intestinal motility. If the ileus is prolonged (>3 to 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 disease or an imperforate anus. Hernias and adhesions are uncommon causes of LBO.

24. What are diverticula and what are common complications?

Diverticula are saclike 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 incidence 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 buildup 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 commonly in the descending and sigmoid regions of the colon but can occur throughout the colon. The abdominal CT scan with intravenous contrast is the diagnostic procedure of choice and can show evidence for abscesses, bowel perforation, and severity of disease.

26. How do I 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 obtain follow-up care, can be managed as outpatients with oral antibiotics and close observation after discharge. 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 and catheter drainage.

27. What are common causes of lower GI bleeding?

Patients often arrive in 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 I 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. They are commonly enlarged and tender. 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 the following:

- Irrigation during the shower or bath
- Stool softeners
- High-fiber diet
- Bulk laxatives (e.g., psyllium or methylcellulose)
- Increased fluid consumption
- Proper anal hygiene
- 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 occur often; 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 common cause of SBO.
4. Patients with SBO should be aggressively resuscitated with intravenous fluids in the ED because of the extensive depletion of intravascular fluid.
5. Although diverticulitis is most commonly detected in the older patient population, younger patients (age 20 to 40 years) also develop it.
6. IBD can cause complicated rectal abscesses or fissures that require surgical consultation.

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QUESTIONS

1. What is the best test to diagnose appendicitis in the ED?
   a. Urinalysis
   b. MRI
   c. CT of the abdomen and pelvis
   d. Radiograph of the abdomen
   The correct answer is *c.*
2. The most common cause of SBO is:
   a. Surgical adhesions
   b. Foreign body
   c. Tumor
   d. Lymphadenopathy
   The correct answer is *a.*
3. Diverticulitis should be treated with:
   a. Immediate surgery
   b. Colonoscopy within 24 hours
   c. Antibiotics
   d. Low-fiber diet
   The correct answer is *c.*

CHAPTER 37

LIVER AND BILIARY TRACT DISEASE

Molly E.W. Thiessen, MD

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, is not colicky, occurs after a fatty meal, and is thought to be the result of 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 resulting from 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 sign, and can be present with or without fever and chills or leukocytosis.
- Choledocholithiasis occurs when a gallstone lodges in the common bile duct (CBD), and can cause cholecystitis, 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 in the CBD) in the presence of a bacterial infection. It presents as right upper quadrant pain, fever and chills, and jaundice (Charcot triad), although only 25% of patients have all three. It may include shock and mental status changes (Reynold 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 common 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 the Murphy 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 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 caused by 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.

Abstract

This chapter describes hepatic and biliary pathology encountered in the ED, as well as recent diagnostic and treatment possibilities.

Keywords:

liver disease, biliary, gallbladder, pancreas, hepatitis, right upper quadrant pain, hepatitis C, liver function tests

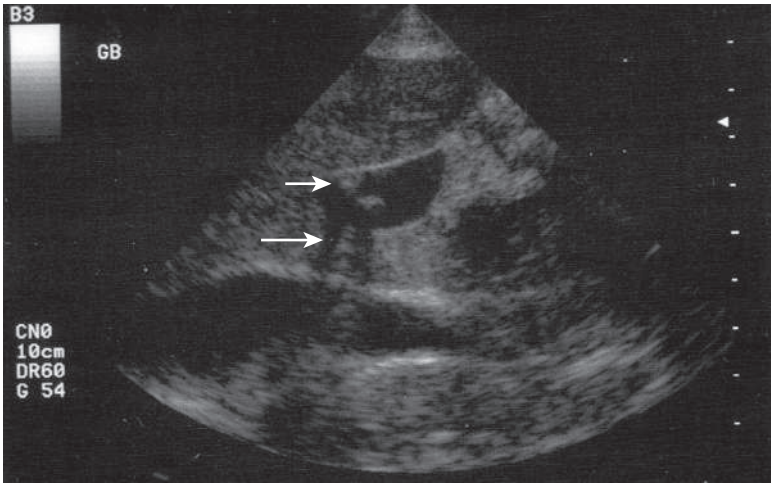

Figure 37-1. Ultrasound image reveals an anechoic gallbladder containing two echogenic stones, which are creating acoustic shadowing inferiorly. The *short arrow* is pointed to the gallstones within the gallbladder, and the *long arrow* points to the shadowing effect of the stones.

6. 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. 37-1). Other helpful findings include a thickened gallbladder wall (>3 mm), fluid collections around the gallbladder (pericholecystic fluid), and common ductal dilation (>6 mm). As noted, the sonographic Murphy sign is very sensitive for cholecystitis. In fact, it has been found to be even more sensitive in the hands of emergency physicians than ultrasound technicians or radiologists. Ultrasound overall is 94% sensitive and 78% specific for identifying cholecystitis.

KEY POINTS: ULTRASOUND FINDINGS OF CHOLECYSTITIS

1. Presence of gallstones
2. Gallbladder wall thickening greater than 3 mm
3. Pericholecystic fluid
4. CBD dilation greater than 6 mm

7. When should elective surgery be considered in patients with asymptomatic cholelithiasis?

Cholecystectomy should be considered in patients with diabetics, patients with a porcelain gallbladder, and patients with a history of biliary pancreatitis.

- Patients with diabetes have increased morbidity and mortality when urgent cholecystectomy is done in the setting of cholecystitis.
- Calcified or porcelain gallbladders have a 20% association with carcinoma.
- The risks of pancreatitis may outweigh the risks of elective cholecystectomy.

8. What are Courvoisier law, Klatskin tumor, and Fitz-Hugh-Curtis syndrome?

- The Courvoisier 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.
- A Klatskin 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.

9. What is porcelain gallbladder?

Porcelain gallbladder is a gallbladder with calcified walls. This is an important finding because 20% are associated with carcinoma, and it is an indication for cholecystectomy in asymptomatic patients.

10. Are all gallstones created equal?

No, the most common are cholesterol stones and usually are found in the stereotypic female patient who is overweight, age 40 years or older and not yet menopausal. Patients of Asian descent, those with parasitic infections *(Ascaris lumbricoides)*, chronic liver/biliary disease, or chronic hemolysis states (i.e., sickle cell disease, spherocytosis) are more likely to have pigment stones.

11. What is endoscopic retrograde cholangiopancreatography (ERCP)? What is the most common complication seen in 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.

12. What are liver function tests?

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are markers of acute liver injury, but they 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 PT. The liver also synthesizes albumin, although its longer half-life makes it a better marker of subacute or chronic liver disease.

13. 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 and is secondary to biliary obstruction from cholestasis, gallstones, tumors, or strictures.

14. State the major causes of acute hepatitis.

Hepatitis is caused by viruses such as hepatitis A through E, 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.

15. 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 as occur through 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. Hepatitis D requires coinfection with hepatitis B to replicate, but when present imparts higher risk of a more severe course and progression to fulminant hepatitis.

16. What is the new treatment for hepatitis C?

Traditionally, treatment has been with interferon and ribavirin, with or without a protease inhibitor. Recently, newer antivirals used alone or in combination have been found to have significant virologic response in patients with hepatitis C. However, with costs ranging from $84,000 to $100,00 for a course of treatment, this may not be a feasible option for many patients.

17. What is the most common form of liver disease in the United States?

Alcoholic hepatitis is the most common form and is most often diagnosed by history, but highly suggestive associated findings include spider angiomas, gynecomastia, palmar erythema, ascites, and elevated AST and ALT in a ratio of greater than 2:1.

18. What are discriminant function, the MELD score, and the Glasgow score?

These are scores that provide the clinician with an indication of the severity of alcoholic hepatitis.

- Maddrey discriminant function is calculated as

$$[4.6 \times (\text{Patient's PT} - \text{Control PT})] + \text{Serum bilirubin level}$$

  PT is reported in seconds, and serum bilirubin level is reported in milligrams per deciliter. Higher values, especially greater than 32, imply more severe hepatitis and should prompt the physician to consider initiating corticosteroid treatment to decrease mortality.
- The Model for End-Stage Liver Disease (MELD) score incorporates bilirubin, international normalized ratio (INR), and creatinine to create a score that gives an indication of prognosis and risk of death while a patient is awaiting liver transplant for alcoholic hepatitis. Multiple calculators are available online. A higher score indicates a worse prognosis.
- The Glasgow score incorporates bilirubin, PT, creatinine, age, and serum albumin to create a score that gives an indication of prognosis. Multiple calculators are available online. Higher scores correlate with a worse prognosis. Higher values, especially if greater than 9, indicate more severe hepatitis and should prompt the physician to consider initiating corticosteroid treatment to decrease mortality.

KEY POINTS: CRITERIA FOR ADMISSION IN PATIENT WITH HEPATITIS

1. Coagulopathy, INR greater than 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. Ammonia is produced in the intestines and liver as a by-product of protein metabolism and intestinal flora. When portal hypertension occurs, postal systemic shunting causes the ammonia to bypass the liver, where it would normally be metabolized. These increased levels of systemic ammonia cause it to cross the blood-brain barrier, resulting in impaired neurotransmission and neuronal dysfunction. Hepatic encephalopathy 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 produce 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 30 to 45 mL orally (per os [PO]) every 6 to 8 hours
3. Neomycin 4 to 12 g PO daily, divided every 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 fever, abdominal pain, or mental status changes. Paracentesis is diagnostic if tests show 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), reduction of portal pressure (octreotide has become the preferred agent in the United States; the alternative, vasopressin plus

nitroglycerin, has been associated with dangerous side effects), and initiation prophylactic antibiotics (ceftriaxone 1 g intravenously [IV]). If necessary, emergent transjugular intrahepatic portosystemic shunt may be indicated to further reduce portal pressure. 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 hepatorenal syndrome (HRS). One study showed a 38% 1-year survival rate in patients with HRS. Of note, rapidly progressive renal failure in HRS developing over 2 weeks indicates a more fulminant course, has an extremely high mortality, and can be an indication for referral for transplantation.

KEY POINTS: PERITONEAL FLUID CRITERIA FOR SBP

1. White blood cell count greater than 1000 cells/mm^3
2. Neutrophil count greater than 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 patient who has had a liver transplant?

Transplant rejection is common and manifests as fever, pain, and elevated transaminases and bilirubin. This can be treated with high-dosage 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, *Pneumocystis,* and fungal infection should be considered.

WEBSITES

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Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. Elan S. Levy and Kaushal H. Shah for their previous contributions to this chapter.

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QUESTIONS

1. A 43-year-old woman comes to the ED with epigastric pain radiating to her right upper quadrant. Abdominal examination shows tenderness to palpation in the epigastrium and right upper quadrant, and when taking deep breaths, she halts inspiration upon palpation of the right upper quadrant. The most appropriate imaging study in the ED is:
   a. HIDA scan
   b. Plain radiograph of the abdomen
   c. Abdominal computed tomography (CT) scan
   d. Abdominal ultrasound
   The correct answer is *d.*
2. A 27-year-old female has nausea, vomiting, and abdominal pain. Laboratory studies reveal elevation in AST and ALT. This is an indication of:
   a. Acute liver failure
   b. Acute liver injury
   c. Chronic hepatitis
   d. Chronic alcohol abuse
   The correct answer is *b.*
3. A 63-year-old male with a history of cirrhosis comes to the ED with bright red emesis. His vital signs are P 123, BP 94/60, R 28, T 36.2°C (97.2°F), SaO_2 99% RA. Appropriate immediate interventions include all of the following except:
   a. Calculation of discriminant function and administration of corticosteroids if appropriate
   b. Two large-bore IVs and fluid resuscitation
   c. Administration of octreotide
   d. Administration of ceftriaxone IV
   The correct answer is *a.*

RENAL COLIC AND SCROTAL PAIN

Christopher M.B. Fernandes, MD

CHAPTER 38

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 may be misdiagnosed as renal colic. 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 conditions. 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. What factors predict a high probability of ureteral stones?

Five factors are most predictive of ureteral stones.
1. Male sex
2. Short duration of pain
3. Nonblack race
4. Presence of nausea or vomiting
5. Microscopic hematuria

6. 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

Abstract

This chapter examines common emergencies related to presenting complaints suggestive of renal colic and scrotal pain. It describes the workup and treatment of these emergencies.

Keywords:

renal colic, scrotal pain, testicular torsion, epididymitis, ureteral calculus

7. Why is helical computed tomography (CT) now the diagnostic test of choice for suspected ureteral calculus?

Helical noncontrast CT has replaced intravenous pyelogram (IVP) as the preferred diagnostic test. 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 test. The marginal cost is less, and it can identify other important causes of flank pain.

8. Is pregnancy a contraindication to CT of the kidney, ureter, and bladder (KUB)?

Ultrasound is the investigation of choice in pregnant patients, but if ultrasound is nondiagnostic, a limited IVP I (scout film and 20-minutes postinjection film, preferably coned to the area of concern) may be appropriate because the risk of radiation from CT KUB is greater than risk of exposure in 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 confirm that a visualized stone is in, rather than overlying, the ureter. Films made when the patient is in a prone position often provide a better view of the ureter than do those made with the patient in a standard supine position.

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.

Common sites include 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 radiographic 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?

Reexamine 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. 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 workup 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 rate of procedural interventions, with an overall cost increase attributable to stones compared with the pre-ESWL era.

21. What are the basics of ED treatment of renal colic?

Basic treatment involves hydration, analgesia, and antiemetics. Patients who have clinical dehydration secondary to vomiting and decreased oral intake, and those for whom radiocontrast media study is planned, should receive intravenous fluid hydration. Various analgesics and antiemetics are available for rapid control of symptoms (Table 38-1). Intravenous pain control is the mainstay of ED treatment. Analgesic treatment should not be delayed while waiting for test results. Opiate analgesics have long been the standard medication. Rectal or intravenous nonsteroidal antiinflammatory 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 and/or urology consultation?

Consider hospitalization for patients with high-grade obstruction, intractable pain or vomiting, associated urinary tract infection, a solitary or transplanted kidney, and an uncertain diagnosis. Obtain urologic consultation for patients with stones larger than 5 mm in diameter, urinary extravasation, and renal insufficiency, regardless of symptoms.

Table 38-1. Analgesics and Antiemetics for Renal Colic

MEDICATION	DOSAGE	FREQUENCY	AS NEEDED (prn)
Opioid Analgesics			
Anileridine (Leritine)	PO 50 mg	q4h	prn
Hydromorphone (Dilaudid)	IV 1-2 mg	q2-4h	
Meperidine (Demerol)	IV 25-50 mg	q5-10min	prn
Morphine	IV 3-5 mg	q5-10min	prn
	IM 0.1-0.2 mg/kg	q3h	prn*
Oxycodone and acetaminophen (Percocet)	PO 2 tabs	q4h	prn
Oxycodone and acetylsalicylic acid (Percodan)	PO 2 tabs	q4h	prn
Antiemetics			
Metoclopramide (Reglan)	IV 10-20 mg	q15min	prn
Perphenazine (Trilafon)	IM 5 mg	q6h	prn*
	PO 4 mg	q6h	prn
Prochlorperazine (Compazine)	IV 5-10 mg	q4h	prn
	IM 5-10 mg	q6h	prn*
	PO 5-10 mg	q4h	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	q6h	
	IM 30 mg	q6h	

IM, Intramuscularly; *IV*, intravenously; *prn*, as needed; *PO*, per os (by mouth); *q*, every.
*Intramuscular route not recommended for ED management of acute, severe pain.

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. A follow-up appointment with a urologist within 1 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. High-grade obstruction
2. Intractable pain or vomiting
3. Associated urinary tract infection
4. Solitary or transplanted kidney
5. 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 about 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 smaller than 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. Calcium channel blockers or α-blocker therapy can be used. With some data suggesting faster passage with tamsulosin, treatment can be initiated for 4 weeks. However, a small randomized double-blind study suggested that tamsulosin did not accelerate expulsion of distal ureteral stones.

28. What is the differential diagnosis in a patient who has an acutely painful scrotum?

- Testicular torsion
- Torsion of the testicular or epididymal appendages
- Epididymitis
- Orchitis
- Scrotal hernia
- Testicular tumor
- Renal colic
- Henoch-Schönlein purpura
- Fournier 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 "bell-clapper deformity"), and the testis can 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. Epididymoorchitis
4. Scrotal hernia
5. Testicular tumor
6. Fournier 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 a 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 consultation with a urologist and surgical exploration. If consultation for surgery 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. Testis viability rates of 100%, 70%, and 20% for 6, 6 to 12, and 12 to 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 workup 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 has supplanted radionucleotide scintigraphy as the diagnostic imaging study 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. Most urologists will require confirmation of testicular torsion on a Doppler ultrasound study before surgical intervention.

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 appendix testis and appendix epididymis?

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 testicular 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 experience increasing, dull, unilateral scrotal pain over several 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 patient who is febrile and appears toxic with epididymitis, or when testicular or epididymal abscess should be excluded. Inpatient 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 empiric 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 care with a urologist within 5 to 7 days is recommended.

44. What is Fournier gangrene?

Fournier 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 gangrene are similar to those of necrotizing fasciitis. Diabetes mellitus, alcohol abuse, and local trauma are known risk factors. Empiric 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.

45. What organisms are commonly seen with Fournier gangrene?

This is typically a polymicrobial infection, with an average of four isolates per case. *E. coli* is the predominant aerobe, and *Bacteroides* is the predominant anaerobe.

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QUESTIONS

1. The diagnostic study with the highest specificity and sensitivity to make the diagnosis of ureteral calculus is:
   a. IVP
   b. Abdominal sonogram
   c. CT KUB with intravenous contrast
   d. CT KUB without contrast
   The correct answer is *d.*
2. Which of the following is least likely to support the diagnosis of epididymitis?
   a. Fever
   b. Sudden onset of pain
   c. Dysuria
   d. Urethral discharge
   The correct answer is *b.*
3. What is the most serious possible cause of scrotal pain?
   a. Epididymitis
   b. Orchitis
   c. Testicular torsion
   d. Inguinal hernia
   The correct answer is *c.*

CHAPTER 39

ACUTE URINARY RETENTION

John P. Marshall, MD

1. What is acute urinary retention (AUR)?

AUR is characterized by a painful inability to urinate. It 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 the absence of pain and overflow incontinence. It most commonly 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 60 years, 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
  - Clogged or crimped Foley catheter
- Neurogenic
  - Spinal cord injuries
  - Herniated lumbosacral disks (cauda equina syndrome)
  - Central nervous system (CNS) tumors
  - Stroke
  - Diabetes
  - Multiple sclerosis

Abstract

Acute urinary retention is a common, painful condition that requires immediate management. This condition is treated with catheter placement and bladder drainage, followed by further evaluation for a cause of the retention and close urologic follow-up care.

Keywords:

bladder, urethra, prostate, Foley catheter, retention, urine, coudé, obstruction, catheter

- Encephalitis
- Tabes dorsalis
- Syringomyelia
- Herpes simplex
- Herpes zoster
- Alcohol withdrawal

- Pharmacologic (see Question 14)
  - Anticholinergics
  - Antihistamines
  - Antidepressants
  - Antispasmodics
  - Narcotics
  - Sympathomimetics
  - Antipsychotics
  - Antiparkinsonian agents
- 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?

Treat AUR with catheterization and bladder decompression using a Foley catheter.

9. What if I can not 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 a bigger catheter better?

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 filiformes and followers or catheter guides. These

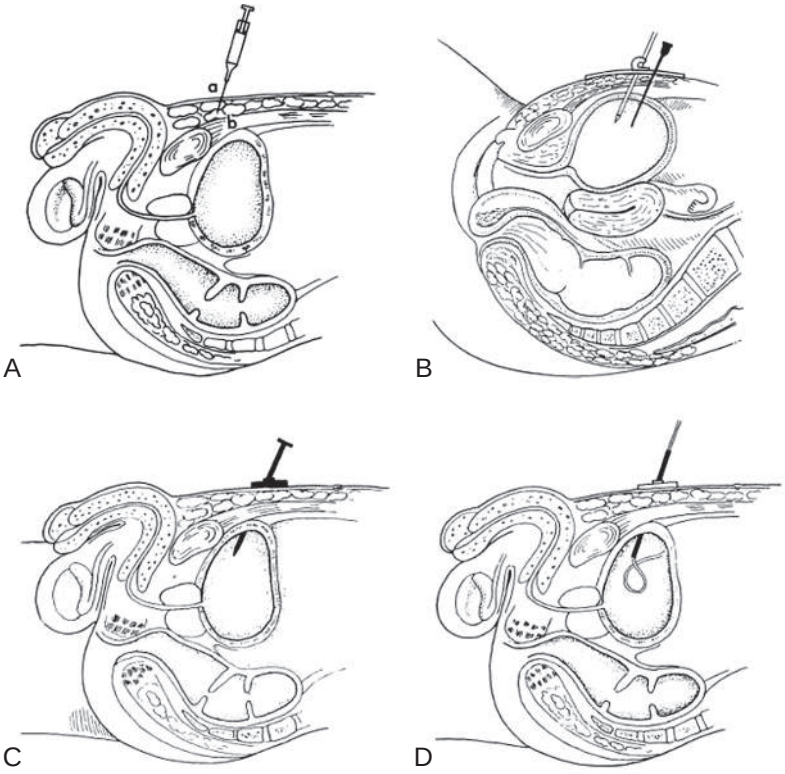

Figure 39-1. Suprapubic catheterization. *(From Roberts J, Hedges J:* Clinical procedures in emergency medicine, *Philadelphia, 2000, Saunders.)*

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?

Suprapubic catheterization is a procedure used to pass a urinary catheter directly into the bladder through the lower anterior abdominal wall (Fig. 39-1). 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.

KEY POINTS: TREATMENT OPTIONS FOR AUR

1. Foley catheter placement
2. Coudé catheter placement
3. Filiformes and followers
4. Suprapubic catheterization

Table 39-1. Medications That Can Cause Acute Urinary Retention

- Sympathomimetics (α-adrenergic)
  - Ephedrine
  - Pseudoephedrine (Sudafed, Actifed)
  - Phenylephrine hydrochloride (Neo-Synephrine, Dimetapp)
  - Phenylpropanolamine hydrochloride (Contac)
  - Amphetamine
  - Cocaine
- Sympathomimetics (β-adrenergic)
  - Isoproterenol
  - Terbutaline
- Antidepressants
  - Tricyclic
  - Fluoxetine (Prozac)
- Antidysrhythmics
  - Quinidine
  - Disopyramide (Norpace)
  - Procainamide
- Anticholinergics
  - Atropine
  - Dicyclomine (Bentyl)
- Antihistamines
  - Chlorpheniramine (Chlor-Trimeton)
  - Diphenhydramine (Benadryl, Unisom)
  - Hydroxyzine (Atarax, Vistaril)
- Antiparkinsonian Agents
  - Benztropine (Cogentin)
  - Amantadine (Symmetrel)
  - Levodopa (Sinemet)
  - Trihexyphenidyl (Artane)
- Hormonal Agents
  - Progesterone
  - Estrogen
  - Testosterone
- Antipsychotics
  - Haloperidol
  - Chlorpromazine (Thorazine)
  - Prochlorperazine (Compazine)
  - Risperidone (Risperdal)
  - Clozapine (Clozaril)
  - Quetiapine (Seroquel)
- Antihypertensives
  - Nifedipine (Procardia)
  - Hydralazine
  - Nicardipine
- Muscle Relaxants
  - Diazepam (Valium)
  - Cyclobenzaprine (Flexeril)
- Narcotics
  - Morphine sulfate
  - Codeine
  - Meperidine (Demerol)
  - Hydromorphone hydrochloride (Dilaudid)
- Miscellaneous
  - Indomethacin
  - Metoclopramide (Reglan)
  - Carbamazepine (Tegretol)
  - Mercurial diuretics
  - Dopamine
  - Vincristine
  - MDMA
  - Cannabis

MDMA, 3,4-methylenedioxymethamphetamine.

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 39-1 presents the broad categories, as well as some specific medications that can cause AUR.

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 to 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, herpes simplex infection, and the postoperative state.

16. Name the most common complications of AUR.

Complications include 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?

Autonomic dysreflexia/hyperreflexia is an abnormality of the autonomic nervous system seen in patients with long-standing cervical or high thoracic spinal cord lesions (i.e., patients with quadriplegia and high paraplegia). 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 is called *postobstruction diuresis*. 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 the urinary system is 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 visits. 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 before discharge from the ED.

20. Do medications play any role in the treatment of AUR?

Although the mainstay of treatment is bladder decompression, medications play an important adjunctive role in the treatment of this condition. First, antibiotics should certainly be given for urinary tract infection in the setting of AUR. The choice of antibiotics should be guided by your local antibiogram. Second, patients with a suspected prostatic or bladder cause may benefit from an α-1 adrenergic blocking agent such as doxazosin, prazosin, or tamsulosin. These agents work at the prostate and bladder neck to decrease resistance and ease normal micturition, and are generally recommended for men without another obvious precipitating cause.

CONTROVERSY

21. I have heard that gradual emptying of the distended bladder best helps prevent complications. Is this true?

Traditionally, the medical literature had 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. 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.

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QUESTIONS

1. Which medication is known to cause AUR?
   a. Ciprofloxacin
   b. Diphenhydramine
   c. Metoprolol
   d. Tamsulosin
   The correct answer is *b.*
2. A patient has painful AUR, but the nurse is unable to pass a Foley catheter. What is the next step in the management of this patient?
   a. Attempt Coudé catheter placement
   b. Passage of filiformes and followers
   c. Suprapubic catheterization
   d. Urology consultation
   The correct answer is *a.*
3. Which cause for AUR is should be treated with an α-1 blocking agent in addition to urinary catheter placement?
   a. Opioid use
   b. Prostatic hypertrophy
   c. Urethral stricture
   d. Spinal cord injury
   The correct answer is *b.*

CHAPTER 40

URINARY TRACT INFECTION: CYSTITIS, PYELONEPHRITIS, AND PROSTATITIS

Renee A. King, MD, MPH

1. Define terminology pertinent to the range of urinary tract infections (UTIs).

- Bacteriuria: The occurrence of bacteria within the urinary tract. The up-to-date parameter for significant bacteriuria is 10^5 colony-forming units (CFU)/mL.
- Cystitis: Inflammation of the bladder caused by significant bacteriuria in conjunction with bladder mucosal (urothelium) invasion. Clinicians commonly use the term *UTI* to mean *cystitis.*
- Pyelonephritis: Infection of the parenchyma and collecting system of the kidney; usually seen with flank pain, fever, and significant bacteriuria.
- Urethritis and acute urethral syndrome: Dysuria, frequency, and urgency in the absence of significant bacteriuria.
- Prostatitis: Inflammation of the prostate. It can be seen with a wide range of symptoms and can be acute or chronic.

KEY POINTS: CHARACTERISTICS OF CYSTITIS

1. Dysuria
2. Frequency
3. Urgency
4. Suprapubic pain
5. Significant bacteriuria

2. What are the most common causes of UTI?

Most UTIs are caused by one pathogen, and the most common pathogen is *Escherichia coli*, which accounts for about 70% to 95% of acute UTIs. *Staphylococcus saprophyticus* is the second most cause of acute cystitis. Complicated UTIs may have a broader range of microbes, including *Enterococcus, Providencia, Pseudomonas, Serratia,* and *Staphylococcus*, and occasionally fungi.

3. What is asymptomatic bacteriuria?

The presence 100,000 CFU/mL of clean-catch midstream urine sample from a patient without symptoms is consistent with asymptomatic bacteriuria. If two successive specimens result in the the same microbe at the same concentration, the likelihood of true bacteriuria is 80% to 95%.

4. Should asymptomatic bacteriuria always be treated?

A healthy female who is not pregnant does not need treatment for asymptomatic bacteriuria.

A pregnant female with asymptomatic bacteriuria does need treatment with antibiotics. About 20% to 40% of these patients go on to develop UTI or pyelonephritis, which is associated with prematurity and low birth weight.

Preoperative patients should be treated with antibiotics, because this has been shown to decrease the potential for postoperative infections.

Evidence indicates that antibiotic treatment is not needed for patients with indwelling catheters who have asymptomatic bacteriuria. This only leads to resistance.

5. List the differential diagnoses of dysuria.

- Infectious causes: Cystitis, epididymitis, prostatitis, pyelonephritis, urethritis (gonococcal versus nongonococcal), vulvovaginitis

Abstract

This section discusses urinary tract infection (UTI), an illness that is commonly diagnosed and treated in the ED setting. In this chapter, definitions of urologic terms, which describe certain bladder, kidney, and urologic disorders, are included. Etiologies, common signs and symptoms, and treatments of various infections of the genitourinary (GU) tract are a significant portion of this discussion. Review questions have been provided at the end, which will allow the reader to review and retain what has been discussed in the chapter.

Keywords:

urinary tract infection (UTI), cystitis, pyelonephritis, prostatitis, bacteriuria

- Structural causes: Calculi, neoplastic lesions
- Traumatic causes: Allergy, blunt trauma, chemical irritants, sexual intercourse or assault

6. When should a pelvic examination be performed in a female patient with dysuria?

If the history and physical raise the concern for cause other than classic UTI, then a pelvic examination should be performed. Clinical conditions such as external inflammation indicative of vulvovaginitis, low abdominal aching or bilateral flank discomfort to rule out pelvic inflammatory disease, any account of injury or presence of irritation caused by a chemical substance, and any person with risk factors for sexually transmitted infection or sexual assault should receive a pelvic examination. Furthermore, patients who are unresponsive to empiric antibiotic treatment for cystitis and patients with negative urine tests or cultures undergo pelvic examinations.

7. What tests can be done to evaluate for UTI?

Urinalysis via laboratory testing with dipstick or microscopy is the first step in evaluating for presence of UTI. Urine culture with Gram staining is not the first choice of testing, because it is a lengthier process and more expensive. However, it is useful for the selection of the most appropriate antibiotic therapy, especially in patients with risks for complicated UTI. Common forms of urinalysis include the following:

- Dipstick test
  - A dipstick test includes blood, glucose, leukocyte esterase (≥+1), nitrite, and protein. A positive dipstick is a good screening test (about 75% to 90% sensitive, with 75% to 85% specific), but if the history obtained is strongly suggestive of UTI, a negative dipstick does not reliably rule out infection.
- Microscopy
  - Bacteriuria: A finding of 10^5 CFU/mL is used to define significant bacteriuria in an asymptomatic patient; however, as few as 10^2 CFU/mL with symptoms and pyuria are suggestive of UTI.
  - Epithelial cells: The presence of epithelial cells are used mainly to estimate perineal contamination of midstream specimens. Although epithelial cells are present throughout the urinary tract, their occurrence on urinalysis is typically a vaginal epithelial cell contaminant.
  - Leukocyte esterase: This enzyme is present in neutrophils, and it is able to convert indoxyl carboxylic acid to an indoxyl moiety in leukocytes. A positive test is indicative, but not confirmatory, for pyuria.
  - Nitrite: This is present in acute cystitis, and it is produced from nitrate by nitrate reductase, an enzyme that is found in gram-negative microbes. To be positive, the bacteria must act on the urine for 6 hours, so a sample from the first urine of the morning is ideal for optimal testing. This test is a specific but not sensitive test for acute cystitis.
  - Pyuria: Greater than or equal to 10 leukocytes/mL observed by direct microscopy correlates significantly with acute cystitis.

8. When should a urine culture be ordered?

For uncomplicated cystitis in healthy patients, urine culture is unnecessary. A culture with sensitivities is useful in cases of complicated UTI or in pyelonephritis cases.

9. What comprises a complicated UTI?

A complicated UTI is any underlying preexisting condition that predisposes the patient to infection but also reduces the effectiveness of standard treatments.

KEY POINTS: RISK FACTORS OF COMPLICATED UTI

1. Anatomic abnormality of urinary tract
2. Diabetes
3. Foreign body (urethral catheter, nephrostomy tube)
4. Hospital-acquired infection
5. Kidney failure
6. Multidrug resistant organism
7. Obstruction of urinary tract (prostate hypertrophy, stone, stenosis)
8. Pregnancy
9. Renal failure/renal transplant
10. Immunosuppression

10. What is the treatment for uncomplicated UTI?

Even without treatment, approximately 25% to 42% of uncomplicated cystitis resolves spontaneously, but standard therapy usually involves antibiotics. Choosing the appropriate antibiotic depends on efficacy (including local resistance rates and geographic variation of pathogen resistance), cost, risk of adverse effects, and antibiotic availability.

First-line treatment for uncomplicated UTI in women includes nitrofurantoin 100 mg twice daily (bid) for 5 to 7 days, trimethoprim-sulfamethoxazole (TMP-SMX) 160 mg/800 mg bid for 3 days, or fosfomycin 3 g orally, one-time dose. Although the aforementioned are considered standard first-line treatment, local recommendations for therapy may vary in different geographic regions over time.

11. Is there a role for nonantibiotic treatment?

With some cases of cystitis, phenazopyridine (Pyridium) may be useful. This is a urinary analgesic that can give some relief in the presence of significant dysuria. A 48-hour course of 200 mg thrice daily after meals is the usual regimen. Patients should be warned that body fluids may likely turn orange. This medication should not be used longer than 2 days, because it may mask worsening symptoms that would require reevaluation.

12. What is the treatment for pyelonephritis?

In healthy patients who are not pregnant, 14 days of antibiotics is recommended. Fluoroquinolones such as ciprofloxacin, levofloxacin, and ofloxacin are considered first-line therapies for uncomplicated pyelonephritis. Other options include cefixime or cefpodoxime. A one-time dose of gentamycin or ceftriaxone can be used parenterally if needed.

KEY POINTS: RISK FACTORS OF PYELONEPHRITIS

1. Fever
2. Flank pain
3. Significant bacteriuria
4. Costovertebral angle discomfort

13. Which patients with pyelonephritis require admission?

Admission for pyelonephritis should be considered for patients who are unable to tolerate oral medications or fluids, immunocompromised patients, patients with severe illness or sepsis, patients in extreme pain, and patients with little or no home support or resources. Admission should also be considered for patients who are pregnant. Although some patients in the early first trimester may be sent home with oral antibiotics, a discussion with their obstetrician specialist is prudent.

14. When should imaging be obtained for pyelonephritis?

If fevers persist for longer than 48 to 72 hours, then obtaining an ultrasound or computed tomography may be warranted to evaluate for abscess or obstruction.

KEY POINTS: POSSIBLE ADMISSION CRITERIA FOR PYELONEPHRITIS

1. Complicated cystitis
2. Inability to tolerate oral medication or hydration
3. Lack of support at home
4. Pregnancy
5. Sepsis/extreme illness

15. What are the differences when treating complicated cystitis?

The length of treatment in complicated UTI is longer. Treatment should be extended to 7 to 10 days. If the patient is pregnant, antibiotic choices have to be made that will be safe during pregnancy. Common medications include amoxicillin, cephalexin, and nitrofurantoin. Intravenous (IV) antibiotics, such as ceftriaxone, may also be needed, especially if the patient cannot tolerate oral medications.

16. What is the presentation of acute bacterial prostatitis?

Most patients (80%) experience dysuria, frequency, and urgency. About 60% of these patients are febrile. Myalgias, perineal pain, and rigors can also be seen with bacterial prostatitis.

17. What is the treatment for acute prostatitis?

A urinalysis should be obtained to evaluate for bacterial infection. Catheterization and prostate massage should not be performed because of pain, but also because of the risk of bacteremia. Antibiotics are the mainstay of treatment. Common medications are aminoglycosides, fluoroquinolones, and cephalosporins. TMP-SMX may also be used, although it has decreased sensitivity. Treatment should be for 30 days to resolve symptoms and prevent chronic prostatitis.

18. What are signs and symptoms of chronic bacterial prostatitis?

Chronic bacterial prostatitis is a recurrent subacute infection, and it is the primary cause of recurrent UTI in males. Examination is variable, but common symptoms include pain with urination, frequency, and urgency. Other complaints include back pain, pain in the scrotum and perineum, painful ejaculation, and hematospermia. Fevers and chills are not usually seen. Symptoms may be present for 3 or more months.

19. What is the treatment for chronic bacterial prostatitis?

Treatment can be difficult because of the relapsing nature of the illness. Generally, long-term treatment is needed for 2 to 3 months. Referral to a urologist is also recommended.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Sara M. Krzyzniak for her previous contributions to this chapter.

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QUESTIONS

1. Which of the following is least likely to be associated with cystitis?
   a. Frequency
   b. Fever
   c. Urgency
   d. Dysuria
   The correct answer is *b.*
2. All of the following support the diagnosis of UTI except:
   a. Positive leukocyte esterase
   b. Bacteruria
   c. Epithelial cells
   d. Nitrite-positive dipstick
   The correct answer is *c.*
3. All of the following are risk factors for complicated UTI except:
   a. Diabetes
   b. Pregnancy
   c. Ureteral calculus
   d. Nausea and vomiting
   The correct answer is *d.*

CHAPTER 41

CHRONIC KIDNEY DISEASE AND DIALYSIS

Allan B. Wolfson, MD, FACEP, FACP

1. Is kidney 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. CKD 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 kidney 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 acute kidney injury (AKI) or 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 a high concentration of glucose that, through osmosis, pulls water from the intravascular space into the dialysate, acting to correct volume overload. For patients with CKD using chronic ambulatory PD (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 PD, termed *automated PD (APD)* or *continuous cycling PD (CCPD),* exchanges are performed by an automatic cycling machine. For both types, special peritoneal access is required in the form of a surgically implanted 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 come 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.

Abstract

The patient with chronic kidney disease (CKD) is subject to the complications of renal failure, as well as those related to either hemodialysis or peritoneal dialysis. When patients come to the ED, potentially life-threatening conditions such as hyperkalemia or severe volume overload are considered first and treated as necessary. Occasionally, immediate emergency dialysis is indicated. Most complaints need to be evaluated promptly, keeping in mind the most likely underlying causes in renal patients, and remembering that fluid administration and medication dosages must be adjusted to account for the altered physiology in chronic kidney disease. It is usually advisable to consult with the patient's nephrologist or dialysis nurse to ensure prompt treatment and follow-up care.

Keywords:

chronic kidney disease (CKD), dialysis, hemodialysis, peritoneal dialysis (PD), vascular access, infection, peritoneal dialysis-associated peritonitis, emergency dialysis, hyperkalemia, pericardial tamponade, pericarditis, dialysis-associated hypotension

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 has signs of inflammation localized to the access site. The difficulty is that many patients only have fever and are 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 obtaining follow-up treatment. A single dose of vancomycin, 15 to 20 mg/kg 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 minimally dialyzable, and its major toxicity is to the kidneys. If gram-negative infection is suspected, a third- or fourth-generation cephalosporin or an aminoglycoside should be added to the regimen. Careful follow-up observation must 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 port. 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 site 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 every 1 to 2 years in the average patient. In contrast to other types of peritonitis, it tends to be mild clinically, and most patients' care 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 (15 to 30 mg/kg) is given intraperitoneally and may be repeated weekly. Gram-negative coverage, with a third-generation cephalosporin or an aminoglycoside, is often added. This is 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. A follow-up visit should occur after 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 instructions for follow-up care.

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?

Indications include 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.

1. 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. Patients with CKD who are in cardiac arrest always should be given IV calcium if they do not respond immediately to the first round of ACLS measures.
2. 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, 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?

Patients with CKD and 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 they are 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 patient using PD who has 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 patients who do not receive dialysis. IV calcium (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 10 U/kg, respectively, as a slow IV infusion) also move potassium into cells but require close serial monitoring of blood glucose levels.

IV sodium (50 mEq over 5 minutes) has a similar action but is not uniformly as effective. Moreover, it can exacerbate volume overload and can acutely decrease the ionized calcium.

Sodium polystyrene (Kayexalate), a sodium-potassium exchange resin typically given orally with sorbitol to enhance passage through the gut, has been found to have limited efficacy and is no longer in general use.

In all cases of acute hyperkalemia, the serum potassium level should be checked often, and continuous ECG monitoring is mandatory until definitive treatment with dialysis can be initiated.

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

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 can be 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. You should be certain before embarking on this course, however, that the patient's symptoms are the result of 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 patients with CKD 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 because of 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 CKD?

The most common entities are hypovolemia after dialysis, sepsis, hemorrhage, and acute pericardial tamponade.

18. What are the major causes of altered mental status in patients with end-stage renal disease (ESRD)?

Disequilibrium 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.

KEY POINTS: TREATMENT OF HYPERKALEMIA

1. IV calcium
2. Inhaled albuterol
3. IV glucose and insulin
4. IV sodium bicarbonate
5. Dialysis

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QUESTIONS

1. Emergency hemodialysis is most likely to be needed in which of the following?
   a. Severe symptomatic hypocalcemia
   b. Acute pulmonary edema
   c. Severe uremic symptoms (e.g., nausea, vomiting, weakness)
   d. Acute ischemic chest pain
   The correct answer is *b.*
2. The most reliable way to diagnose peritonitis associated with chronic peritoneal dialysis is:
   a. Emergency CT scan of abdomen and pelvis, without contrast
   b. Increased peripheral white blood cell count with more than 85% polymorphonuclear leukocytes
   c. Free air seen on upright plain abdominal film
   d. Cloudy dialysate drainage reported by patient's family
   The correct answer is *d.*
3. In a hemodialysis patient with no fever, which of the following is the least likely to be the cause of acute hypotension?
   a. Hypovolemia
   b. Cardiac tamponade
   c. Bacteremia
   d. Increased response to antihypertensive medication
   The correct answer is *c.*

HEMOSTASIS AND COAGULOPATHIES

Kathryn Getzewich, MD, MBA, and Alvin Wang, DO, FAAEM

CHAPTER 42

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.

1. 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.
2. 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.
3. 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

Abstract

Hemostasis is a balance between excessive bleeding and thrombosis. It is normally regulated in the body by platelets and numerous clotting factors. This chapter discusses the normal process of regulation, the various defects in these hemostatic mechanisms, and the means to treat clotting and bleeding disorders.

Keywords:

hemostasis, clotting, bleeding, coagulation, platelet, coagulopathy, transfusion, hemorrhage, hemophilia, warfarin, heparin, international normalized ratio (INR), thrombin, fresh frozen plasma (FFP)

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 commonly knowledgeable about their individual diseases. Platelet disorders commonly result in petechia, purpura, epistaxis, and gum and other mucosal bleeding. These disorders are common in women and usually acquired, as opposed to congenital. In contrast, problems with coagulation factors 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), hemolysis with elevated liver enzymes and low platelets (HELLP) syndrome
- Splenic sequestration (hypersplenism, rare): Hematologic malignancy, portal hypertension, autoimmune hemolytic anemia, hereditary spherocytosis
- Dilution: Massive transfusion
- Laboratory 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 in men. Severity waxes and wanes with only a 1% mortality, but spontaneous remission is rare. It may respond to therapy with glucocorticoids and/or intravenous (IV) immunoglobulin. Splenectomy, monoclonal antibody therapy, and immunosuppressive agents can be considered in patients refractory to this treatment. 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.

1. Fluctuating change in mental status
2. Thrombocytopenia
3. Fever (in 90% of patients)
4. Microangiopathic hemolytic anemia
5. 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 between 10 and 40 years of age and 60% are female. When untreated, there is 80% mortality at 3 months as a result of microthrombi in the heart, brain, and kidneys. Plasmapheresis has reduced this rate 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 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* species.

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 5 days. Platelet counts should be monitored, and transfusion of platelets should begin if the count drops to 50,000/μL. Massive transfusion is the replacement of the patient's total body volume of blood or the transfusion of 10 units or more of packed red blood cells (PRBCs) over 24 hours. In a patient who has sustained major trauma and is likely to require massive transfusion, a ratio of 1:1:1 of PRBCs to platelets to FFP is widely recommended.

13. How does aspirin increase bleeding?

Aspirin blocks cyclooxygenase, which decreases thromboxane formation, thereby leading to decreased platelet aggregation and less vasoconstriction. Aspirin poisons this reaction for the life of the platelet. Nonsteroidal antiinflammatory 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 caused by the deficiency is unlikely. In patients with platelet counts of 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 5000/mL. They are usually ordered six at a time.

15. What is the most common inherited bleeding disorder?

It is von Willebrand disease (5 to 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 (DDAVP) in the mild, most common, type I form of the disease.

In more severe types, therapy is with factor VIII concentrate, with dosing based on the patient's factor VIII level.

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 caused by 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.

Note: 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?

Use 25 U/kg for moderate bleeding, and 50 U/kg for severe hemorrhage or life-threatening bleeding site (GI, neck, sublingual, retroperitoneal, intraabdominal, 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?

Christmas disease is hemophilia B, which involves decreased factor IX activity. The clinical presentation is the same as that for hemophilia A. The genetic pattern is the same, although it is less prevalent in the population, with only one fifth of 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 Desamino-D-arginine vasopressin (DDAVP) do?

DDAVP is a synthetic analog of antidiuretic hormone. It causes release of vWF from endothelial storage sites, and increases levels of factor VIII in hemophilia A and some cases of von Willebrand disease. The dosage is 0.3 μg/kg IV; it lasts 4 to 6 hours and is most effective in mild to moderately deficient patients. Administration of DDAVP can also be helpful in obtaining hemostasis in patients with bleeding caused by uremia (i.e., patients with end-stage renal disease or those receiving dialysis).

20. What factors are affected by vitamin K deficiency, warfarin, liver disease, and banked blood?

- Vitamin K deficiency affects factors II, VII, IX, and X, the same ones affected by warfarin.
- Hepatic insufficiency affects all factors except VIII.
- Stored blood is low in factors V and 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 disease.
3. PT reflects extrinsic pathway abnormality through factor VII deficient activity.
4. Factor VII has the shortest half-life of the factors (3 to 5 hours) and causes the first manifestations of production deficiency.
5. An INR of 2 to 3 is recommended with most warfarin therapy.
6. Deficiency of factors VIII, IX, and XI account for 99% of inherited bleeding disorders. If a 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.

Definitive treatment is to treat the underlying cause (i.e., sepsis), but the coagulopathy is temporized with transfusion of platelets and FFP. Heparin may be used if fibrin deposition and thrombosis dominate the clinical picture.

22. What are heparin-induced thrombocytopenia (HIT) and HIT 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 IV 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 receiving unfractionated heparin anticoagulation therapy and 0.2% of patients medicated with low–molecular-weight heparin (LMWH). 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 studies have found subclinical deep venous thrombosis (DVT) in up to 50% of patients with HIT.

23. Need help with hemolysis, elevated liver enzymes, and low platelet count (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 to 150,000/mL) is found in 5% to 10% of third trimester pregnancies. It is even more common in pregnancies complicated by preeclampsia (15% to 20%) and eclampsia (40% to 50%). Fetal and maternal mortality are increased. Treatment is primarily supportive, although platelet transfusion may be required before cesarean section. DIC may develop.

24. How do heparin and 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 dosages, 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 dosage of 1 mg/100 U of circulating heparin to a maximum dosage of 50 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, six bags random donor platelets yields 30,000/mL increase
2. PT and INR: Extrinsic and common paths (i.e., II, VII, IX, X), warfarin
3. PTT: Intrinsic and common paths (all factors except VII and XIII), heparin
4. Severe bleeding with hemophilia A: 50 U/kg factor VIII
5. To support hemostasis until definitive diagnosis: FFP 15 mL/kg

26. How does warfarin work? How do I deal with elevated INR?

Warfarin (an oral anticoagulation therapy [OAT]) inhibits the reduction of vitamin K to its active form, causing depletion of factors II, VII, IX, and X. The starting dosage is 5 mg/day, with 4 to 5 days required for the full anticoagulant effect. Heparin or LMWH is continued in the interim owing to the early inactivation of proteins C and S, which causes a temporary procoagulant effect. The target is usually an INR of 2 to 3. Significant bleeding occurs in 3% of patients receiving 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 an elevated INR less than 5 can be treated by withholding doses until the 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 to 15 mL/kg) and 10 mg IV vitamin K, given slowly (FFP for immediate effect; vitamin K effect takes several hours).

27. What about prothrombin complex concentrates (PCCs)?

PCCs have become more commonly used to reverse warfarin. PCCs have been show to reverse elevated INR much faster than FFP, because PCC is a concentrate powder and does not need thawing. It is usually given via slow IV push and a much lower volume than FFP. All PCCs contain factors II, VII, and IX, but some products contain no or little factor VII. PCCs with normal amounts of factor VII are known as *4-factor PCCs* and also contain proteins C and S; those without factor VII are called *3-factor PCCs.*

28. What are all these new oral anticoagulants I keep hearing about?

Some new oral anticoagulants have provided stable metabolism across patients and have caused very few food or drug interactions. Because of this, they do not require monitoring of the INR. Some examples are rivaroxaban and apixaban (factor Xa inhibitors) and dabigatran (a direct thrombin inhibitor). These agents have been found to be as effective as warfarin with less risk of bleeding.

29. That sounds great! So what's the catch?

Cost is an issue, and caution is needed with the Xa inhibitors in patients with renal disease. However, the biggest concern in the ED is that there are no reversal agents. Their mechanism of action makes vitamin K and FFP ineffective.

30. So what do I do if someone taking a factor Xa or thrombin inhibitor comes in with severe bleeding?

Start with large-bore IV access for fluid administration and prepare cross-matched blood. PT/PTT and INR are not reliable measurements of the degree of anticoagulation from Xa or thrombin inhibitors, so they cannot be used to guide management. Although normal PT and PTT essentially rule out any significant amount of active medication in the blood stream, elevated values do not correlate with degree of toxicity or anticoagulation.

There may be a role for PCC, activated PCC (aPCC; also known as *antiinhibitor coagulant complex [Feiba]*), and recombinant factor VIIa. In aPCC, factor VII is in its activated form. Although animal studies have shown decreased bleeding with these agents, human in vivo studies are lacking.

31. Can anything be done to control massive hemorrhage from trauma?

Although not approved by the U.S. Food and Drug Administration (FDA) for this indication, tranexamic acid (TXA) is an antifibrinolytic agent with American Heart Association (AHA) class A evidence showing proven mortality benefit for use in patients with significant hemorrhage from trauma. As a plasmin and plasminogen activation inhibitor, TXA works by inhibiting clot breakdown rather than promoting clot formation. Maximum mortality benefit occurs when given less than 3 hours after initial injury, and is most effective when given within 1 hour after injury. Administration more than 3 hours after onset of bleeding is associated with increased mortality.

The military has been using new clotting agents to stabilize patients with traumatic bleeding on the combat field. QuickClot (Z-Medica, Wallingford, CT) is a brand of kaolin-impregnated gauze that was shown to be the most effective in controlling hemorrhage in areas not amenable to tourniquet placement. Kaolin is a nonbotanical, nonhuman, nonanimal mineral compound that promotes the activation of factor XII and platelet-associated factor XI. The HemCon (HemCon Medical Technologies, Portland, OR) 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. These two agents are already approved by the FDA for civilian use. Additional human studies are ongoing with a 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.

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QUESTIONS

1. A patient taking warfarin can be expected to have:
   a. An elevated PT/INR and elevated PTT
   b. An elevated PT/INR and normal PTT
   c. A normal PT/INR and normal PTT
   d. A normal PT/INR and elevated PTT
   The correct answer is *b.*
2. Which abnormality is a hallmark of TTP, but seen less commonly in HUS?
   a. Thrombocytopenia
   b. Hemolytic anemia
   c. Renal impairment
   d. Altered mental status
   The correct answer is *d.*
3. A patient taking warfarin with hematemesis, hypotension, and an INR of 5 should be treated by holding warfarin and:
   a. Giving 10 mg of IV vitamin K
   b. Giving 10 mg of oral vitamin K
   c. Giving FFP
   d. *a* and *c*
   The correct answer is *d.*

SICKLE CELL DISEASE

Daniel Willner, MD, and Louisa Canham, MD

CHAPTER 43

1. What is sickle cell disease (SCD)?

SCD is an inherited disorder of hemoglobin (Hb) that causes repeated vasoocclusive episodes and anemia. SCD is caused by a single nucleic acid substitution within the β-globin gene, which causes valine to be substituted for glutamic acid on the β-globin component of the Hb tetramer ($\alpha_2\beta_2$). This Hb molecule (HbS) is poorly soluble in deoxygenated conditions and polymerizes with other Hb molecules in the circulating erythrocyte, creating linear Hb polymers, which are the cause of the erythrocyte's sickled shape.

2. What are the variants of SCD?

In SCD, both copies of the β-globin gene produce the abnormal Hb (HbSS). Several other variants of SCD also exist and cause a spectrum of clinical presentations. HbSC disease occurs when one β subunit contains the sickle cell mutation and the other subunit contains the HbC mutation (substitution of lysine for glutamic acid); these patients suffer from fewer bacterial infections than individuals with SCD. In patients with both the sickle cell mutation and β-thalassemia, which causes decreased β-globin production, there is significant clinical heterogeneity based upon the degree of inhibition of β-globin synthesis. In patients with HbSβ^+-thalassemia, there are fewer clinical implications, and the clinical course is milder than in individuals with HbSS. In patients with HbSβ0-thalassemia, only HbS is produced, and these patients are at risk for the same set of clinical complications as patients with SCD. Patients with one normal copy of the β-globin gene and one mutant copy (HbAS; termed *sickle cell trait*) are asymptomatic carriers and do not suffer from the same set of clinical consequences as individuals with SCD.

3. What is the epidemiology of SCD?

SCD affects approximately 1 in 500 African Americans, and 1 in 12 African Americans carry the sickle cell trait. Between 70,000 and 100,000 Americans have SCD. More than half of patients with SCD now survive into the fifth decade of life, and more than 90% of children in the United States and United Kingdom survive into adulthood. The median life expectancy for patients with SCD is 42 years in males and 48 years in females.

4. What is the pathophysiology of SCD?

The clinical manifestations of SCD, anemia and repeated vasoocclusive episodes, are the result of the conformational change induced in HbS while it is in the deoxygenated state. As the erythrocyte travels through the microvasculature, oxygen is unloaded from the Hb molecule; in patients with SCD, this deoxygenated state promotes polymerization of HbS molecules. This process occurs repeatedly as the erythrocyte passes through the capillary system, and this stress causes changes to the red blood cell (RBC). These changes include decreased deformability of the RBC (making transit through the capillary system more difficult), changes to the erythrocyte cell membrane, and increased expression of cell surface markers, which promote adherence to the vascular endothelium. The increased cellular stress decreases the lifespan of a circulating erythrocyte from 120 days to between 16 and 20 days in a patient with SCD.

5. What are the typical laboratory findings?

Patients with SCD have mild to moderate anemia (hematocrit 20% to 30%), reticulocytosis (3% to 15%), a baseline leukocytosis and thrombocytosis, elevated L-lactate dehydrogenase (LDH) and unconjugated bilirubin, and decreased haptoglobin from increased RBC destruction. As patients with SCD age, their creatinine rises because of progressive renal dysfunction from microvascular infarcts. A peripheral blood smear demonstrates sickled cells, polychromasia from reticulocytes, and Howel-Jolly bodies from functional asplenia. Erythrocytes should appear normochromic and normocytic. Patients with SCD may show elevations in acute-phase reactants, such as C-reactive protein (CRP), fibrinogen, LDH, interleukin (IL)-2, and tumor necrosis factor (TNF).

Abstract

This chapter reviews the clinical manifestations of sickle cell disease (SCD), including painful episodes, acute anemia, infection, and other associated complications.

Keywords:

sickle cell disease (SCD), anemia, hematologic disorders, acute chest syndrome (ACS)

6. What are the causes of acute anemia in SCD?

Acute anemia is described as a drop in Hb of at least 2 g/dL. Splenic sequestration, aplastic anemia, and increased hemolysis are the three principle causes of acute anemia in SCD.

1. Splenic sequestration is an emergency. It occurs more commonly in infants and children who have not yet undergone splenic infarctions and fibrosis. It is characterized by splenomegaly with or without tenderness, acute anemia, thrombocytopenia, and reticulocytosis. Patients may develop hemodynamic instability because of the shift of blood volume into the spleen, and require aggressive supportive care. Transfusion is indicated for severe anemia, but care must be taken to not overtransfuse, because the sequestered erythrocytes can cause a hyperviscosity syndrome and increase the risk of vasoocclusion when the blood reenters the circulation.
2. Aplastic anemia is the arrest of erythropoiesis and is most often caused by infection. It most often manifests clinically as lethargy, fatigue, and possibly syncope or fever. The laboratory assessment demonstrates a decrease in the reticulocyte count (<1%). The most common cause is parvovirus B19, which directly invades erythrocyte progenitor cells, although many other infections, including *Streptococcus pneumoniae, Salmonella,* and Epstein-Barr virus (EBV), may also trigger an aplastic crisis. Severe anemia necessitates transfusion.
3. Increased hemolysis may play a role in a small subset of patients with SCD. This entity is less well characterized, and other etiologies should first be excluded.

7. What is an acute painful episode?

An acute painful episode (previously known as *sickle cell* or *vasoocclusive crisis*) is a paroxysmal event caused by vasoocclusion at the capillary level. It is the most common reason a patient with SCD will seek medical care. It is most often the result of vasoocclusion and ischemia within bone or bone marrow, with pain occurring most commonly in the back, chest, and extremities. In infants and young children, an acute painful episode may present as pain in the hands and/or feet, a condition termed *dactylitis.* The rate of painful episodes in adults with SCD correlates with higher mortality.

8. What are the triggers of acute painful episodes?

These episodes are caused by a variety of triggers, including infection, stress, dehydration, changes in weather, cigarette smoke, and hypoxia. There are no laboratory tests or vital signs that accurately diagnose an acute painful episode.

9. How is an acute painful episode treated?

Expedient treatment with intravenous (IV) (or subcutaneous) opioid pain medicine, usually either morphine or hydromorphone, is recommended. Meperidine should not be used because of possible neurologic side effects of repeated dosing. After 15 to 30 minutes, if pain is not adequately controlled from the first dose, give a second dose of opioid pain medicine at the same or a 25% greater dose. Adjuvant therapies with nonsteroidal antiinflammatory drugs (NSAIDs), such as ketorolac, have been shown benefit. Patients experiencing an acute painful episode should also receive IV hydration until they are clinically euvolemic. Supplemental oxygen should be provided if the oxygen saturation is below 95%. Nonpharmacologic adjuvant therapies, such as heat, may be employed. Patients needing ongoing pain control should be admitted.

10. Are patients with SCD at increased risk of infection?

Yes, patients with SCD are functionally asplenic as a result of repeated splenic infarctions. In addition to the scheduled childhood vaccinations, these patients should receive vaccinations against the encapsulated organisms (*S. pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis*) and a yearly inactivated influenza vaccination. Children coming to the ED should be queried about the use of prophylactic penicillin, commonly prescribed to protect against infection.

11. How should I manage fever in a patient with SCD?

Fever in a patient with SCD should always prompt a thorough evaluation for a source of infection. Any patient with SCD who has a temperature above 38.5°C (101.3°F) should have a complete blood count (CBC) with differential, reticulocyte count, blood cultures, urinalysis, and urine culture. Any respiratory symptoms necessitate a chest radiograph to evaluate for acute chest syndrome (ACS). Bone tenderness with or without swelling and erythema necessitates an evaluation for osteomyelitis. Focal neurologic findings require computed tomography (CT) of the head and lumbar puncture to evaluate for infection. First-line antibiotic therapy is with a third-generation cephalosporin (and a macrolide if there are respiratory symptoms). Admit patients with temperatures above 39.5°C (103.1°F).

12. What is ACS?

ACS is distinguished by a new infiltrate visible on the chest radiograph and at least one of the following: chest pain, cough, wheezing, tachypnea, fever, or hypoxia. ACS is the second leading cause of hospitalization and the most common cause of death in SCD. Causes of ACS include infection, fat embolism from marrow infarction, rib infarction causing splinting and hypoventilation, atelectasis, and pulmonary edema. These factors cause localized vasoocclusion, leading to ischemia, infarction, and inflammatory changes. The most common infectious causes of ACS include *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and respiratory viruses. Bacterial infection with *S. pneumoniae* is less common. ACS is often preceded by an acute painful episode. There are no specific laboratory values or vital signs that confirm or exclude ACS. Physical examination findings may include rales; however, a normal physical examination is common.

13. What is the treatment of ACS?

Patients with ACS require treatment with antibiotics (a third-generation cephalosporin and macrolide), supportive care with IV fluids, supplemental oxygen to maintain blood oxygen saturation (SpO_2) greater than 95%, incentive spirometry, and possibly transfusion. Simple transfusion is indicated if a patient has symptomatic anemia and a Hb that has fallen more than 1 g/dL below baseline. Exchange transfusion is indicated if a patient has refractory hypoxemia ($SpO_2 < 90\%$ despite supplemental oxygen), rapidly progressive disease (increasing respiratory distress or worsening seen on the chest radiograph), or worsening anemia despite simple transfusion. Patients with ACS always require admission.

14. Are patients with SCD at increased risk of cardiac complications?

Yes, chronic anemia leads to a high-output cardiac state, which over time can lead to dilated cardiac chambers and heart failure. Patients with SCD are prone to arrhythmias, which can be secondary to abnormal chamber size and prolonged QT intervals. Patients may also develop cardiac ischemic secondary to vasoocclusion or insufficient oxygen-carrying capacity.

15. What are the neurologic effects of SCD?

Children and adults with SCD are at increased risk of stroke; nearly 25% of patients will have a stroke by age 45. Many children will experience silent ischemic events, which can lead to neurologic and functional impairment. Children with SCD are now screened with transcranial Doppler ultrasound to detect abnormal blood flow in the brain, a marker of increased stroke risk. Early identification and treatment with preventative exchange transfusions has significantly decreased stroke incidence. Children who come to the ED with symptoms of transient ischemic attack (TIA) or stroke should be treated with exchange transfusion, whereas adults are generally treated the same as stroke patients without SCD. Other neurologic sequelae of SCD include spinal infarction, intracranial hemorrhage, seizures, and hearing loss.

16. How does SCD affect pregnancy?

SCD increases the risk of both fetal and maternal complications during pregnancy. Fetal risks are usually related to abnormality of blood flow to the placenta or placental abruption, and include fetal loss and demise, growth restriction, and preterm labor. Maternal risks include preeclampsia, eclampsia, infections, and thromboembolic events. Rates of cesarean section are higher in pregnancies complicated by SCD. Finally, pregnant women are more susceptible to painful crises and have increased rates of hospitalization for these episodes.

17. What is the role and indication for blood transfusion in SCD?

Blood transfusion dilutes the amount of abnormal Hb in the circulation, which can offset the direct effects of sickled cells in the circulation, as well as the abnormal effect that sickled cells have on vascular endothelium, inflammatory cells, and clotting. Transfusion of normal RBCs also suppresses the production of abnormal Hb. The risks of blood transfusion are not trivial and include infection, allergic reaction, and increased blood viscosity, as well as antibody formation to donor blood antigens, which may limit the ability to receive future transfusions. Some ways to reduce the risks of transfusion are to only give blood products that have undergone special screening, including the removal of any sickled cells, leukoreduction to decrease risk of infection and transfusion reactions, and detailed antibody cross-matching. Most patients with SCD have compensated chronic anemia; therefore transfusion should only be considered for specific indications, such as aplastic crisis or splenic sequestration, preparation for surgery, ACS, and stroke.

18. What are the types of transfusions that are available?

Blood can be given via simple transfusion or exchange transfusion. Simple transfusion involves giving whole units of packed RBCs. Also easily given via peripheral IV line, simple transfusions will increase the hematocrit level and therefore blood viscosity, which increases risk of vasoocclusive events. Exchange transfusion involves removing a patient's sickled RBCs and replacing them with normal donor blood cells. In addition to exposing the patient to more donor blood, exchange transfusion requires a central line and specialized equipment. The benefit is that the amount of sickled Hb is significantly reduced without increasing viscosity or causing iron overload.

19. What are the interventions for priapism in SCD?

Priapism is a common occurrence in male patients with SCD and is described as a sustained erection lasting longer than 4 hours. Stuttering priapism is also common. Recurrent episodes can lead to impotence over time. First-line treatment for priapism includes aggressive hydration and pain control. Local aspiration and injection of α-agonists, such as pseudoephedrine and epinephrine, can also be performed. Urology should be consulted in all cases. Exchange transfusion may be used if these measures are unsuccessful.

20. What are the ocular complications of SCD?

SCD patients with trauma to the eye are at risk of traumatic hyphema, a layering of blood in the anterior chamber. Sickled blood cells are unable to drain from the chamber, leading to ischemia and increased intraocular pressure. Thus hyphema is considered an ocular emergency in patients with both SCD and sickle cell trait. Failure to quickly treat hyphema in these patients can result in glaucoma, central retinal artery occlusion, and optic nerve ischemia. An ophthalmologist should be emergently consulted, and most patients should be admitted for serial examinations and ocular pressure measurements. Patients with SCD are also at an increased risk of central retinal artery occlusion, retinopathy, retinal detachment, and orbital infarction.

21. What are the orthopedic complications of SCD?

Patients with SCD are at increased risk of avascular necrosis caused by repeated sickling events in the bone marrow, with the hip being the most common site. Infarcted bone is at higher risk of infection, leading to an increased incidence of osteomyelitis, with *Salmonella* being the most common infectious agent identified. Historical features, such as fever and location of pain, can help in differentiating between osteomyelitis and vasoocclusive crisis.

KEY POINTS: TREATMENT OF ACS

1. Antibiotics
2. Fluids
3. Supplemental oxygen to maintain SpO_2 greater than 95%
4. Incentive spirometer
5. Consider transfusion
6. Admission

KEY POINTS: FEVER WORKUP IN PATIENTS WITH SCD AND NO CLEAR SOURCE

1. CBC with differential
2. Blood, urine, and throat cultures
3. Chest radiograph
4. Urinalysis
5. Lumbar puncture
6. Bony fluid aspiration if bone pain present
7. Joint aspiration if isolated joint pain present

KEY POINTS: CLINICAL PEARLS

1. Painful crises are the most common reason that patients with SCD come to the ED. Most patients with SCD are anemic and should not receive transfusions unless there is an additional indication.
2. ACS, a leading cause of death in patients with SCD, should be suspected in any patient with chest pain and any respiratory or infectious symptoms. All patients with ACS should be treated with antibiotics, and transfusion should be considered.
3. Children with SCD may have uncommon diseases for the pediatric population, such as stroke and thromboembolic events.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Brad Talley for his previous contributions to this chapter.

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QUESTIONS

1. Which of the following statements is correct regarding priapism in SCD?
   a. Exchange transfusion should be performed in all cases.
   b. Most patients do not require pain control.
   c. Injection with α-agonists and aspiration is a common treatment.
   d. *Priapism* is defined as an erection lasting longer than 6 hours.
   The correct answer is *c.*
2. Which of the following statements is a benefit of exchange transfusion?
   a. It can be given via peripheral IV.
   b. Patients are exposed to less donor blood.
   c. There is a lower risk of alloimmunization.
   d. Blood viscosity is not increased.
   The correct answer is *d.*
3. Which of the following statements is true regarding acute painful episodes in SCD?
   a. Patients should not be treated with narcotic pain medication because of the risk of addiction.
   b. Blood transfusion is part of the standard treatment.
   c. Meperidine should not be used, because it can have neurologic side effects.
   d. Laboratory tests will help determine whether a patient is having a painful crisis.
   The correct answer is *c.*

CHAPTER 44

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, therapeutic interventions against the cancer, or the resulting psychosocial issues.

2. Is this important in the ED?

Yes, cancer is the second leading cause of death in the United States. As treatments improve and patients with cancer live longer, there will be an ever-increasing number of ED patients with oncologic emergencies.

3. Name several oncologic emergencies.

See Table 44-1.

4. Which of the entities listed in Table 44-1 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, such as 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 is 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 cause, usually the small cell or squamous types. Adenocarcinoma of the breast and lymphoma are also common. Metastatic lesions from distant primary sites may also cause SVCS. Diagnosis is clinical and is verified by imaging. Treatment usually involves radiation therapy or chemotherapy. Endovascular stenting is becoming more common.
- Cardiac tamponade usually occurs secondary to metastatic disease of the pericardium. Patients with cardiac tamponade usually have a large tumor burden and a poor prognosis for 6-month survival. Malignant cardiac effusion occurs most commonly in lymphoma, and in lung and breast carcinomas. An enlarged cardiac silhouette on chest radiograph is suspicious for this entity, and pericardial fluid with wall motion abnormalities seen on bedside ultrasound confirm the diagnosis. Treatment involves pericardial drainage either with ED bedside ultrasound guidance or by operative window.
- All patients with tumors are by definition immunocompromised, so the variety of potential infections is unlimited. Immune status may be further compromised by chemotherapeutic agents, with consequent neutropenia (neutrophil count <500) a particularly high-risk feature. These patients can quickly develop septic shock, adult respiratory distress syndrome, and death; the patient who is neutropenic and febrile should be placed in isolation and treated quickly with broad-spectrum antibiotics after obtaining cultures from all potential infection sources.
- Hypercalcemia occurs in up to 30% of patients with cancer and is the most common life-threatening oncologic emergency. Common presenting signs are lethargy, constipation, and altered mental status. Treatment involves hydration with normal saline and bisphosphates, such as pamidronate or zoledronate.
- 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 has with back pain, motor loss, paresthesias, or incontinence should be considered to have SCC. Prompt diagnosis with emergent magnetic resonance imaging (MRI) can save neurologic function.

Abstract

Cancer is the second leading cause of death in the United States. Many patients with cancer have emergency conditions related to their disease. Some conditions, including superior vena cava syndrome (SVCS), spinal cord compression, hypercalcemia, cardiac tamponade, and febrile neutropenia, can be life threatening. The emergency physician must have a high level of clinical suspicion to diagnose and treat oncologic emergencies.

Keywords:

oncologic emergency, superior vena cava syndrome (SVCS), spinal cord compression, hypercalcemia, cardiac tamponade, febrile neutropenia, cancer

Table 44-1. Emergencies in Patients with Underlying Neoplastic Diseases (Partial List)

- Airway compromise
  - Head and neck mass
  - Tracheal compression
- Adrenal crisis
  - Primary tumor
  - Metastatic lesion
  - Anemia
  - Bone marrow replacement with tumor
  - Chemotherapy effects
- Bleeding
  - Primary mass
  - Low platelet count
  - Abnormal clotting factors secondary to liver metastases
- Carcinoid syndrome
- Complications of chemotherapy
  - Bone marrow suppression
  - Cardiac toxicity
  - GI toxicity
  - Pulmonary toxicity
  - Renal toxicity
- Graft versus host disease
- Hemorrhagic cystitis
  - Chemotherapy induced
  - Radiotherapy induced
- Hyperviscosity syndrome
- Infection
- With neutropenia
  - Postobstructive pneumonia
- Intestinal obstruction
- Intestinal perforation
- Pericardial effusion
- Cardiac tamponade
- Metabolic abnormalities
  - Hypercalcemia
  - Acute tumor lysis syndrome
  - Hyponatremia/SIADH
  - Hyperuricemia
  - Hypoglycemia
- Obstructive jaundice
- Obstructive uropathy
- Pain
- Complications of radiotherapy
  - Dermatitis
  - GI toxicity
- Emotional stress
  - Death and dying
  - DNR orders
  - Family issues
- Seizures
- Spinal cord compression
  - Motor/sensory loss
  - Incontinence
  - Back pain
- Superior vena cava syndrome
- Tinnitus

DNR, Do not resuscitate; *GI*, gastrointestinal; *SIADH*, syndrome of inappropriate secretion of antidiuretic hormone.

Up to 40% of patients with SCC may have normal plain radiographs. Steroids should be given in the ED, and treatment includes emergent radiation therapy or surgical decompression. The strongest predictor of neurologic outcome is the neurologic deficit at the time of initial diagnosis and treatment.

KEY POINTS: SCC

1. Negative plain films do not rule out SCC; up to 40% of patients with SCC may have normal plain radiographs.
2. Suspicion of SCC is an indication for emergent MRI.
3. Steroids and analgesics are the initial ED management while arranging for appropriate specialty 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, and 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.

9. What symptoms can be related to an underlying oncologic emergency?

Common ED complaints such as abdominal pain (colon cancer), headache (metastatic disease), 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 complains of chronic pain, unexplained weight loss, weakness, dizziness, altered mental status, or new-onset seizures, especially in adults with no seizure history.

KEY POINTS: PATIENT WHO IS NEUTROPENIC AND FEBRILE

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?

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.

11. Anything special about care plans?

It is best to discuss treatment plans, including admission, 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 home with their loved ones as much as possible.

12. 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, head and neck, anal cancers) treatments. Many patients today survive for a long time, giving them ample opportunity to develop complications that need emergency care.

13. 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, especially in the out-of-hospital arena. It is vital to communicate well with the patient so as to arrive at the very best individualized treatment plan. Beginning palliative care in the ED is often the best treatment option (see Chapter 7).

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QUESTIONS

1. Which of the following is the most common oncologic emergency?
   a. Hypercalcemia
   b. Pneumonia
   c. SVCS
   d. SCC
   The correct answer is *a*.
2. What is the first line of treatment for SCC?
   a. Radiation
   b. Surgery
   c. Steroids
   d. Bisphosphonates
   The correct answer is *c*.
3. Which of the following is the most appropriate treatment for a cancer patient with fever of 39.5°C (103.1°F) and a neutrophil count of 350?
   a. Blood cultures and empiric penicillin, with close outpatient follow-up care
   b. Blood cultures and close outpatient follow-up care
   c. Empiric cultures, and treatment not indicated
   d. Admission, appropriate cultures, and broad-spectrum antibiotics
   The correct answer is *d*.

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 K^+ (155 mEq) and the relatively low amount of K^+ 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 to 14) and normal gap (AG <12 to 14).

4. There are two types of acidosis: wide gap and normal gap. What is hyperchloremic metabolic acidosis?

Hyperchloremic acidosis is just another name for normal gap metabolic acidosis. Just think, if the AG is going to be normal and the formula for AG is $Na^+ - (Cl^- + HCO_3^-)$, if HCO_3^- goes down, then Cl^- has to rise, or, more simply, a patient becomes hyperchloremic, hence the name *hyperchloremic metabolic acidosis*.

5. Is there an easy way to remember the differential diagnosis for wide gap metabolic acidosis?

This author's favorite mnemonic is called *MUDPILES*.[1]

Methanol
Uremia
Diabetic ketoacidosis (DKA) and alcoholic ketoacidosis (AKA)
Phenformin/metformin and **p**aracetamol overdose, the English version of acetaminophen; **p**ropylene glycol
Isoniazid (INH) and **i**ron
Lactic acidosis
Ethylene glycol
Salicylates and **s**olvents

6. What are the clues to each of the entities in MUDPILES?

See Table 45-1.

Abstract

This chapter provides key teachings in the two types of metabolic acidosis; the diagnosis and treatment of hyperkalemia, hypernatremia, and hypercalcemia; and an overview of the most common intravenous (IV) fluids.

Keywords:

acidosis, acid-base, hyperkalemia, hypercalcemia, hyponatremia, anion gap (AG), MUDPILES, normal saline, lactated Ringer (LR) solution

Table 45-1. 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, polyuria, and polydipsia, glucose >500 mg/dL
AKA	Alcoholism, glucose <250 mg/dL, nausea and vomiting
Phenformin/metformin	Diabetes, medication history, recent contrast study
Paracetamol	Acetaminophen use, hepatotoxicity, fulminant hepatic failure
Propylene glycol	Large amounts of IV diazepam or lorazepam (propylene glycol used as diluent)
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; *IV*, intravenous.

7. What are the causes of narrow gap acidosis?

Memorize the mnemonic *HARDUPS.*

Hyperventilation (chronic)
Acetazolamide, **a**cids (e.g., hydrochloric), **A**ddison disease
Renal tubular acidosis
Diarrhea
Ureterosigmoidostomy
Pancreatic fistulas and drainage
Saline (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 (LR) solution, rather than half-normal saline (0.45 NS) dextrose in 5% water (D_5W), be given to someone who needs volume replacement?

Fluid goes into three different body compartments:
1. Inside blood vessels (intravascular)
2. Into cells (intracellular)
3. In between the two (interstitial)

NS and LR solution go into all three compartments, and only 25% to 33% of a solution 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 solution provide; of each liter of 0.45 NS provided, only 125 to 175 mL stays in blood vessels

(versus 250 to 333 mL for NS and LR solution). 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 solution?

Both fluids are excellent for early volume replacement.

- NS has a pH of 4.5 to 5.5, and has a Na^+ and Cl^- content of 155 mEq/L each. It is acidotic, has an osmolarity of 310, and has a little more Na^+ than serum and a lot more Cl^- than serum (155 mEq/L of Cl^- in NS versus about 100 mEq/L of Cl^- in serum). Critics of NS say that too much NS given too quickly may cause hyperchloremic metabolic acidosis.
- LR is considered to be more physiologic, in that it is much closer to serum in its content. Its Na^+ content is lower than NS at 130 mEq/L, and its Cl^- is only 109 mEq/L (versus 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 HCO_3^- once it is in the body. LR solution has 4 mEq of K^+ (none in NS) and has 3 mEq/L of calcium. Critics of LR solution do not like all the HCO_3^- in it, and believe that K^+ therapy should be individualized.

The bottom line is that neither NS nor LR solution is better; they are essentially equivalent in quantities of 2 to 3 L over 24 hours. Patients with protracted vomiting should be given NS; they develop hypochloremic metabolic alkalosis from vomiting stomach contents (rich in hydrogen and Cl^-), thus NS will correct both. Patients with severe diarrhea and resultant hyperchloremic metabolic acidosis should be given LR solution, which has the equivalent of a half an ampule of HCO_3^- per liter and does not have the high Cl^- of NS.

10. What is the most dangerous electrolyte abnormality? What are its most common causes?

Hyperkalemia is the most dangerous electrolyte abnormality.[2] It may result in sudden arrhythmogenic death because of its effect on the cells' resting membrane potential. The most common explanation for 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.

Thus the most common cause of hyperkalemia is spurious elevation. Other common causes are as follows:

- Chronic renal failure (the true number-one cause of hyperkalemia)
- Acidosis (K^+ moves out of the cell as the pH falls)
- Drugs or medications (including nonsteroidal antiinflammatory drugs, K^+-sparing diuretics, digoxin, angiotensin-converting enzyme inhibitors, and administration of intravenous [IV] potassium chloride)
- Cell death (when K^+ 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 K^+ values rise to between 5.5 and 6.5 mEq/dL. Loss of the P wave may follow as K^+ levels rise to between 6.5 and 7.5 mEq/dL. The most dangerous ECG finding (generally associated with levels of 8 mEq/dL) is widening of the QRS complex, which may merge with the abnormal T wave and create a what appears to be a sine wave on the graph, which indicates ventricular tachycardia. Always suspect hyperkalemia in patients with agonal bradycardia.

12. Summarize the best treatment for hyperkalemia.

Treatment is based on serum levels, the presence or absence of ECG changes, and underlying renal function. If the patient has life-threatening ECG changes of hyperkalemia (widening QRS complex, a sine wave–like rhythm or bradycardia/heart block), 10% calcium chloride should be given in an initial dose of 5 to 10 mL to temporarily reverse deleterious electrical effects of K^+. Most patients with hyperkalemia, however, usually just need K^+ moved intracellularly and then removed from the body, rather than a potentially dangerous calcium infusion.

13. How can K^+ 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, K^+ is

carried along. The usual dosage of glucose is 2 ampules of dextrose 50% in water ($D_{50}W$; 100 mL) and 10 U of regular insulin. Another excellent first-line method of driving K^+ into the cell is use of inhaled β-agonist bronchodilators. β-Agonists may be especially helpful in a patient who has renal failure with fluid overload, because they additionally treat the bronchospasm of pulmonary edema. HCO_3^- may be used to drive K^+ into the cell, but it is effective only in acidotic patients. Usually 1 to 2 ampules of HCO_3^- (50 mEq of HCO_3^- per ampule) are given over 1 to 10 minutes, depending on how sick or acidotic the patient is. Intravenous magnesium, which also drives K^+ into the cell, is not used because most hyperkalemic patients are also hypermagnesemic.

KEY POINTS: HYPERKALEMIA

1. Hyperkalemia is asymptomatic; you must check the ECG.
2. The ECG changes seen as K^+ rises are a tall, peaked T wave; loss of the P wave; and widening of the QRS complex.
3. Administering glucose and insulin, supplemented by an inhaled β-agonist, is the most effective method to drive K^+ into the cell and acutely lower serum K^+.
4. HCO_3^- only works to lower serum K^+ in acidotic patients.
5. Only give calcium for hyperkalemia if there is a wide QRS complex or life-threatening bradycardia.

14. After K^+'s electrical effects have been counteracted (if indicated) and K^+ has been driven intracellularly, how do I remove it from the body?

K^+ can be removed from the body by diuresis, K^+-binding resins, and hemodialysis. Diuresis with saline, supplemented by furosemide, is an excellent way to lower total body K^+. 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 (Kayexalate) is a Na^+-containing resin that exchanges its Na^+ content for the patient's K^+. Each 1 g of sodium polystyrene, which must be mixed with sorbitol, can remove about 1 mEq of K^+ from the patient's body. The best method of lowering K^+ 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 Na^+ of less than 135 mEq/dL. Most patients with mild hyponatremia (levels >125 to 130 mEq/dL) take diuretic medication or have some degree of fluid overload as a result of heart failure, renal failure, or liver disease.[3] Diuretic-induced hyponatremia is the most common cause of hyponatremia 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 Na^+ excess). Moderate to severe hyponatremia (levels <125 mEq/dL) is most commonly caused by 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?

SIADH is abnormally high levels of hormone from the posterior pituitary gland, which blocks free water excretion. Normally, when Na^+ levels fall, levels of antidiuretic hormone (ADH) also decrease, resulting in urinary loss of water (diuresis). In this syndrome, ADH is released inappropriately, and serum Na^+ 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, it's not a misprint; these questions are just a trick to wake you up after antidiuresing. K^+ causes cardiovascular, not neurologic, symptoms via its effects on the ECG (see Question 11). Na^+ 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) Na^+ should be corrected. If serum Na^+ is less than 120 to 125 mEq/L, serum Na^+ should be corrected slowly, to rise by no more than 0.5 mEq/h. This approach avoids the possible development of central pontine myelinolysis (which is also called *osmotic demyelinating syndrome* by some purists), a catastrophic neurologic illness of coma, flaccid paralysis, and usually death seen with too-rapid correction.

19. Should Na^+ levels ever be treated quickly?

There are some specific indications for raising a patient's Na^+ rapidly by infusing 3% saline. In general, patients who have serum Na^+ levels of 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 carefully to these acutely ill patients (100 mL over 10 minutes and possibly a second 100-mL bolus over the next 50 minutes). Other than these rare patients with severe, symptomatic hyponatremia, gradual 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 Na^+ by 2 and adding the amount of glucose (GLU) divided by 18, plus the blood urea nitrogen (BUN) level divided by 2.8. Normal is approximately 280 to 290 mOsm.

$$\text{Osmolarity} = 2 \times \text{Na} + \text{GLU}/18 + \text{BUN}/2.8$$

The osmolal gap is determined by using this formula, and then asking the laboratory to measure the osmolality. The difference in the laboratory'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 I use the osmolal gap to figure out whether someone has ingested methanol or ethylene glycol?

If the osmolal gap is elevated, you should measure the patient's serum ethanol level immediately in percent of milligrams and divide it by 4 for a rapid estimation of ethanol's osmolar contribution.[4] If the alcohol level is 100 mg/dL, the patient's osmolal gap should be about 30 to 35 mOsm (about 25 from alcohol, added to the normal osmolal gap, which is about 5 to 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 metabolic acidosis plus an unexplained osmolal gap often means a life-threatening overdose. Hints to methanol and ethylene glycol overdose appear in the Table 45-1.

22. What are the most common causes of hypercalcemia? How do they present?

Mild hypercalcemia is usually caused by 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 and is often secondary to a malignancy.

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 the 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.[5] Older patients and patients with impaired cardiac function must be closely monitored as they are volume resuscitated and given the saline infusion; otherwise, turn to Chapter 30, 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. Do not raise serum Na^+ by more than 0.5 mEq/L/h or by more than 10 to 12 mEq/L/day in patients with Na^+ less than 120 to 125 mEq/L.
4. Seizures, coma, and acute neurologic 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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QUESTIONS

1. Which of the following is not a cause of an AG acidosis?
   a. Renal tubular acidosis
   b. INH toxicity
   c. Uremia
   d. Aspirin overdose
   The correct answer is *a*.
2. What is the most common cause of hyponatremia in the elderly?
   a. Diuretics
   b. Heart failure
   c. Liver failure
   d. Psychogenic polydipsia
   The correct answer is *a*.
3. What is the most effective way to move K^+ intracellularly?
   a. Inhaled β-agonists
   b. HCO_3^-
   c. Insulin and glucose
   d. Kayexalate
   The correct answer is *c*.

ACID-BASE DISORDERS

Jenelle A. Holst, MD, and Jason A. Hoppe, DO

CHAPTER 46

1. Which laboratory values do I need to determine a patient's acid-base status?

You need a basic metabolic panel (BMP) and a venous blood gas (VBG) level. A VBG level will give you all the information you need for acid-base status (pH and venous partial pressure of carbon dioxide [$PvCO_2$]), and an arterial blood gas (ABG) level gives additional information about oxygenation. The $PvCO_2$ can be used instead of the arterial partial pressure of carbon dioxide ($PaCO_2$), because it is nearly the same except when the patient is in circulatory shock.

2. Which six questions do I need answered to determine a patient's acid-base status?

1. Is the pH acidemic or alkalemic (or normal)?
2. Is the primary disturbance respiratory or metabolic?
3. If a primary respiratory disturbance is present, is it acute or chronic?
4. Is there an increased anion gap (AG)?
5. Is there adequate compensation?
6. Is there more than one primary disorder (mixed disorder)?

3. Name four types of primary acid-base disorders seen, describe the typical bicarbonate (HCO_3) and partial pressure of carbon dioxide (PCO_2) patterns, and give a common example of each.

See Table 46-1.

4. How do I determine whether a primary respiratory disturbance is acute or chronic?

The rate at which the pH changes is determined by the acute or chronic nature of the change in minute ventilation. Acute changes lead to larger changes in pH, and chronic changes lead to smaller changes in pH (Table 46-2).

Table 46-1. The Primary Acid-Base Disorders

ACID-BASE DISORDER	HCO_3	PCO_2	EXAMPLE
Metabolic acidosis	↓↓	↓	Lactic acidosis in sepsis
Metabolic alkalosis	↑↑	↑	Protracted vomiting
Respiratory acidosis	↑	↑↑	Chronic obstructive pulmonary disease with CO_2 retention
Respiratory alkalosis	↓	↓↓	Hypoxic drive leading to hyperventilation

CO_2, Carbon dioxide.

Table 46-2. Chronicity of Respiratory Acid-Base Disorder

Acute respiratory acidosis	↓0.08 pH = ↑10 $PaCO_2$
Chronic respiratory acidosis (3-5 days)	↓0.03 pH = ↑10 $PaCO_2$
Acute respiratory alkalosis	↑0.08 pH = ↓10 $PaCO_2$
Chronic respiratory alkalosis (2-3 days)	↑0.03 pH = ↓10 $PaCO_2$

$PaCO_2$, Arterial partial pressure of carbon dioxide.

Abstract

This chapter provides an approach to the diagnosis and management of metabolic and respiratory acid-base disturbances in patients who come to the ED.

Keywords:

metabolic acidosis, metabolic alkalosis, respiratory acidosis, respiratory alkalosis, blood gas, mixed acid-base disorders, anion gap (AG), osmolal gap, lactic acidosis, thiamine deficiency, hyperketonemia, diabetic ketoacidosis (DKA), alcoholic ketoacidosis (AKA)

Table 46-3. Renal/Pulmonary Compensation

PRIMARY DISORDER	ANTICIPATED COMPENSATION	LIMIT OF COMPENSATION
Metabolic acidosis	$PCO_2 = (1.5 \times HCO_3) + 8 \pm 2$ PCO_2 = last 2 digits of pH	PCO_2 down to 10
Metabolic alkalosis	$\uparrow PCO_2 = 0.75 \times \Delta HCO_3$	PCO_2 up to 60 (limited by hypoxia)
Acute respiratory acidosis	$\uparrow 1\ HCO_3 = \uparrow 10\ PCO_2$	
Chronic respiratory acidosis	$\uparrow 4\ HCO_3 = \uparrow 10\ PCO_2$	
Acute respiratory alkalosis	$\downarrow 2\ HCO_3 = \downarrow 10\ PCO_2$	HCO_3 down to 18
Chronic respiratory alkalosis	$\downarrow 5\ HCO_3 = \downarrow 10\ PCO_2$	HCO_3 down to 12-15

HCO_3, Bicarbonate; *PCO_2*, partial pressure of carbon dioxide.

5. How do I determine whether compensation is adequate, and what is the physiologic limit of compensation?

Proportional changes in the PCO_2 (lungs) and HCO_3 (kidneys) are made to correct the pH. Note that compensation will never completely return the pH to normal (Table 46-3).

6. What are the three ways I can identify more than one primary acid-base disturbance (a mixed disorder)?

- If the compensation is higher or lower than anticipated
  - If the PCO_2 is too low or too high, there is additional respiratory alkalosis or acidosis, respectively.
  - If the HCO_3 is too low or high, there is additional metabolic acidosis or alkalosis, respectively.
- If the pH is normal, but the PCO_2, HCO_3, or AG is abnormal
  - If the PCO_2 and HCO_3 are high, there is respiratory acidosis and metabolic alkalosis.
  - If they are both low, there is respiratory alkalosis and metabolic acidosis.
  - If the PCO_2 and HCO_3 are normal but the AG is elevated, there is AG metabolic acidosis (AGMA) and metabolic alkalosis.
  - If the PCO_2, HCO_3, and AG are normal, there is either no acid-base disturbance or there is non-AGMA and metabolic alkalosis.
- If the AG is increased
  - The delta-delta ($\Delta AG/\Delta HCO_3$) should be calculated to determine whether there is additional non-AGMA or metabolic alkalosis.

7. What are four major etiologies of an AGMA, and which laboratory tests differentiate them?

1. Lactic acidosis; lactate level
2. Ketoacidosis; urine dipstick acetoacetate (AcAc) or serum β-hydroxybutyrate (βHB) level
3. Renal failure and uremia; blood urea nitrogen (BUN) and creatinine levels (elevated)
4. Toxicologic ingestions: aspirin level, acetaminophen level, toxic alcohol levels (ethanol, methanol, ethylene glycol, propylene glycol), cyanide level, carbon monoxide level, iron level, elevated osmolal gap

MUDPILES is also a common pneumonic.

Methanol
Uremia
Diabetic ketoacidosis (DKA)
Propylene glycol
Isoniazid
Lactic acidosis
Ethylene glycol
Salicylate

Table 46-4. The Three Types of Lactic Acidosis

LACTIC ACIDOSIS	CAUSE	EXAMPLE
Type A	Impaired tissue oxygenation and lactate overproduction through anaerobic metabolism	Shock, respiratory failure, sepsis, ischemic bowel, carbon monoxide, cyanide, severe anemia
Type B	Compromised lactate metabolism without hypoxia, usually toxicologic ingestion, often causing uncoupling of oxidative phosphorylation	Biguanides (metformin), antiretrovirals, isoniazid, salicylates, valproic acid, iron, liver disease, thiamine deficiency, catecholamine excess, malignancies, inherited metabolic deficit in lactate clearance
D-lactic acidosis	Metabolism by-product of bacteria in the gut	Accumulates in patients with short gut syndrome or gastric bypass. Note that this type of lactate is not detected by traditional laboratory assays.

8. Name three types of lactic acidosis, their causes, and examples of each.
See Table 46-4.

9. Name a vitamin deficiency associated with a fatal lactic acidosis.
Thiamine deficiency (vitamin B_1 deficiency), which is associated with neurologic deficits (e.g., Wernicke encephalopathy, Korsakoff syndrome), high-output cardiac failure (Beriberi), and lactic acidosis, has been cited as a cause of fatal metabolic acidosis. In thiamine deficiency, oxidative decarboxylation of pyruvate and α-ketoglutarate is inhibited, leading to accumulation of pyruvate and lactate production, which causes type B lactic acidosis. Thiamine deficiency should be considered in such high-risk populations as those who have a history of alcohol abuse or nutritionally deficient states.

10. List disorders that can cause a hyperketonemic state.
- DKA
- Alcoholic ketoacidosis (AKA)
- Starvation
- Isopropyl alcohol intoxication
- Hyperemesis gravidarum
- Salicylate toxicity
- Paraldehyde intoxication
- Stress hormone excess

11. In a patient with DKA who is clinically improving with appropriate therapy, why might the urine ketones increase?
There are three ketone bodies: βHB, AcAc, and acetone. βHB and AcAc are acids; acetone is not. The proportion of βHB to AcAc depends on the oxidation-reduction status of the patient. Patients experiencing DKA are often severely dehydrated, and the preponderance of ketone bodies may be in the form of βHB. The urine test by which ketones are noted is the nitroprusside reaction test (Acetest, Ketostix), which measures AcAc and acetone but is not sensitive to βHB. As fluids and insulin therapy are instituted, the amount of βHB converted to AcAc increases. The nitroprusside reaction, which initially may have been weakly positive or even negative, becomes increasingly positive. In a case where DKA is suspected but the urine nitroprusside test is negative, a serum βHB level can be tested.

12. How can glucose and albumin affect calculation of the AG?
A high glucose can cause a hypertonic hyponatremia, and a correction factor must be used to determine the calculated concentration of sodium (Na^+) (for each 100 mg/dL increase in glucose greater than 100 mg/dL, increase the Na^+ by 1.6 mEq/L); however, when calculating the AG, use the measured, not the calculated, Na^+.

A patient's expected AG is dependent upon the concentration of albumin.

$$\text{Expected AG} = (\text{Albumin}) \times 2.5$$

Therefore if the patient is hypoalbuminemic, he or she could have AGMA at a lower calculated AG (e.g., normal AG = 10 if albumin is 4 g/dL; normal AG = 5 if albumin is 2 g/dL).

13. How can a patient have a metabolic acidosis without evidence of an elevated AG?

A patient with a hyperchloremic metabolic acidosis (non-AGMA) may have no evidence of an elevated AG. This condition is caused by adding hydrogen chloride to the serum. The fall in serum HCO_3 is offset by the addition of Cl^-; consequently, there is no increased AG. Non-AGMAs are disorders caused by inappropriate hydrogen ion (H^+) retention or HCO_3 excretion, usually caused by either the kidneys (positive urine AG) or the gastrointestinal tract (negative urine AG).

14. How can I remember some of the causes of non-AGMA?

Use the mnemonic *USED CARP.*

Ureteroenterostomy
Small bowel fistula
Extra chloride (normal saline intravenous [IV] fluid)
Diarrhea
Carbonic anhydrase inhibitors
Adrenal insufficiency
Renal tubular acidosis
Pancreatic fistula

15. Which electrolyte is most commonly affected by a change in acid-base status?

Serum potassium (K^+) is affected. Because of H^+/K^+ cell membrane exchange pumps, a change of pH of 0.1 will cause an inverse change in serum K^+ of about 0.5 mEq/L (range, 0.3 to 0.8 mEq/L). If the pH is elevated by 0.1, the serum K^+ falls by about 0.5 mEq/L; if the pH is diminished by 0.1, the serum K^+ rises by about 0.5 mEq/L. For example, in DKA, although the patient's total body K^+ may be severely depleted, serum K^+ levels may be elevated because of acidosis. As the patient is treated and acidosis resolves, K^+ supplementation is indicated, because serum levels may fall precipitously as K^+ moves intracellularly.

16. What are potential causes of a metabolic acidosis in a patient with alcohol abuse?

AKA typically occurs in the undernourished alcohol abuser who stops drinking because of postbinge abdominal pain and vomiting. This patient will have an AGMA secondary to elevated ketones, which is treated by administering dextrose-containing fluids. Alcohol abusers are also at risk for thiamine deficiency, which can lead to a type B lactic acidosis. Liver insufficiency, AKA, alcohol withdrawal seizures, and acute alcohol intoxication can all lead to lactic acidosis. Ingestion or co-ingestion of other toxic alcohols should be considered as well.

17. What are the etiologies of a metabolic alkalosis?

- Patients with saline responsive states have a low urine chloride (<10) and are usually hypovolemic. Causes include gastrointestinal losses (vomiting, nasogastric tube drainage, high volume ileostomy, villous adenomas) and renal losses (prior diuretic use).
- Patients with saline resistant states have a high urine chloride (>20). Patients are either hypertensive, which is caused by hyperaldosteronism, or are hypotensive or normotensive, which is caused by current diuretic use, severe hypokalemia, exogenous alkali ingestion, or Barter or Gittelman syndromes.

18. How does a patient with metformin-associated lactic acidosis present, and what is the treatment?

These patients have a very high lactate and a low pH out of proportion to the patient's clinical state. Chronic toxicity usually arises from the patient who develops renal failure for some reason and continues to take therapeutic doses of his or her metformin. Because metformin is renally cleared, it starts to accumulate, resulting in a severe lactic acidosis. The mechanism for the lactic acidosis is unknown. The treatment is emergent dialysis for patients with renal failure and a severe acidosis.

19. How can the osmolal gap and the AG be used to differentiate toxic alcohol ingestions?

All toxic alcohols cause an elevated osmolal gap. The osmolal gap equals measured osmoles minus calculated osmoles.

$$\text{Calculated osmoles} = (2 \times \text{Na}) + (\text{Glucose}/18) + (\text{BUN}/2.8) + (\text{EtOH}/4.6)$$

An osmolal gap is considered elevated when it is greater than 10. Isopropyl alcohol is the only toxic alcohol that does not produce an elevated AG, because it metabolizes to acetone (which is not an anion). All the other toxic alcohols (ethanol, methanol, ethylene glycol, propylene glycol) lead to an elevated AG.

20. What etiologies should be considered when evaluating a patient with respiratory acidosis? How are they treated?

- Central causes: Sedatives, intracranial trauma, chronic hypoxia leading to decreased hypoxic drive, obesity hypoventilation syndrome
- Upper airway causes: Obstructive sleep apnea, laryngospasm, acute airway obstruction
- Lower airway causes: Chronic obstructive pulmonary disease, asthma, lung protective permissive hypercapnia for acute respiratory distress syndrome (ARDS)
- Muscular causes: Guillain-Barré syndrome, myasthenia gravis, amyotrophic lateral sclerosis, muscular dystrophy, severe hypophosphatemia, botulism
- Thoracic cage causes: Chest wall trauma, severe scoliosis, pectus carinatum or excavatum

Respiratory acidosis is treated by increasing the minute ventilation, which is equal to tidal volume multiplied by the respiratory rate.

21. Why do patients suffer carpopedal spasms during hyperventilation?

Hyperventilation leads to a respiratory alkalosis that increases the pH of the blood. As albumin is alkalinized, its affinity for calcium increases, thus decreasing the amount of available ionized (not bound to albumin) calcium for use by the muscles, leading to tetany.

KEY POINTS: ACID-BASE DISORDERS

1. Fully evaluating a patient's acid-base status requires a blood gas and chemistry panel.
2. Physiologic compensatory mechanisms to achieve a normal pH include the HCO_3 buffering system in the blood, minute ventilation in the lungs, and renal excretion or retention of HCO_3.
3. AG metabolic acidosis is common and life threatening; the four major causes are lactic acid, ketones, renal failure, and toxic ingestions.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. Stephen L. Adams and Morris S. Khrasch for their previous contributions to this chapter.

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QUESTIONS

1. Toxic ingestion of which of these substances causes an elevated osmolal gap but no change in the AG?
   a. Acetaminophen
   b. Methanol
   c. Isopropyl alcohol
   d. Propylene glycol
   The correct answer is *c*.
2. A 56-year-old man is somnolent after an unknown ingestion. His pH is 7.24, his PCO_2 is 60, and his HCO_3 is 26. What type of acid-base disorder does this patient have?
   a. Acute respiratory acidosis, compensated
   b. Acute respiratory acidosis, uncompensated
   c. Chronic respiratory acidosis, compensated
   d. Chronic respiratory acidosis, uncompensated
   The correct answer is *a*.
3. Which of the following is a cause of non-AGMA?
   a. Sepsis leading to end-organ hypoperfusion and lactic acidosis
   b. Large-volume resuscitation with normal saline
   c. Diabetic ketoacidosis
   d. Isoniazid overdose
   The correct answer is *b*.

CHAPTER 47

DIABETES MELLITUS

C. Ryan Keay, MD, FACEP

1. Describe the classifications of diabetes.

- Type I disease is characterized by autoimmune pancreatic β-cell destruction, which causes an absolute insulin deficiency. Patients with type I disease have little or no endogenous production of insulin and develop diabetic ketoacidosis (DKA) without exogenous supplementation of insulin. This makes insulin essential to treatment of type I diabetes.
- Type II disease is characterized by peripheral insulin resistance, with varying degrees of progressive defective productivity of insulin by pancreatic β cells. Glucose levels often respond to oral dietary modification, weight loss, exercise, and oral hypoglycemic agents; however, insulin is sometimes necessary to control glucose levels.
- Diabetes from other causes is a subset of diabetes caused by other hereditary or organ system dysfunctions leading to pancreatic disruption. These include etiologies such as cystic fibrosis, disrupting the exocrine function of the pancreas; toxicologic causes; mutations in insulin function; and drug/chemical causes, such as treatment for HIV/AIDS or transplantation medications.
- Gestational diabetes mellitus (GDM) is a state of insulin resistance and impaired insulin production diagnosed in pregnancy that is not overt diabetes and often resolves postpartum.

2. What are the diagnostic criteria for diabetes mellitus?

In 2010, the American Diabetes Association (ADA), International Diabetes Federation (IDF), and the European Association for the Study of Diabetes (EASD) instituted new diagnostic criteria (Table 47-1). Patients should now be diagnosed with diabetes based on a hemoglobin A1c (HgA1c) level greater than or equal to 6.5%, or fasting plasma glucose (FPG) level greater than or equal to 126 mg/dL (7 mmol/L), or 2-hour oral glucose tolerance test (OGTT) greater than or equal to 200 mg/dL (11.1 mmol/L) during an OGTT. Alternatively, patients with classic symptoms of hyperglycemia or hyperglycemic crisis and random glucose levels of 200 mg/dL (11.1 mmol/L) or greater also meet criteria. Two separate measurements are recommended to increase sensitivity of testing.

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 and some type II patients)

4. Describe the pertinent clinical and laboratory findings of DKA.

A patient with DKA has polyuria and polydipsia because of osmotic diuresis. This results in dehydration, drowsiness, and potentially some degree of altered mentation. Patients often develop nausea, vomiting, and abdominal pain secondary to gastric distention or stretching of the liver capsule. Other clinical signs include weight loss, tachypnea or Kussmaul respirations, and fruity breath odor from ketones. Laboratory findings include hyperglycemia, metabolic acidosis, elevated serum potassium (intracellular potassium migration to the extracellular space), hyponatremia (and pseudohyponatremia from hyperglycemia), hypochloremia, hypocalcemia, hypomagnesemia, and hypophosphatemia.

Abstract

Diabetes is a chronic illness that affects an increasingly large number of the world's population. An infallible understanding of the pathophysiology, diagnostic criteria, and emergent complications of diabetic conditions is essential for any emergency medicine physician. In addition, this chapter outlines electrolyte imbalances, hypoglycemic conditions, and up-to-date treatment algorithms.

Keywords:

diabetes, diabetic ketoacidosis (DKA), hyperosmolar hyperglycemic state (HHS), insulin-resistance, hypoglycemia

Table 47-1. Diagnostic Criteria for Diabetes

HgA1c ≥6.5%
or
FPG ≥126 mg/dL (7 mmol/L)
or
OGTT with 2-hour glucose ≥200 mg/dL (11.1 mmol/L)
or
Classic symptoms of hyperglycemia and random glucose levels ≥200 mg/dL (11.1 mmol/L)

FPG, Fasting plasma glucose; *HgA1c*, hemoglobin A1c; *OGTT*, oral glucose tolerance test.

Table 47-2. Diagnostic Criteria for Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State

	DKA			HHS
	Mild	Moderate	Severe	
Plasma glucose (mg/dL)	>250	>250	>600	>600
Arterial pH	7.25-7.30	7-7.24	<7.00	>7.30
Serum bicarbonate (mEq/L)	15 to 18	10 to <15	<10	>15
Serum ketones	Positive	Positive	Positive	Small
Urine ketones	Positive	Positive	Positive	Small
Serum Osms (mOsm/kg)	Variable	Variable	Variable	>320
Anion gap	>10	>12	>12	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

DKA, Diabetic ketoacidosis; *HHS*, hyperosmolar hyperglycemic state.

5. What causes DKA?

DKA is a state of insulin deficiency most commonly triggered by infection (30% of cases), medication errors of noncompliance (15%), new-onset diabetes (10%), other physiologic stressors (5%), and no identified cause (40%). 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 do I make the diagnosis of DKA?

- Blood glucose greater than 250 mg/dL (>13.9 mmol/L)
- Low bicarbonate (<15 mEq/L)
- Low pH (<7.3) with ketonemia and ketonuria (Table 47-2)

7. How should DKA be treated in the ED?

- Fluid resuscitation in adults: Patients often have a fluid deficit of 5 to 10 L. Normal saline (NS) should be administered by giving 15 to 20 mL/kg/h in the first hour. After this, titrate fluid resuscitation to urine output, blood pressure, heart rate, mental status, and serum electrolytes. If patients are eunatremic or hypernatremic, give 0.45% saline at 250 to 500 mL/h. In patients with hyponatremia, continue 0.9% sodium chloride (NaCl) at 250 to 500 mL/h. The goal is to replace all fluid deficits in the first 24 hours.
- Fluid resuscitation in pediatrics: In normotensive children, give 10 to 20 mL/kg/h of fluid for the first hour. Over the next 4 hours, most patients should receive two more 10-mL/kg/h boluses with electrolyte repletion (potassium acetate/potassium phosphate). Overaggressive fluid resuscitation in children can cause cerebral edema with devastating consequences.

- Insulin: Initial dosage is 0.1 kg IV bolus, followed by 0.1 U/kg/h via IV infusion. Alternatively, start a 0.14-U/kg/h infusion without a bolus, with no difference in clinical outcome between the two methods. Blood sugar level should be checked frequently, with a goal of dropping the glucose level by 50 to 75 mg/dL/h. Do not start insulin before checking a potassium level, and replete potassium to greater than 3.5 mEq/L before starting insulin. In children, do not give bolus insulin. After initial fluids, start an insulin infusion at 0.1 U/hg/h.
- Potassium replacement: Because patients have an elevated serum potassium caused by acidosis, the serum potassium will drop as it moves intracellularly with correction of the metabolic acidosis. Once the serum potassium is less than 5.5 mEq/L, adding 20 to 40 mEq in each 1 L bag of crystalloid will help correct the deficit slowly. Goal levels are between 4 and 5 mEq/L.
- Phosphate: Randomized studies showed no benefit in phosphate repletion in DKA. In addition, it may cause hypocalcemia in some patients.
- Bicarbonate: Patients with a pH of 6.9 or greater do not require bicarbonate therapy. There are no prospective randomized trials studying bicarbonate in patients with pH less than 6.9. Given the adverse effects of severe acidosis, critically ill patients with expected deterioration may get 100 mmol of sodium bicarbonate ($NaHCO_3$) in 400 mL of sterile water with 20 mEq of potassium chloride (KCl) at a rate of 200 mL/h for 2 hours until the pH is greater than 7.
- Glucose: When serum levels drop below 300 mg/dL, IV fluids should be switched to half NS with the addition of 5% dextrose. Insulin infusion is still required until serum ketones are eliminated, at which point the patient can be transitioned to subcutaneous insulin.
- Magnesium and calcium: Levels should be monitored and replaced accordingly.

8. List the potential complications of therapy for DKA in the ED.

- Hypoglycemia
- Hypokalemia (risk of dysrhythmias)
- Hypophosphatemia
- Adult respiratory distress syndrome
- Cerebral edema

9. 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 greater than 15 mEq/L, arterial pH greater than 7.3, negative serum ketones (can be mildly positive), and altered mental status (see Table 47-2).

10. How is plasma osmolarity determined?

$$\text{Osmolarity (mOsm/kg water)} = 2(\text{Serum sodium}) + (\text{Serum glucose}/18 + \text{BUN}/2.8)$$

where *BUN* is *blood urea nitrogen.*

11. What occurs pathophysiologically to cause HHS?

HHS usually occurs in older patients with type II diabetes who have significant comorbidities. The pathophysiology is similar to DKA, without the marked generation of ketones. As in DKA, elevated glucose levels result in glucosuria and osmotic diuresis, leading to profound dehydration. Why these patients are not ketotic remains controversial. There may be some available insulin in HHS, inhibiting lipolysis. Additionally, there are lower levels of catabolic hormones found in HHS patients compared with their DKA counterparts, a poorly understood pathogenesis.

12. What are the precipitants of HHS?

Patients with type II diabetes and comorbid conditions, such as chronic renal disease and heart failure, are at risk of developing HHS, especially when combined with an event leading to dehydration. Causes include infections, such as pneumonia and urinary tract infections (UTIs); stroke; intracranial hemorrhage; myocardial infarction; and pulmonary embolism. Drugs are commonly implicated, including thiazide diuretics, β-blockers, histamine-2 blockers, antipsychotics, alcohol, cocaine, and total parenteral nutrition (TPN).

13. What are the four key points in ED management of patients with HHS?

- Fluid administration: 15 to 20 mL/kg of NS should be administered in the first hour. 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, which can be achieved with administration of 0.45% saline at 250 to 500 mL/h for subsequent hydration.

- Potassium: Potassium should be repleted at 10 to 20 mEq/h in patients with normal renal function.
- Insulin: Low-dose insulin infusion protocols used in DKA are appropriate for HHS. Regular insulin with an initial IV bolus of 0.1 units/kg followed by infusion of 0.1 units/kg/h or continuous insulin infusion (without an initial bolus) at 0.14 units/kg/h is indicated.
- Glucose: Add 5% dextrose to IV fluids when levels are 300 mg/dL or less.

14. Describe hypoglycemia.

Outside of the neonatal period, hypoglycemia is a serum glucose level less than 70 mg/dL (3.9 mmol/L), although symptoms usually occur at less than 50 mg/dL (2.8 mmol/L).

15. Who develops hypoglycemia?

Patients who are taking hypoglycemic medications are at greatest risk for hypoglycemia. Sulfonylurea drugs (e.g., glipizide and glimepiride) stimulate release of insulin from pancreatic β cells and may inhibit both gluconeogenesis in the liver and lipolysis. They have long-acting metabolites, and their pharmacokinetics are affected by other medications, including antibiotics. Overdoses of sulfonylurea drugs usually require admission for monitoring of repeat hypoglycemic episodes. Other causes include accidental or intentional overdose (insulin, pentamidine, aspirin, and haloperidol), insulinomas, renal failure, sepsis, adrenal insufficiency, alcoholism, and heart failure.

16. 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 $NaHCO_3$ or hemodialysis.
- Thiazolidinediones (glitazones) increase peripheral tissue glucose use and do not cause hypoglycemia. Hepatotoxicity 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.

17. What are the presenting signs of hypoglycemia?

Three different mechanisms interact to produce symptoms. As blood glucose falls, the counter-regulatory hormones (adrenalin, glucagon) cause shakiness, diaphoresis, tachycardia, pallor, mydriasis, hunger, and nausea/vomiting. As glucose levels drop in the brain, there are neurologic manifestations that include wide range of symptoms, such as decreased level of consciousness, slurred speech, pins and needles sensation, emotional lability, lethargy, 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 alternative diagnosis.

18. Which patients with hypoglycemia require admission to the hospital?

Admit 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

19. 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 and are competent by all other measures (e.g., not intoxicated, not suicidal, no head injury), they may refuse transport. Patients who may have taken an intentional overdose of insulin or oral hypoglycemic agents must be transported. In addition, patients taking therapeutic dosages of oral hypoglycemics with repeat hypoglycemic episodes should be transported for further treatment.

20. Describe GDM.

GDM is any degree of glucose intolerance that usually develops in the second or third trimester of pregnancy and occurs when a woman's pancreatic function cannot overcome the insulin resistance created by placental antiinsulin hormones. International consensus guidelines (2010) define GDM as an FPG level greater than 92 mg/dL (5.1 mmol/L), 1-hour glucose level greater than 180 mg/dL (10 mmol/L), or a 2-hour glucose level greater than 153 mg/dL (8.5 mmol/L). Diagnosis of overt

diabetes in pregnancy is determined with an FPG level of 126 mg/dL or greater (≥7 mmol/L) or HgA1c greater than or equal to 6.5%. GDM 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.

21. What types of infections are seen more commonly in patients with diabetes 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 saprophytic 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 have 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 diabetic patients. 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.

22. What are the common manifestations of diabetic neuropathy?

Patients typically exhibit 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 better treated with duloxetine (60 mg daily), 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 diabetic patients must be aggressively treated, because they may spread rapidly and can precipitate DKA/HHS.
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.
3. Because of the risk of cerebral edema, crystalloid volume replacement for DKA in children should not exceed 20 mL/kg in the first hour.

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QUESTIONS

1. When might you consider administering serum bicarbonate in a patient with DKA?
   a. Only when the patient is having a cardiac arrest
   b. If the pH is below 7
   c. Never
   d. Only in renal failure patients with DKA
   The correct answer is *b*.
2. What and how much fluid should an adult with DKA receive in the first hour?
   a. 1 to 1.5 liters NS
   b. 3 L NS rapid infusion
   c. 30 mL/kg/h of NS
   d. 2 L of lactated Ringer solution
   The correct answer is *a*.
3. How is DKA different from HHS?
   a. HHS occurs in young, healthy diabetics.
   b. DKA patients are usually more altered.
   c. HHS patients can usually be discharged home.
   d. HHS has minimal ketones and marked hyperglycemia.
   The correct answer is *d*.

THYROID AND ADRENAL DISORDERS

David R. Saxon, MD, and Daniel H. Bessesen, MD

CHAPTER 48

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 rates of thyroid storm and myxedema coma without treatment are 80% to 100%. Rarely, eye complications from 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 (only seen with Graves disease), 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: Multiple thyroid nodules
- Hyperfunctioning nodule: Large thyroid nodule, with the rest of the gland 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 after 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 after the delivery of a child
- Drug-induced thyroiditis: Drugs, including amiodarone, lithium, cytokines, interferon. Condition is often resolved when drug is discontinued.
- Radiation-induced inflammation: Exacerbation of Graves disease that occurs 7 to 10 days after 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: May occur because the patient takes too much hormone, or the physician prescribes too much

4. What laboratory 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 a free thyroxine (T_4) level. When hyperthyroidism is caused by overproduction of

Abstract

Thyroid storm and adrenal crisis are two truly emergent endocrine disorders that rarely are seen in an ED. Emergency medicine physicians must know how to diagnose and treat these conditions. Less emergent endocrine disorders are more common presentations in an ED, and these are discussed in detail in this chapter.

Keywords:

thyroid storm, thyrotoxicosis, myxedema coma, hyperthyroidism, hypothyroidism, pheochromocytoma, adrenal insufficiency (primary and secondary), adrenal crisis Graves disease

thyroid hormone, TSH should be completely suppressed (<0.01 mIU/L). A patient with suppressed TSH and a normal T_4 level has subclinical hyperthyroidism.

5. What is apathetic thyrotoxicosis?

Apathetic thyrotoxicosis is a commonly missed presentation of hyperthyroidism seen most often in the elderly, but which may present at any age, even in children. The typical patient is 70 to 80 years of age without goiter or ophthalmologic findings. The diagnosis should be considered in elderly patients with chronic weight loss, proximal muscle weakness, depressed or "apathetic" 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 and mortality. Clinical features characteristic of thyroid storm are temperature greater than 38°C (100.4°F), altered mental status, and cardiovascular decompensation. A common clinical challenge is to determine whether one of these features is the result of the thyroid hyperfunction or some other underlying disease, such as drug or alcohol intoxication, an infectious process, or underlying cardiac disease.

7. What is the Burch-Wartofsky score?

Several scoring systems have been devised to assist in making the diagnosis of thyroid storm. The Burch-Wartofsky score is a point scale that helps assess the degree of thyrotoxicosis independent of thyroid hormone levels. The clinical and physical criteria included in this scoring system are temperature, central nervous system effect, hepatogastrointestinal dysfunction, cardiovascular dysfunction (heart rate, evidence of heart failure, and presence of arrhythmia), and the patient's history. A score greater than 45 is highly suggestive of thyroid storm (Table 48-1).

8. Which patients with hyperthyroidism should be admitted to the hospital?

Patients with suspected thyroid storm should be admitted. Because severe hyperthyroidism is a hypercoagulable state, those with atrial fibrillation should be admitted and anticoagulated to prevent atrial thrombus. Patients with heart failure should be admitted to determine the appropriate dosage of β-blockers in the outpatient setting. Tachycardia alone, even if marked, is not an indication for admission in otherwise young and healthy patients; β-blockade can safely be instituted as an outpatient.

9. What conditions are included in the differential diagnosis of thyroid storm?

A history of goiter, thyroid disease, or previous treatment with an antithyroid medication is helpful in distinguishing thyroid storm from the following other conditions:

- Toxicity caused by cocaine, amphetamines, other sympathomimetics, and anticholinergics
- Alcohol withdrawal syndromes
- Infections such as encephalitis, meningitis, and sepsis.

10. What conditions precipitate thyroid storm?

Thyroid storm is commonly precipitated by one of the following:

- Infection or serious illness
- Surgery
- Trauma
- Childbirth
- Myocardial infarction, stroke, or pulmonary embolus
- Withdrawal of antithyroid therapy
- Recent ^{131}I thyroid ablation therapy

11. How is hyperthyroidism treated in the ED?

For most patients, treatment with a β-blocker can be initiated. Although propranolol blocks the conversion of T_4 to triiodothyronine (T_3), it needs to be taken at least three times a day in the patient with hyperthyroidism because of more rapid metabolism. Metoprolol or atenolol are alternatives that are taken twice a day, with potential for better compliance. Although methimazole or propylthiouracil can be initiated, their use interferes with thyroid scanning. For this reason, it is typically best to start medication with a β-blocker and refer the patient for follow-up consultation. ED management of thyroid storm is outlined in Table 48-2.

Table 48-1. Burch-Wartofsky Thyroid Storm Diagnostic Criteria

PARAMETERS	SCORING SYSTEM*
Thermoregulatory Dysfunction	
Oral temperature (°F)	
99-99.9	5
100-100.9	10
101-101.9	15
102-102.9	20
103-103.9	25
>104	30
Cardiovascular Dysfunction	
Tachycardia (beats/min)	
90-109	5
110-119	10
120-129	15
130-139	20
>140	25
Congestive Heart Failure	
Absent	0
Mild (pedal edema)	5
Moderate (bilateral rales)	10
Severe (pulmonary edema)	15
Atrial Fibrillation	
Absent	0
Present	10
Central Nervous System Symptoms	
Absent	0
Mild agitation	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizure, coma)	30
Gastrointestinal/Hepatic Dysfunction	
Absent	0
Moderate (diarrhea, nausea, vomiting, abdominal pain)	10
Severe (unexplained jaundice)	20
Precipitating Event	
Absent	0
Present	10

Modified from Burch HB, Wartofsky L: Life-threatening thyrotoxicosis. Thyroid storm. *Endocrinol Metab Clin North Am* 22:263–277, 1993.

*Scores greater than 45 are highly suggestive of thyroid storm, 25 to 44 are suggestive of thyroid storm, and a score less than 25 indicates that thyroid storm is improbable.

KEY POINTS: THYROID STORM

1. Thyroid disease is extremely common.
2. Thyroid storm and myxedema coma are true medical emergencies.
3. Thyroid storm should be included in the differential for suspected toxic ingestions (such as methamphetamine or cocaine).

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.

Table 48-2. 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 and is typically used as the metabolism is increased with hyperthyroidism. IV esmolol is preferred with congestive heart failure. Begin with 500 μg/kg load over 1 minute, followed by 50 μg/kg/min IV. 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 given rectally)
3. Blockade of hormone release: Iodides* (at least 1 hour after step 2)
   - Lugol solution or SSKI 20-30 drops/day PO divided three or four times per day

   or
   - Ipodate (Oragrafin), 0.5-3 g/day (especially useful with thyroiditis or thyroid hormone overdose)
4. Blockade of the peripheral conversion of T_4 to T_3
   - High-dose steroids: Hydrocortisone 100 mg IV every 8 hours, or prednisone 60 mg PO daily

From Bahn RS, et al; American Thyroid Association; American Association of Clinical Endocrinologists. Hyperthyroidism and other causes of thyrotoxicosis: management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. *Endocr Pract* 17:456–520, 2011. Erratum in: *Endocr Pract* 19:384, 2013.

IV, Intravenous; *PO*, per os (orally); *PTU*, propylthiouracil; *SSKI*, supersaturated potassium iodide; T_3, triiodothyronine; T_4, thyroxine.

*Preferred medication.

13. When is treatment of Graves ophthalmopathy an emergent condition?

Patients with compression of the optic nerve or corneal ulceration require immediate ophthalmologic evaluation. Visual blurring that persists with eye closure and diminished color brightness suggests compression of the optic nerve. Surgical decompression of the orbit may be required. Severe proptosis can cause keratitis or corneal ulceration, and may present 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 to 2 mg). Corneal ulcers, with or without keratitis, require topical antibiotics.

14. What is thyrotoxic periodic paralysis?

Thyrotoxic periodic paralysis is a rare condition typically seen in Asian men who have muscle weakness and hypokalemia in the setting of hyperthyroidism (most commonly Graves disease). This disorder has been linked to genetic mutations in genes that code for specific ion channels. Appropriate treatment includes potassium replacement followed by treatment of the hyperthyroidism.

15. What are the common clinical manifestations of hypothyroidism?

- Constitutional: Fatigue, cold intolerance, weight gain, lethargy, hoarse or deep voice, slow speech, facial puffiness, 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

- Gastrointestinal: Constipation, anorexia
- Musculoskeletal: Joint swelling, myalgias, muscle weakness
- Dermatologic: Cool, dry skin and hair loss
- Gynecologic: Metromenorrhagia

16. What are the most common causes of hypothyroidism?

Primary hypothyroidism caused by thyroid gland dysfunction (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 thyroiditis (90% of cases); thyroid gland may be firm or small
- Thyroiditis: After a period of hyperthyroidism, the gland may be hypofunctioning permanently (or transiently over 1 to 2 years).
- Hypothyroidism after 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 [FSH]/luteinizing hormone [LH] deficiency [amenorrhea in women, hypogonadism in men] and may have signs of adrenocorticotropic hormone [ACTH] deficiency [signs and symptoms of cortisol deficiency.])

- Pituitary tumor
- Pituitary infarction: Sheehan syndrome (after childbirth) or pituitary apoplexy (hemorrhage into a preexisting pituitary tumor)
- Meningioma or craniopharyngioma near the hypothalamus

17. What additional features are present in severe hypothyroidism (or 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 with associated respiratory acidosis, or respiratory failure. Electrocardiogram (ECG) may show bradycardia with low voltages that may be caused by a pericardial effusion. The chest radiograph may show cardiomegaly, pleural effusions, or pulmonary edema.

18. What precipitates myxedema coma in the patient with hypothyroidism?

As with thyroid storm, myxedema coma is typically precipitated by an intercurrent illness, such as a pulmonary or renal infection, sedatives and anesthetic agents, trauma, myocardial infarction, cerebrovascular accident, or gastrointestinal hemorrhage. Prolonged cold exposure may also be a trigger. Even moderate hypothyroidism may be life threatening in patients with underlying hypoxia, hypercapnia, or congestive heart failure.

19. What is the treatment for myxedema coma?

See Table 48-3.

20. 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 adults. Most are benign adenomas and not a health threat. Because a small percentage of solitary nodules are thyroid carcinomas, referral for fine-needle aspiration biopsy is indicated. Biopsy results identify 70% of nodules as benign and 5% as malignant. For the remainder, cytologically indeterminate nodules, newer molecular tests can help stratify the risk of cancer.

21. 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. These types of nodules can be seen in 30% to 50% of the general population. Measure serum levels of TSH and free T_4, and inform the patient that the risk of cancer in incidentally discovered thyroid nodules is about 0.5% to 13%. A follow-up thyroid ultrasound can identify high-risk features, such as microcalcifications. If thyroid function tests are normal and the ultrasound is not concerning, ultrasound should be repeated in 6 to 12 months.

22. What are the adrenal emergencies that I need to worry about?

The two most serious adrenal emergencies are acute adrenal insufficiency (adrenal crisis) and pheochromocytoma crisis.

Table 48-3. Treatment for Myxedema Coma

1. Supportive care
   - Airway control, oxygen, IV access, and cardiac monitor (ABCs)
   - Hypotension: Crystalloids
   - Vasopressors as indicated (ineffective without thyroid hormone replacement).
   - Baseline thyroid function studies
   - Hypothermia: Passive rewarming (e.g., Bair Hugger warming system)
   - Perform an ACTH stimulation test, and then empirically treat with hydrocortisone (100 mg IV 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 IV, then 50 μg IV until oral medication is tolerated)
3. Identification and treatment of precipitating factors
4. Treatment of concomitant metabolic abnormalities, including hyponatremia, hypoglycemia, and hypercalcemia

From Citkowitz E: Myxedema coma or crisis treatment and management. Available at http://emedicine.medscape.com/article/123577-treatment; accessed 7-13-2015.
ABCs, airway, breathing, circulation; *ACTH*, adrenocorticotropic hormone; *IV*, intravenous; T_3, triiodothyronine; T_4, thyroxine.

Hypercortisolism can be caused by a pituitary tumor secreting adrenocorticotropic hormone (ACTH), ectopic ACTH secretion from a non-pituitary tumor, or an adrenal tumor secreting cortisol. It may present with weight gain, hypertension, amenorrhea, 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 underrecognized cause of hypertension that may present with hypokalemia, which may be particularly severe when the patient is also taking a thiazide diuretic.

23. What is the difference between primary and secondary adrenal insufficiency?

- Primary adrenal insufficiency, also referred to as Addison disease, is caused by destruction of the adrenal gland.
- Secondary adrenal insufficiency is the result of inadequate production of ACTH by the pituitary gland.

24. List the signs and symptoms of primary adrenal insufficiency.

- Fatigue and weakness
- Salt craving
- Anorexia and weight loss
- Hyperpigmentation: Marked "tanning" in palmar creases as well as mucosal membranes. Seen in primary adrenal insufficiency, this sign is caused by increased melanocyte-stimulating hormone (MSH), which is oversecreted along with ACTH.
- 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 104°F (40°C) may be seen in acute adrenal insufficiency.

25. List the causes of adrenal insufficiency.

See Table 48-4.

26. What are the most common causes of primary adrenal insufficiency?

Ninety percent of cases are the caused by tuberculosis and autoimmune destruction. Other etiologies include granulomatous diseases, uncontrolled HIV infection, metastatic cancer, and adrenal hemorrhage.

Table 48-4. 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	Infiltrative disorders of the pituitary or hypothalamus
Adrenolytic agents (metyrapone, aminoglutethimide, or mitotane)	Sarcoidosis
Etomidate	Hemochromatosis
Ketoconazole	Histiocytosis X
Infections	Metastatic cancer
Fungal or bacterial sepsis	Lymphoma
Infiltrative disorders	Infectious diseases
Sarcoidosis	Tuberculosis
Hemochromatosis	Meningitis
Myeloidosis	Fungus
Lymphoma	Isolated ACTH deficiency
Metastatic cancer	
Bilateral surgical adrenalectomy	
Hereditary	
Adrenal hypoplasia	
Congenital adrenal hyperplasia	
Adrenoleukodystrophy	
Familial glucocorticoid deficiency	

ACTH, Adrenocorticotropic hormone.

27. What is the most common cause of secondary adrenal insufficiency?

Long-term glucocorticoid therapy (e.g., prednisone, methylprednisolone, dexamethasone, or topical/inhaled steroids in patients taking highly active antiretroviral medication) is the most common cause of secondary adrenal insufficiency and is the result of suppression of the hypothalamic-pituitary-adrenal (HPA) axis.

28. 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?

Patients receiving maximal stress doses of steroids (e.g., ≥60 mg/day of prednisone) for over 1 week may have a blunted response to ACTH. This will typically resolve over a few weeks or months. If a person has been taking maximal stress doses of steroids for many months or years and then the medications are gradually tapered, they may be able to make enough cortisol for normal daily functioning. However, when ill or injured, they may exhibit signs and symptoms of adrenal insufficiency even 1 to 2 years later.

29. What are the characteristic laboratory findings of primary adrenal insufficiency?

Hyperkalemia may be present from lack of aldosterone as well as cortisol deficiency. Hyponatremia may be present and is caused by the syndrome of inappropriate secretion of antidiuretic hormone (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.

30. 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, hypoglycemia, and hyponatremia can be seen in both primary and secondary adrenal insufficiency. Patients who have adrenal insufficiency from a

suppressed HPA axis (i.e., 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.

31. 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 stress (e.g., an acute myocardial infarction, systemic infection, surgery, or trauma), and is unable to increase their circulating cortisol levels.

32. What is the most common iatrogenic cause of acute adrenal crisis?

The most common iatrogenic cause of acute adrenal crisis is rapid withdrawal of steroids in patients who have been taking long-term steroids.

33. List some clinical features of acute adrenal insufficiency.

- Vasodilation with consequent hypotension and shock
- Anorexia, nausea, and vomiting.
- Severe abdominal pain mimicking an acute abdomen
- Fever from infection or adrenal insufficiency itself
- Confusion, disorientation, and lethargy

34. How is adrenal crisis diagnosed?

Although many of the signs and symptoms are nonspecific (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. A rapid ACTH stimulation test can confirm the diagnosis. Presumptively treating without testing not only precludes a definitive diagnosis but also makes future testing difficult. A random cortisol level is often indeterminate.

35. How is the rapid ACTH stimulation test performed?

A baseline cortisol level is determined at time 0 and is followed by an intravenous (IV) 0.25-mg dose of cosyntropin (synthetic ACTH). Cortisol levels are then checked 30 and 60 minutes later.

36. 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, begin treatment using a glucocorticoid that will not cross react with the cortisol assay. A test for cortisol level can be done and then followed by IV dexamethasone (4 to 10 mg). Cosyntropin 0.25 mg is then given, and serum cortisol levels are checked 30 and 60 minutes later.

37. 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 has been initiated. IV hydrocortisone (100 mg) and crystalloid IV fluids containing dextrose is the standard approach. Perform a detailed history and examination to identify any precipitant of the adrenal insufficiency. In unstable patients, begin empiric broad-spectrum antibiotics while waiting for culture results. Mineralocorticoid replacement is usually unnecessary if the patient receives hydrocortisone, because 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 they are unresponsive to vasopressors.
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. More than 1 week of high-dose steroid use can cause adrenal suppression, making a patient more prone to adrenal crisis.

38. What should be done for the patient with chronic adrenal insufficiency who comes to the ED with a minor illness or injury?

Administer a dose of hydrocortisone or prednisone that is between the daily replacement dosage and the maximal stress dosage (and appropriate for their degree of illness). A usual daily replacement dosage of steroids for someone with adrenal insufficiency, who is otherwise healthy, would be 20 to 30 mg/day of hydrocortisone (usually split into twice-daily dosing) or 5 to 6 mg of prednisone once daily. Someone who is critically ill would typically be given 100 mg of hydrocortisone three times per day (300 mg daily) or 60 mg of prednisone per day. This increased dose should be continued for 24 to 48 hours, or until symptoms improve. If the patient is ill but does not require hospitalization, it is generally recommended that the home steroid dosage be tripled for 2 to 3 days to prevent adrenal crisis should the illness worsen. Adding a mineralocorticoid is usually not necessary. Follow-up care should be coordinated closely with the primary care physician or endocrinologist. Patients should return if nausea or vomiting prevents oral medication dosing. All patients should be counseled to have a medical identification bracelet for future episodes, should they be critically ill and not able to communicate.

39. 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 severe headache, palpitations, and sweating. These symptoms, occurring in the setting of severe hypertension, especially if symptoms are episodic, should raise suspicion for pheochromocytoma. Other symptoms include nervousness, tremor, weight loss, and hyperglycemia.

40. Which patients with hypertension should be evaluated for pheochromocytoma?

Patients at risk include those with episodic hypertension, hypertension that requires four or more medications to control, or hypertension that began before age 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) are also at risk.

41. What is unique about the treatment of hypertension in a patient with pheochromocytoma?

The most important thing to remember is to not use β-blockers as first-line treatment. 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. Institute α-blockade early with medications like phenoxybenzamine or prazosin. Labetalol or carvedilol, having both α- and β-blocking activities, are also useful.

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QUESTIONS

1. Which steroid will not interfere with the ACTH stimulation test and is the preferred initial treatment for a stable patient with suspected adrenal insufficiency?
   a. Prednisone
   b. Dexamethasone
   c. Hydrocortisone
   d. Cortisol
   The correct answer is *b*.
2. Hyperpigmentation is a clinical feature of which of the following endocrine disorders?
   a. Primary adrenal insufficiency
   b. Secondary adrenal insufficiency
   c. Thyroid storm
   d. Myxedema coma
   The correct answer is *a*.
3. An 80-year-old man has shortness of breath and is found to have atrial fibrillation with congestive heart failure. His wife states that he has been depressed lately and losing weight over the last year. Which endocrine disorder should be suspected?
   a. Graves disease
   b. Secondary adrenal insufficiency
   c. Pheochromocytoma
   d. Apathetic thyrotoxicosis
   The correct answer is *d*.

SEPSIS SYNDROMES AND TOXIC SHOCK

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.

2. What are the SIRS criteria?
A patient must meet two of the following four criteria to be diagnosed with SIRS:
1. Temperature greater than 100.4°F (38°C) or less than 95°F (35°C)
2. Heart rate greater than 90 beats per minute
3. Respiratory rate greater than 20 breaths per minute or arterial partial pressure of carbon dioxide ($PaCO_2$) less than 32 mm Hg
4. Serum white blood cell count greater than 12,000 mm^3 or less than 4000 mm^3, or 10% band forms

3. How is *sepsis* defined?
In the ED, *sepsis* is defined clinically as a syndrome that has the presence of both SIRS and presumed bacteremia.

4. What distinguishes sepsis from severe sepsis?
Severe sepsis is sepsis that is 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.

5. 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.

6. 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 of 4 mmol/L or greater
- Hematologic: Increasing prothrombin time (PT), international normalized ratio (INR), partial thromboplastin time (PTT), hemolysis and thrombocytopenia, or disseminated intravascular coagulation (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

7. 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.

8. 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.

Abstract

Sepsis is a time-critical medical emergency. The immediate recognition and aggressive therapy as outlined in this chapter have been shown to increase survival.

Keywords:

sepsis, severe sepsis, septic shock, toxic shock syndrome (TSS)

9. What is an easy way to decrease an affected tissue's increased oxygen demand from sepsis?

Administer appropriate antibiotics early. The Institute for Healthcare Improvement recommends initiation of antibiotics within 3 hours of ED presentation.

10. What are two means of increasing oxygen supply to affected tissues in a septic state?

- High-flow supplemental oxygen
- Early goal-directed therapy (EGDT)

11. What is the mortality benefit to initiating EGDT in patients with severe sepsis?

There is a 25% reduction in mortality.

12. 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:

- Central venous pressure (CVP): Goal of 8 to 12 mm H_2O
- Mean arterial pressure (MAP): Goal of greater than 65 mm Hg
- Urine output: Goal of more than 0.5 mL/kg/h
- Venous oxygenation saturation: Goal of central venous oxygen saturation ($ScvO_2$) greater than 70% or mixed venous oxygenation saturation (SvO_2) of greater than 65%

13. 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, and repeat the dose based on response. Caution needs to be used in patients with contraindications to significant volume resuscitation (e.g., patients with heart failure or renal failure).

14. 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 mm H_2O with fluid resuscitation (at least 20 to 40 mL/kg), initiate vasopressor support to increase MAP to 65 mm Hg.

15. Does one vasopressor have a proven benefit over another in the setting of severe sepsis?

Evidence suggests that norepinephrine is a better first-line vasopressor than dopamine in the setting of sepsis, resulting in a lower 28-day mortality rate. Epinephrine is associated with higher mortality in animal models, and is generally reserved for use if dopamine and norepinephrine are both failing to produce the desired result in the patient. Given its pure α-adrenergic activity, phenylephrine can be considered in patients with tachyphylaxis, dysrhythmias, or a concerning cardiac strain.

16. What are the implications of a low venous oxygenation?

This simply means there is a global tissue hypoxia. An $ScvO_2$ of less than 70% (or SvO_2 <65%) suggests that the tissue extraction of oxygen (O_2) is greater than the delivery needed to sustain the metabolic demands (i.e., poor perfusion).

17. What intervention should be initiated for low venous oxygenation?

If the venous oxygenation is low despite a CVP of at least 8 to 12 mm H_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.

18. 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.

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 (1999) demonstrated a 16% decrease

in the absolute 28-day mortality rate by implementing EGDT through the first 6 hours after a patient's arrival in 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 in septic shock?

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 glycemic control in sepsis syndromes?

There are data to demonstrate that in critically ill patients, there is a reduction in intensive care unit (ICU) mortality with appropriately controlled glucose (level between 110 and 180 mg/dL). Protocols aiming for tighter glycemic control (<110 mg/mL) has been shown to be associated with higher rates of severe hypoglycemia and mortality. It is recommended that an appropriate insulin-controlled glucose protocol be started in critically ill patients in the ED.

23. What is toxic shock syndrome (TSS)?

TSS is clinical syndrome characterized by shock and multiorgan failure. It 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. Symptoms include high fever, headache, confusion, conjunctival hyperemia, and gastrointestinal symptoms, which are accompanied by a characteristic scarlatiniform rash and severe shock.

24. Which bacteria are associated with toxic shock syndrome (TSS)?

Although originally linked to *Staphylococcus aureus,* the same toxin-mediated syndrome has been described with other bacterial infections, including community-acquired methicillin-resistant *S. aureus* (MRSA), group A streptococcus, and certain clostridial infections, each of which cause TSS-like symptoms through the production of different endotoxins.

25. 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. Current risks factors include air-containing foreign bodies (e.g., tampons, nasal packing), recent surgery, postpartum state, burns, and focal infections (e.g., cutaneous and subcutaneous lesions, mastitis, sinusitis, and wound infections).

26. Describe the pathophysiology of TSS.

Three stages have been identified:

1. Local proliferation of the toxin-producing strain of bacteria
2. Toxin production
3. 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.

27. List the criteria for defining a case of TSS caused by *S. aureus*.

- Temperature greater than 102°F (38.9°C)
- Hypotension
- Diffuse macular erythematous rash with desquamation, usually of the palms or soles, after 1 to 2 weeks after symptom onset
- 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 BUN 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: Platelet count less than 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

28. How is the diagnosis of streptococcal TSS made?

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 DIC or thrombocytopenia
- Hepatitis
- Adult respiratory distress syndrome
- Necrotizing soft-tissue infections
- Skin changes similar to those seen in *S. aureus* TSS.

29. 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 being like a sunburn. It appears early in the illness and fades in about 3 days. It may be subtle and can be missed in dark-skinned patients.

30. 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.

31. Given the previously mentioned criteria for TSS, list the differential diagnoses.

- Colorado tick fever
- Drug reactions
- Erythema multiforme
- Kawasaki disease
- Leptospirosis
- Measles
- Meningococcemia
- Rocky Mountain spotted fever
- Sepsis
- Staphylococcal scalded skin syndrome
- Stevens-Johnson syndrome
- Streptococcal scarlet fever
- Toxic epidermal necrolysis

32. Summarize the treatment for TSS.

- Supportive care with early goal-directed therapy
- Identification and removal of the source of infection (e.g., tampon, abscess, nasal packing)
- Appropriate antibiotics

33. Do antibiotics help?

Although no prospective studies have shown that antibiotics alter the severity or the course of TSS, antibiotics do reduce the recurrence rate (which can be as high as 28%). Additionally, 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.

34. 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.

35. Are there other therapies that can help control the immune response to the toxin?

IV 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 because of 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 efficacy. Steroid use in sepsis is still debated, and steroids are not routinely used in the management of TSS.

36. 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.

37. What about the asplenic patient?

Any asplenic patient who meets SIRS criteria should be presumed to have pneumococcal sepsis and should immediately be treated with the appropriate antibiotics.

KEY POINTS: SEPSIS SYNDROMES AND TOXIC SHOCK

1. Aggressively manage sepsis in all patients who come to the ED with SIRS and a presumed infectious source.
2. In patients with severe sepsis, initiate EGDT to reduce mortality by 25%.
3. Consider norepinephrine as the first-line vasopressor for treatment of sepsis with hypotension refractory to the initial fluid bolus.
4. Consider TSS in any patient with a rapidly progressive shock syndrome and diffuse erythematous rash, and ensure there is no removable infected source of endotoxin production.

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QUESTIONS

1. Which of the following is not considered to be a SIRS criteria?
   a. White blood cell count of 13,800/mm^3
   b. Temperature (94.8°F) 34.9°C
   c. Lactate 4.2 mmol/L
   d. $PaCO_2$ 30 mm Hg
   The correct answer is *c*.
2. Which is the vasopressor of choice in a hypotensive patient with severe sepsis who is not responsive to appropriate IV fluid boluses?
   a. Dobutamine
   b. Dopamine
   c. Norepinephrine
   d. Phenylephrine
   The correct answer is *c*.
3. In a febrile, hypotensive female patient with a diffuse macular rash and a retained vaginal tampon, which of the following findings would not be a criteria for making the diagnosis of TSS?
   a. BUN or serum creatinine levels twice normal
   b. Vomiting or diarrhea
   c. Platelet count less than 100,000/mm^3
   d. White blood cell count of 18,000/mm^3
   The correct answer is *d*.

CHAPTER 50

SOFT-TISSUE INFECTIONS

Jason J. Lewis, MD

1. What is the difference between cellulitis and an abscess?

Cellulitis is an acute skin and subcutaneous tissue infection characterized by pain, warmth, swelling, and erythema. An abscess is a localized collection of purulent material (pus) that usually presents as a red, painful, indurated, and fluctuant mass.

2. What are the causes of cellulitis? How does it progress?

Cellulitis is most often caused by group A *Streptococcus* and *Staphylococcus aureus*. Usually the first sign is local skin discomfort, which is followed by tenderness, erythema, and swelling. Over the course of 24 hours, the area noticeably expands. Lymphangitic "streaking" can occur from the primary area and is a very specific diagnostic sign for cellulitis. The most common areas are the lower extremities, upper extremities, and face, respectively.

3. What are the causes of abscesses? How do they progress?

Abscesses can occur in any part of the body through localized breaks in the skin. *S. aureus* is the most common infecting organism, but *Streptococcus*, gram-negative rods, and *Pseudomonas* also must be considered. They are most commonly seen on the extremities, axilla, and perirectal region. Untreated follicular infection can evolve into a cutaneous abscess. Blockage of the apocrine glands can lead to abscess formation in the axilla and groin, whereas blockage of Bartholin gland ducts can lead to abscesses in the vaginal area. Obstruction of a sebaceous gland can lead to abscess formation on the head and neck. Superficial abscesses may rupture spontaneously, but often they continue to enlarge until they are incised and drained.

4. Who is at increased risk for abscesses?

People with diabetes, inflammatory bowel disease, and other immune disorders are at greater risk. Intravenous (IV) drug abusers are at increased risk for cutaneous abscesses caused by community-acquired methicillin-resistant *S. aureus* (CA-MRSA).

5. What is pus? What does the presence of pus signify?

Pus is a mixture of cellular debris and bacteria in various stages of digestion by polymorphonuclear leukocytes. The presence of pus signifies abscess formation.

6. How do I know if pus is present?

Physical examination can indicate the presence of purulent material, or pus. A painful area of localized fluctuance and induration is indicative sign of an abscess. There may also be a small focus of purulent drainage from the abscess. Whereas formal ultrasound and computed tomography (CT) scan are not often needed to identify cutaneous abscesses, imaging will help identify suspected deep-tissue abscesses. If you are unsure from the clinical examination, it is best to use a bedside ultrasound to identify a fluid collection, and perform needle localization and/or abscess incision and drainage under ultrasound guidance. An abscess will appear as a subcutaneous, anechoic fluid collection on bedside ultrasound.

7. What are the differential diagnoses for cellulitis?

- Thrombophlebitis: A superficial clot of the vein leading to inflammation and irritation must be considered, particularly on the lower extremities
- Viral exanthems and drug-induced rashes
- Dermatitis: Often associated with pruritus and scaling
- Insect stings: Associated with less pain, pruritus edema
- Fungal infections: Particularly *Candida,* which are characteristically located in the intertriginous areas and have a moist, red appearance with satellite locations

Abstract

Soft-tissue infections, including cellulitis and abscesses, comprise a significant number of ED visits annually. The range of conditions includes simple cellulitis that requires oral antibiotics and outpatient follow-up care to necrotizing infections that require emergent surgical intervention, intravenous (IV) antibiotics, and intensive care. This chapter discusses the essential topics for appropriate ED care and management. From simple bedside incision and drainage for uncomplicated abscess, to surgical debridement and hyperbaric oxygen therapy for necrotizing fasciitis, the optimal management is outlined. Discussion of common causative bacteria, including the increasing prevalence community-acquired methicillin-resistant Staphylococcus aureus *(CA-MRSA), and the appropriate antibiotic coverage is detailed. Upon review of this chapter, you should feel confident in treating uncomplicated cellulitis and abscesses, as well as promptly recognizing and managing of serious skin and soft-tissue infections.*

Keywords:

cellulitis, abscess, soft-tissue infection, necrotizing fasciitis, incision and drainage

8. What are the differential diagnoses for abscesses?

Differential diagnoses include acne vulgaris, fungal infections, and insect bites. Noninfectious nodular lesions include cutaneous cysts, tumors or other growths, and granulomas. Recurrent or multiple abscess may signify a more complicated (e.g., hidradenitis suppurative) or systemic disease process (e.g., endocarditis).

9. What is folliculitis?

Folliculitis is irritation and inflammation of the hair follicles. It is typically the result of infection, chemical irritation, or injury to the skin. It most commonly involves the apocrine areas, but can occur in any hair-bearing region. Infecting organisms include *S. aureus* (most common), streptococci, and gram-negative rods (including *Pseudomonas*).

10. What is erysipelas?

Erysipelas is a distinctive form of cellulitis caused primarily by group A streptococci. It is characterized by a shiny red, sharply demarcated, and palpable lesion that can rapidly expand. It is typically associated with fever and an elevated white blood cell (WBC) count.

11. What is the role of wound culturing for cellulitis or abscesses?

Although abscesses are commonly caused by community-acquired MRSA, they are treated with incision and drainage and do not require culturing or antibiotics. Typically, cellulitis is caused by group A *Streptococcus* or *S. aureus*. However, culturing can be useful for patients who are immunocompromised or in those whose initial treatment fails.

12. What is the role of blood cultures in management of cellulitis?

Blood cultures are typically not warranted in immunocompetent patients with uncomplicated cellulitis. In immunocompromised hosts or those suspected cases of *Haemophilus influenzae* type B, blood cultures may be collected, because bacteremia has been reported in up to 90% of these cases. However, a retrospective study of blood culturing of complicated cellulitis in immunocompromised patients found that results changed empiric management only 2% of the time, with most alterations being narrowing of antibiotic coverage.

13. What is CA-MRSA?

CA-MRSA has become increasingly prevalent since its first recognition as a community-associated pathogen in skin and soft-tissue infections. Today, CA-MRSA causes 21% to 80% of skin infections and abscesses. It is important to note that CA-MRSA is genetically and phenotypically distinct from hospital-associated MRSA infections and has different antibiotic sensitivities and susceptibilities.

14. What are the risk factors for CA-MRSA?

Risk factors include:

- Incarceration
- Men who have sex with men
- Multiple abscesses
- IV drug use
- Chronic medical conditions, such as end-stage renal disease with dialysis
- Diabetes
- Peripheral vascular disease
- Immunosuppression

Athletes who share equipment or play on artificial turf are also at risk.

15. Should I order routine laboratory tests?

No, consider laboratory tests (e.g., WBC count, lactate) and blood cultures for immunocompromised patients or those who appear systemically ill.

16. What is the appropriate ED treatment for cellulitis?

Uncomplicated cellulitis can be treated as on an outpatient basis with a 7- to 14-day course of oral antibiotics with strict return precautions (e.g., spreading infection, persistent fevers, or increasing pain).

17. What is the appropriate ED treatment of an abscess?

1. After appropriate analgesia with or without sedation, prepare the area with betadine or chlorhexidine.

2. Using a scalpel, incise approximately two thirds the diameter of the abscess cavity to allow for instrument-assisted breaking of loculations and full drainage. (Subsequent irrigation with normal sterile saline or water is optional.)
3. Lightly pack the wound with packing tape to create a continued wicking of purulent fluid.
4. Instruct patients to attend follow-up appointment in 48 to 72 hours for a wound check and packing removal.

18. What is the treatment for suspected CA-MRSA?

Simple abscesses with suspected CA-MRSA are treated with incision and drainage and do not require antibiotics. If the patient is being treated for systemic illness or surrounding cellulitis, trimethoprim-sulfamethoxazole, clindamycin, rifampin, and doxycycline are optional oral agents. Rifampin should be used in conjunction with trimethoprim-sulfamethoxazole, because CA-MRSA can develop resistance to rifampin if it is used as a single agent. For more serious infections, start vancomycin or linezolid.

19. What are the concerning anatomic areas that may be affected by cellulitis and/or abscess formation?

- Midface: The orbital spaces are very concerning and should be treated aggressively with early intervention and IV antibiotics. An orbital abscess can cause blindness and extend into the intracranial space, forming brain abscesses, or cause other intracranial infections, and can lead to death. If concern for an orbital infection exists, a CT scan is usually required to detect abscess formation and extension into the cavernous sinus of intracranial involvement.
- Perirectal or perianal space: Differentiate between a simple buttock abscess and a true perianal abscess. Perianal abscesses originate from anal crypts and can dissect proximally into the ischiorectal space, becoming perirectal abscesses, which require surgical management.
- Bartholin gland: Abscesses arise from duct obstruction and infection from vaginal floral, causing pain in the vaginal vestibule and labia minora.
- Retropharyngeal space or sublingual tissues (e.g., Ludwig angina): An abscess here can lead to airway compromise and may require surgical intervention.
- Deep space abscess (e.g., in the neck and groin): These commonly require surgical intervention, given the proximity to neurovascular structures.

20. What are the physical examination findings that help differentiate orbital and preseptal cellulitis?

Proptosis, ophthalmoplegia, and pain with extraocular movement are indicators of orbital infection. Fever, systemic symptoms, and toxicity are variable; up to 30% of patients with orbital soft-tissue infections are afebrile.

21. What is the appropriate treatment for a Bartholin abscess?

The treatment of choice is placement of a small balloon-tipped Word catheter into the abscess cavity after incision of the medial portion of the abscess, closer to the vaginal introitus. The catheter typically stays in place for 4 to 6 weeks, with the patient having twice-daily sitz baths.

22. Who requires hospital admission?

Along with IV antibiotics, any patient who appears systemically ill or septic needs admission, as well as those whose treatment has failed or who have progressing disease. Any concern for necrotizing fasciitis or Fournier gangrene requires immediate surgical consultation with liberal IV fluid resuscitation and antibiotic therapy. Patients with infections of the hand may need admission to monitor for neurovascular compromise, whereas those with retropharyngeal/sublingual space infections should be monitored until ongoing airway patency is assured.

23. What is necrotizing fasciitis?

Necrotizing fasciitis is a rapidly progressive, polymicrobial infection of the skin and soft tissues that is limb and life threatening, rapidly progressing to vascular occlusion and tissue necrosis. Mortality rates range from 25% to 75%. The most common etiology is a mixed bacterial picture of gram-negative enteric bacilli, gram-positive *Streptococcus,* and other anaerobes. However, it can be caused by a single organism, such as group A *Streptococcus* with toxic superantigens. Although bacteria can be introduced from skin trauma, abdominal surgery, perirectal infections, cutaneous ulcers, or IV drug abuse (IVDA), the entry point often goes unidentified.

Table 50-1. The LRINEC Score

VARIABLE	SCORE
C-reactive protein level (mg/L)	
<150	0
≥150	4
Total white blood cell count (per mm^3)	
<15	0
15-25	1
>25	2
Hemoglobin level (g/dL)	
>13.5	0
11-13.5	1
<11	2
Sodium level (mmol/L)	
≥135	0
<135	2
Creatine level (μmol/L)*	
≤141	0
>141	2
Glucose level (mmol/L)*	
≤10	0
>10	1

LRINEC, Laboratory risk indicator for necrotizing fasciitis.
*To convert the values of creatinine to milligrams per deciliter, multiply by 0.01131. To convert the values of glucose to milligrams per deciliter, multiply by 18.015.

24. How does necrotizing fasciitis progress?

Bacterial exotoxins cause an acute onset of severe systemic toxicity. Early in the presentation, the skin appears erythematous and is minimally painful. As the disease progresses, there is separation of the dermal connective tissues, inflammation and necrosis, and painful rapidly expanding edema. Gas can form under the skin, manifesting as crepitus on physical examination. If not treated aggressively with early broad-spectrum IV antibiotics and surgical debridement, limb ischemia, septicemia, and death can occur.

25. How do I diagnose necrotizing fasciitis?

Suspect necrotizing fasciitis in patients with severe pain and tenderness that is out of proportion to the degree of visible cellulitis. Feel for crepitus, sometimes also appreciated on plain radiographs. Outline the area of visible infection to monitor for rapidly expanding signs of infection. CT can help evaluate the extent of the disease. Patients may appear septic, but this can occur later in the course of the disease.

26. What is the laboratory risk indicator for necrotizing fasciitis (LRINEC) score?

The LRINEC score is an objective scoring system that was retrospectively developed to help distinguish necrotizing fasciitis from other soft-tissue infections. It is calculated from six predictive factors, as seen in Table 50-1. In the initial publication, a score of greater than or equal to 6 had a positive predictive value of 92% and a negative predictive value of 96% for presence of necrotizing fasciitis. Additional studies have found increased mortality and amputation rates for a LRINEC score greater than 6. However, a retrospective case review of 52 patients with confirmed necrotizing fasciitis showed that a score of greater than 6 had a sensitivity of only 52%, highlighting the importance of clinical suspicion rather then reliance on this scoring system.

27. Who should be consulted for patients with suspected necrotizing fasciitis?

A surgeon should be consulted immediately upon suspicion of necrotizing fasciitis, a surgical emergency that must be treated early and aggressively with extensive operative debridement.

28. What antibiotics should I order if I suspect necrotizing fasciitis?

Broad-spectrum IV antibiotics need to be initiated immediately. Use vancomycin plus clindamycin plus piperacillin/tazobactam *or* vancomycin plus clindamycin plus aztreonam.

29. What other treatment is beneficial?

Hyperbaric oxygen (HBO) produces a tissue oxygenation level of 300 mm Hg, which exceeds the tissue requirement to cause bacteriostasis and arrest the release of tissue toxins. HBO treatment (in combination with early surgical intervention and IV antibiotics) may help arrest the spread of infection.

30. What is Fournier gangrene?

Fournier gangrene is a fulminant, necrotizing soft-tissue infection involving the perineal, genital, or perianal regions; the mortality rate reaches 30%. Men are most commonly affected. It is typically caused by mixed flora, and is usually localized in the genitourinary tract, lower gastrointestinal tract, or groin skin. Patients exhibit a rapidly spreading necrotizing fasciitis with significant scrotal and perineal swelling and induration, which can progress to scrotal wall necrosis and sepsis with multiorgan failure.

31. Who is at increased risk for Fournier gangrene?

Patients at risk include those with diabetes mellitus, chronic alcoholism, and immunosuppression.

32. What is the treatment for Fournier gangrene?

Like other necrotizing soft-tissue infections, Fournier gangrene is a surgical emergency that requires early and extensive surgical debridement. ED management includes early consultation with a general surgeon or urologist, IV fluid resuscitation, and broad-spectrum antibiotics.

33. Are cat and dog bites concerning?

Cat and dog bites account for approximately 1% to 2% of ED visits. Dog bites typically cause tissue maceration, whereas cat bites cause deep puncture wounds, causing bacterial inoculation of tendons, joints, and bone. *Pasteurella multocida* causes infection in 80% of cat bites, although only 5% of dog bites result in infection from *Pasteurella canis*. Alcoholic, immunocompromised, and asplenic patients should receive antibiotics, because they are at risk of overwhelming sepsis and disseminated intravascular coagulation from *Capnocytophaga canimorsus*, a gram-negative bacillus found in dog saliva.

34. Any other needed treatment for patients with cellulitis or abscess?

Tetanus prophylaxis is needed for patients who are not up-to-date on their immunizations.

KEY POINTS: SOFT-TISSUE INFECTIONS

1. Cellulitis is an infection of the soft tissues and should be treated with antibiotics.
2. Simple abscesses are treated with incision and drainage, and do not require antibiotics.
3. Necrotizing infections are a surgical emergency that require emergent operative debridement, IV fluid resuscitation, and broad-spectrum IV antibiotics.
4. All cat bites should be treated with antibiotics that cover *P. multocida.*

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. Harvey W. Meislin and Megan A. Meislin for their previous contributions to this chapter.

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QUESTIONS

1. What is the appropriate antibiotic choice for a patient with an abscess on the lower extremity without surrounding cellulitis?
   a. Cephalexin
   b. Trimethoprim-sulfamethoxazole
   c. Clindamycin
   d. No antibiotics needed
   The correct answer is *d*. The appropriate treatment for a simple abscess without surrounding cellulitis is incision and drainage. Antibiotics are not indicated.
2. Which antibiotic is an appropriate choice for a cat bite?
   a. Amoxicillin-clavulanate
   b. Cephalexin
   c. Dicloxacillin
   d. Clindamycin
   The correct answer is *a*. Amoxicillin-clavulanate is the only antibiotic on the list that treats *P. multocida*.
3. Which is the most common culprit organism in erysipelas?
   a. *S. aureus*
   b. *Streptococcus pyogenes*
   c. *Clostridium perfringens*
   d. *Enterobacteriaceae*
   The correct answer is *b*. *S. pyogenes* (group A streptococci) accounts for the majority of cases; *Staphylococcus* is a rarer causative agent.

CHAPTER 51

SEXUALLY TRANSMITTED DISEASES AND HUMAN IMMUNODEFICIENCY VIRUS INFECTION

Kerryann B. Broderick, BSN, MD

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) estimates that 20 million new STD infections occur annually in the United States, nearly half of them among persons aged 15 to 24 years.

- Chlamydia is the most prevalent STD in the United States, estimated to infect at least 3 million people annually, and a major health problem for young women because of the sequelae of infertility, ectopic pregnancy, chronic pelvic pain, and cotransmission of HIV. In 2012, more than 1.4 million cases were reported to the CDC, the first year since 1995 in which the rate of chlamydial infections among females did not increase.
- Gonorrhea incidence peaked at more than 1 million cases per year in the late 1970s. In 2012, more than 330,000 cases were reported to the CDC. The rate of gonococcal infections is highest among adolescents and young adults.
- Trichomoniasis is the most common curable STD in young, sexually active women. Worldwide, the World Health Organization (WHO) estimates nearly 250 million new cases per year, 80% of which are asymptomatic.
- Genital human papillomavirus (HPV) has a prevalence of more than 26% in women. More than 80% of American women will have contracted at least one strain of HPV by age 50. Types 16 and 18 cause almost 100% of cervical cancers and precancers, resulting in 270,000 worldwide deaths in 2012. HPV types 16 and 18 are also responsible for significant percentages of vulvar, vaginal, and anal cancers; types 6 and 11 cause warts only.
  - The CDC recommends HPV vaccination of women age 9 to 26 years and boys 11 to 12 years (up to age 21). The two current U.S. Food and Drug Administration (FDA)-approved vaccinations are Gardasil and Cervalix. Both are given as three separate injections over 6 months. Gardasil also guards against warts.
- Genital herpes occurs in one of five adolescents and adults, with many unaware of their infection. Annually, 776,000 people in the United States contract herpes.
- In 2012, 50,000 new cases of syphilis were reported, the greatest increase in males having sex with males (MSM) and bisexual males. Syphilis is more common in non-Hispanic African Americans than in other ethnic groups, with an estimated occurrence rate of greater than 5.2 times than seen in non-Hispanic whites.
- HIV prevalence continues to rise. Worldwide, approximately 35 million people are infected with HIV, and more than 26 million have died of AIDS, including 1.5 million in 2013. Approximately 1.2 million people in the United States are living with HIV, including 250,000 individuals who are unaware. In 2011, 50,000 people in the United States were newly diagnosed.

2. How should I evaluate abnormal vaginal discharge?

Take a complete sexual history.

- Ask about how many partners the patient has had in the last several months, both male and female.
- Inquire about protective barriers, such as condoms and dental dams, with each episode.

Abstract

This chapter contains a review of current sexually transmitted diseases (STDs) in the United States and globally. It includes information about prevalence and presentation, and common questions associated with STDs.

Keywords:

sexually transmitted disease (STD), HIV, genital ulcers, gonorrhea, syphilis, chlamydia, trichomonas, human papillomavirus (HPV), Jarisch-Herxheimer reaction

- Ask about previous STDs.
- Obtain a pregnancy test to guide needed treatment.

Note any discharge on the pelvic examination, and take a sample for wet preparation and gonorrhea/chlamydia testing.

- 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, as are diabetes and HIV. Treatment is single-dose oral fluconazole or any of the topical imidazoles.
- Bacterial vaginosis (not an STD) is 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 typically with metronidazole. The role of probiotics (oral or vaginal) treatment has yielded neutral results.
- *Trichomonas vaginitis* is a true STD and causes a green, frothy discharge, with an erythematous and friable "strawberry" cervix. Diagnosis is based on finding the motile trichomonads on wet preparation or in urine. Treatment is with metronidazole.

3. How do I evaluate a sexually active young man with dysuria?

Dysuria in young men is almost always the result of urethritis from an STD. The urinalysis will be positive for leukocytes, making a urinalysis unhelpful and confusing to the novice. The likely pathogens include *Neisseria gonorrhoeae, Chlamydia, Ureaplasma, Trichomonas*, and herpes simplex virus (HSV). A purulent discharge is often caused by *N. gonorrhoeae*, whereas a mucoid discharge is more likely from *Chlamydia,* which can also infect the urethra of women. Consider *Chlamydia* in a woman with dysuria and no bacteria on urinalysis.

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 with possible 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?

Unfortunately, no; cefixime is no longer recommended because of emerging resistance. Uncomplicated urethral, endocervical, or rectal gonorrheal infections can be treated adequately with dual treatment of a single intramuscular (IM) injection of ceftriaxone (250 mg), and oral azithromycin (1 g PO) or doxycycline (100 mg twice daily for 7 days). Fluoroquinolones are no longer recommended owing to high rates of fluoroquinolone-resistant *N. gonorrhoeae.*

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 *N. gonorrhoeae* or *Chlamydia* is a precursor to upper genital tract infection. It is recommended to test using nucleic acid amplification tests (NAATs), because these have the best test performances.

7. How do I evaluate a sexually active young person who has an acutely swollen, warm, painful right ankle?

This patient, with acute monarticular arthritis, should be presumed to have disseminated gonococcal infection. There are two syndromes: (1) a triad of dermatitis, tenosynovitis, and septic arthritis, and (2) a purulent arthritis without skin lesions; knees wrists and ankles are most commonly involved. Arthrocentesis should be done on the involved joint, and the fluid should be sent for Gram stain, culture for gonococcus (GC), 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. Patients with the polyarthritis triad form may yield a better result from blood cultures. A patient suspected of having disseminated gonococcal infection should be admitted initially and treated with parenteral antibiotics (intravenous [IV] or IM ceftriaxone, 1 g/day in a single daily dose for 7 days). If the patient responds in 24 to 48 hours, the dose may be changed to 250 mg IM every 24 hours. Patients should also receive 1 g of azithromycin or 7 days of doxycycline to cover potential *Chlamydia* coinfection.

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 resulting from HSV is the most common cause of genital ulcers in the United States, presenting as itching, pain, and burning with vesicles (must be present). Less commonly, patients with primary HSV infection can have myalgias, headache, and fever with associated inguinal adenopathy. Lesions heal in 2 to 3 weeks. 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. Current work is being done on vaccine development.
- 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 (1 g) or ceftriaxone (250 mg) or 3 days of ciprofloxacin (500 mg twice daily) or 1 week of erythromycin (500 mg three times daily). Erythromycin is rarely used due to possible side effects.
- Syphilis: Primary syphilis presents with a painless indurated ulcer called a *chancre*. Reverse screening treponemal testing (fluorescent treponemal antibody absorption [FTA-ABS], various enzyme immunoassays [EIAs] and chemiluminescence testing) provides early detection. However, this will be positive for life despite successful treatment. Thus if the treponemal test is positive, a follow-up examination with a nontreponemal test (Venereal Disease Research Laboratories [VDRL], rapid plasma reagin [RPR]) is needed to confirm active disease. Direct testing with dark-field microscopy must be done immediately after obtaining the sample and consulting with a pathologist. Primary, secondary, or early latent syphilis can be treated with a single IM injection of 2.4 million units of benzathine penicillin. Combination therapies such as Bicillin CR provide too low a dose to treat.

9. What is the Jarisch-Herxheimer reaction?

After initiation of treatment for syphilis (most commonly with penicillin), the patient may experience onset of fever, chills, myalgias, headache, tachycardia, increased respirations, increased neutrophil count, and mild hypotension. This occurs approximately 2 to 5 hours after initiation of treatment, with peak temperatures at approximately 7 hours, and defervescence at 12 to 24 hours. This reaction occurs in 50% of primary syphilis, 90% of secondary syphilis, and 25% of early latent syphilis. In patients with secondary syphilis, the mucocutaneous lesions may become more edematous and erythematous.

10. Proctitis is a problem primarily seen in MSM. 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 tested for gonorrhea, *Chlamydia*, and HSV. All patients should have serologic testing for syphilis. These patients should have empiric treatment for gonorrhea and chlamydial infection. 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?

- Education about STDs is the responsibility of every ED physician, because you may be the only contact the patient has with the medical system.
- Instruct patients to refer all of 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.

- 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 ED patients in an inner city, 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?

To make the diagnosis of HIV infection, the CDC recommends a multitest algorithm consisting of a positive initial HIV antibody or combination antigen/antibody test, and a subsequent positive result from a supplemental HIV test different than the initial test. AIDS is diagnosed by laboratory evidence of HIV infection and the presence of one defining illness, including recurrent bacterial infection, opportunistic infection, herpes simplex, lymphoma, and progressive multifocal leukoencephalopathy. 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.

15. Should EDs test for HIV infection?

Yes, in 2014 the CDC has released new recommendations for diagnostic HIV testing, including use of fourth-generation assays and nucleic acid testing as the sole way to confirm. Western blots will soon be supplanted by these newer tests, mostly because they are much less sensitive. The most common HIV testing approach is diagnostic testing (i.e., where physicians are able to test patients based on clinical signs or symptoms). However, some agencies, including the CDC, have advocated for performing routine opt-out rapid HIV screening. In addition to the CDC, the U.S. Preventative Services Task Force also endorses routine screening. An increasing number of EDs are now performing HIV testing, recognizing that integrating HIV testing into ED operations is possible. 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 show symptoms in the ED?

Patients may have 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 have 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 observation.

17. What tests should be done for the patient who is infected with HIV and has systemic symptoms?

Approximately 25% of new HIV diagnoses are discovered in acutely infected patients. It is important to differentiate the evaluation of an "immunocompetent" patient with HIV (i.e., CD4 >500; undetectable viral load) from someone with AIDS. For those with AIDS, 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 include HIV-related fever, systemic infections such as *Mycobacterium avium* complex, cytomegalovirus, Hodgkin disease, and non-Hodgkin lymphoma.

Many patients with HIV and fever may be managed as outpatients, although this will depend heavily on the patient's CD4 count. A CD4 count less than 200 cells/μL defines AIDS, and these patients should be hospitalized for further evaluation. Patients with high CD4 counts (e.g., >350 cells/μL) may be managed as outpatients if they appear 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 observation can be arranged. Advanced HIV is defined by CD4 cell count below 50/mm^3, and the predicted survival time for the patient is 12 to 18 months without antiretroviral therapy (ART).

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 have mild headache. ED evaluation should include a complete neurologic examination, computed tomography (CT) or magnetic resonance imaging (MRI), and lumbar puncture. Specific cerebrospinal fluid studies that may be of value, including cell count, glucose and protein levels, Gram stain, bacterial culture, viral culture, fungal culture, *Toxoplasma* and cryptococcal antigen tests, and coccidioidomycosis titers. The most common causes of neurologic symptoms include *Cryptococcus neoformans*, *Toxoplasma gondii*, HIV-associated dementia, cytomegalovirus (CMV) encephalitis, progressive multifocal encephalopathy, and CNS lymphoma.

20. What is HIV encephalopathy?

HIV-associated dementia (HAD) is an organic brain syndrome manifested by decline in attention, cognitive reasoning, speech, motor function, and motivation. HAD is increasingly uncommon with ART treatment, but affects 33% to 60% of untreated patients. Although other causes must be ruled out, HAD may be the presenting sign of overt AIDS in 25% of patients.

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. Compared with the general population, patients with HIV have a 10- to 25-times increased risk of developing bacterial pneumonia (usually *Streptococcus*); recurrent bacterial pneumonia is an AIDS-defining illness. Pneumococcal septicemia is 100 times more common in patients infected with HIV. *Pneumocystis jiroveci* pneumonia (PJP; previously known as *Pneumocystis carinii* pneumonia or *PCP*), is now uncommon with ART therapy and PJP prophylaxis but still occurs in untreated patients. It typically presents with dyspnea, dyspnea with exertion, nonproductive cough, fever, and weight loss. Rapid institution of therapy with IV trimethoprim-sulfamethoxazole (TMP-SMX) and oral steroids (for patients with arterial partial pressure of oxygen [PaO_2] breathing room air ≤70 mm Hg or alveolar-arterial oxygen gradient ≥35 mm Hg) may prevent excessive morbidity and mortality. Other causes include *Mycobacterium tuberculosis* pneumonia, *Histoplasma capsulatum*, other traditional community-acquired pneumonia organisms, and neoplasm.

22. How should GI complaints be managed?

Approximately 50% of AIDS patients have GI complaints at some time during their illness. Esophageal complaints are common and may be most commonly caused by *Candida* esophagitis, CMV, or herpes simplex esophagitis. Patients with esophagitis should receive a 2-week empiric course of oral antifungal agents, followed by endoscopy if the condition is 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 patients with AIDS. 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. CMV and bacterial infections *(Clostridia, Salmonella, Shigella, Campylobacter)* are the most common causes and are associated with prolonged watery diarrhea. Other common infectious agents include *Candida*, Kaposi sarcoma, *M. avium* complex, HSV, *Giardia,* and *Cryptosporidium*. Management should be directed at repletion of fluid and electrolytes, cultures, possible endoscopy, and appropriate antimicrobial coverage.

23. What are the common cutaneous presentations of AIDS, and how are they treated?

Kaposi 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.

Other dermatologic conditions that occur with increased incidence in patients with HIV include drug reactions to ART (especially TMP-SMX), seborrheic dermatitis, psoriasis, atopic dermatitis, and alopecia. Consult an infectious disease specialist and a dermatologist, and admit patients with any disseminated cutaneous infection requiring IV antibiotics or antiviral agents.

24. Describe ophthalmologic emergencies that occur in patients with AIDS.

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 25% of untreated patients with AIDS and 12% of patients treated with ART. 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 patients with HIV receive tetanus and other immunizations?

According to the U.S. Public Health Service Immunizations Practices Advisory Committee, routine immunization recommendations for diphtheria, tetanus, and pertussis (DTP); 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?

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. TMP-SMX, sulfadiazine, and amino penicillins have the highest incidence of adverse cutaneous drug reactions in HIV patients. Severe cutaneous reactions are most commonly a reaction to sulfa-based drugs. An infectious disease specialist should be consulted before discontinuing any drug, because the benefits may outweigh the side effects.

27. How can health care providers protect themselves from acquiring HIV?

With the use of universal precautions, the risk of acquiring HIV infection by occupational exposure is extremely low.

However, because HIV infection is often undiagnosed, the use of universal precautions is imperative and should be performed in all patients, including the appropriate use of gown, gloves, mask, and goggles for procedures. The Needlestick Safety and Prevention Act of 2000 mandates that safety-engineered devices be used whenever possible, and that institutions maintain exposure control plans.

28. What constitutes high-risk exposure to HIV?

- Nonoccupational exposures
  - 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
- 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 less than 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 after 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 tenofovir-emtricitabine plus raltegravir). 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 ART?

ART typically includes two nucleotide reverse transcriptase inhibitors (NRTIs), such as tenofovir-emtricitabine, a non-NRTI (NNRTI), and/or boosted protease inhibitors, such as lopinavir or ritonavir. Adverse reactions to ART are common and may include bone marrow suppression, cutaneous reactions, GI distress, jaundice, nephrolithiasis, abnormal lipid profiles, and neuropathy. ART is recommended for all patients infected with HIV, regardless of CD4 count. Untreated HIV infection is a risk factor for coronary artery disease (CAD), renal disease, neurocognitive deficits, liver disease, and non–HIV-associated malignancy.

KEY POINTS: STDS

1. STDs affect 20 million people per year in the United States, 50% of whom are ages 15 to 24 years.
2. Annual targeted screening is recommended for all STDs.
3. Chlamydial infection is the most prevalent STD in the United States, and it is often asymptomatic in women.
4. Every patient with HIV should be treated with ART to prevent comorbidities.
5. Bacterial infection is the most common pulmonary infection in patients with HIV, but at a rate 10 to 25 times that of the general population.

WEBSITES

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QUESTIONS

1. Which newly diagnosed patients with HIV should start receiving ART therapy?
   a. Only those with CD4 counts less than 200
   b. Only those with CD4 counts less than 350
   c. Those with confirmation of HIV
   d. Those with an AIDS defining illness
   The correct answer is *c*.
2. Regarding STDs, which of the following statements is correct?
   a. Chlamydia is the most prevalent STD in the United States.
   b. HIV incidence has leveled off.
   c. Oral therapy for GC is effective.
   d. Syphilis is more common in non-Hispanic whites.
   The correct answer is *a*.
3. Which of the following immunizations are not recommended in an HIV-infected patient?
   a. DTP
   b. MMR
   c. Smallpox
   d. Td
   The correct answer is *c*.

CHAPTER 52

TETANUS, BOTULISM, AND FOOD POISONING

Dazhe James Cao, MD

TETANUS

1. What is the causative agent of tetanus, and what is its mechanism of action?

Clostridium tetani is an obligate anaerobic bacteria that produces 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 membranes and irreversibly disables inhibitory neurotransmitter release (γ-aminobutyric acid [GABA] and glycine) in both the autonomic and somatic nervous systems. Although *C. tetani* is heat- and oxygen-sensitive, spores are extremely resilient and able to survive household disinfectants, extremes in temperatures and humidity, and in an autoclave for several minutes.

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 in peripheral injuries. Spasm, rigidity, and pain are usually limited to the injured area.
- Cephalic tetanus occurs after a head wound, presents as cranial nerve paralysis (most commonly lower motor neuron weakness of cranial nerve VII), and often proceeds to generalized tetanus.
- Tetanus neonatorum (neonatal tetanus), although rare in developed countries, is the most common form of tetanus worldwide because of lack of immunizations and poor umbilical hygiene.

3. How is tetanus contracted?

Tetanus generally originates from a deep, usually grossly contaminated wound (soil, manure, or metal), that facilitates anaerobic bacterial growth. Other sources include burns, ulcers, snakebites, middle ear infections, tattooing, piercings, septic abortions, childbirth, surgery, and intramuscular injections. A prior episode of tetanus 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. Irritability and poor feeding progress to generalized spasms, pneumonia, and pulmonary or CNS hemorrhage. Toxin load is high, and the mortality rate ranges from 40% to 95%.

5. What is the presentation of generalized tetanus?

Initial symptoms proceed head to toe, including trismus (from masseter and parapharyngeal spasm), dysphagia, neck muscle spasm/pain, *risus sardonicus* (the sardonic smile of tetanus from facial muscle spasm), and opisthotonos (from paraspinous and abdominal wall muscle spasm). 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). Autonomic instability is the most common cause of death from tetanus in developed countries.

6. What is the time course of tetanus?

The incubation period after exposure averages from 8 to 11 days (range, 3 to 21 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.

Abstract

Clostridium tetani *and* Clostridium botulinum *are obligate anaerobic bacteria that produce potent toxins with diametrically opposite symptoms. Tetanus disables the release of inhibitory neurotransmitter and thereby causes unopposed muscle contraction and spasm. Botulism prevents the release of acetylcholine, a neurotransmitter required for muscle contraction, and thereby causes flaccid paralysis. Food poisoning is caused by a wide variety of xenobiotics, including viruses, bacteria, parasites, mushrooms, and secreted exotoxins. Traveler's diarrhea is typically a profuse watery diarrhea that begins within 2 weeks of travel to high-risk countries. In adults, antibiotics may be beneficial. In children, bloody diarrhea may herald enterohemorrhagic* Escherichia coli, *and therefore antibiotics should be avoided. Ingestion of contaminated fish may lead to scombroid, ciguatera, and tetrodotoxin poisoning. Algal toxins may lead to amnesic, diarrhetic, paralytic, and neurotoxic shellfish poisoning (NSP).*

Keywords:

tetanus, Clostridium tetani, *botulism,* Clostridium botulinum *food poisoning, scombroid, ciguatera, shellfish poisoning,* Amanita phalloides

7. How do I treat generalized tetanus in the ED?

Initial management requires close attention to the patient's airway, including potential endotracheal intubation, and liberally administered benzodiazepine for sedation. To bind free toxins, give human tetanus immunoglobulin (500 IU) intramuscularly at the site of the wound and before any needed surgical debridement. Administer metronidazole (500 mg) orally or intravenously every 6 hours; doxycycline, macrolides, clindamycin, and cephalosporins are alternatives.

8. 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.

9. How do I vaccinate someone against tetanus?

Previously unimmunized patients require a three-dose primary series, starting on day of presentation, with the second dose given more than 4 weeks after the first and the third dose given 6 to 12 months later. Previously immunized patients should receive a booster if more than 10 years from most recent vaccination have passed, after a clean wound, or if more than 5 years from most recent vaccination have passed and they have a tetanus-prone wound (devitalized tissue, gross contamination, and/or wounds from crush injuries). The tetanus and diphtheria (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 the tetanus vaccine to those with grossly contaminated wounds who lack a primary vaccination series (Table 52-1).

10. What are the side effects of tetanus vaccine?

Side effects are generally limited to local reactions, such as 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 the tetanus vaccine safe for pregnant and immunocompromised patients?

The tetanus vaccine is a toxoid (inactivated toxin) that is safe and effective in pregnancy and can help prevent neonatal tetanus. All pregnant women should receive the tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis vaccine (Tdap) immunization during each pregnancy, irrespective of prior vaccination status. The tetanus vaccine is also safe for administration in immunocompromised patients.

Table 52-1. Postexposure Tetanus Prophylaxis

	Clean, Minor Wounds		*All Other Wounds**	
VACCINATION HISTORY	**Td†**	**TIG**	**Td†**	**TIG**
Unknown number or <3 doses	Yes	No	Yes	Yes
≥3 doses ≥10 yr since most recent dose	Yes	No	Yes	No
5-9 yr since most recent dose	No	No	Yes	No
<5 yr since most recent dose	No	No	No	No

From: Lambo JA, Anokye EA: Prognostic factors for mortality in neonatal tetanus: a systematic review and meta-analysis. *Int J Infect Dis* 17:e1100–1110, 2013.

Td, Tetanus and diphtheria; *TIG*, tetanus-specific immune globulin.

*Wounds greater than 1 cm in depth, incurred more than 6 hours earlier, or with stellate or avulsion configuration; crush injuries or burn injuries; devitalized tissue; and wounds contaminated with dirt, feces, or saliva.

†For children younger than 7 years, vaccination with diphtheria, tetanus toxoid, and pertussis (DTaP or DTP, or diphtheria and tetanus toxoid [DT] alone, if pertussis vaccine is contraindicated) is preferred to vaccination with tetanus toxoid alone. For children older than 7 years, vaccination with Td is preferred to vaccination with tetanus toxoid alone. For adolescents and adults up to age 64 years, vaccination with tetanus toxoid given as Tdap is preferred if the patient has not previously been vaccinated with Tdap.

KEY POINTS: TETANUS

1. Initial management of generalized tetanus is to monitor the airway and provide adequate sedation to control spasms and pain.
2. Human tetanus immunoglobulin, surgical debridement, and metronidazole are initial treatments of choice.
3. In developed countries with ICU capabilities, autonomic instability is the most common cause of mortality.

BOTULISM

12. What is the causative agent of botulism? How does it cause disease?

Botulism is caused by toxins produced by an obligate anaerobic bacteria, *Clostridium botulinum*. Botulinum toxins are taken up preferentially by skeletal and autonomic peripheral cholinergic nerve terminals, where the toxins prevent the release of acetylcholine, producing a life-threatening, paralytic illness. By weight, botulinum toxin is the most potent toxin known.

13. What are the five types of botulism?

- Food-borne botulism (10% to 20% of U.S. cases, 17 to 43 cases per year) results from the food-borne ingestion of preformed toxin. Undercooked home-canned vegetables, fruits, and fish products are the most common sources; cases occur in isolation or in small clusters.
- Infant botulism (approximately 70% to 80% of U.S. cases) is caused by the ingestion of *C. botulinum* spores, which proliferate in the gastrointestinal (GI) tract in the absence of competitive flora in infants. It is usually seen between the ages of 6 days and 1 year, with a median age of 10 weeks. The source in most cases is unknown but may be related to contaminated soil; contaminated raw honey ingestion accounts for 20% of cases.
- Wound botulism (approximately 5% to 20% of U.S. cases) is caused by the contamination of traumatic wounds with *C. botulinum* spores. In the United States, it is seen almost exclusively in "black-tar" heroin users who partake in "skin popping" (injection into subcutaneous tissue).
- Botulism of undetermined etiology describes cases in which the patient's stool contains *C. botulinum* with 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 have moderate to severe clinical weakness. Focal neurologic deficits can be seen from craniofacial migration of injected toxin.

14. What are the differential diagnoses of botulism?

- Myasthenia gravis
- Lambert-Eaton myasthenic syndrome
- Guillain-Barré syndrome (especially Miller-Fischer variant)
- Tick paralysis
- Diphtheritic neuropathy
- Stroke syndromes
- Magnesium intoxication
- Organophosphate poisoning
- Tetrodotoxin
- Paralytic shellfish poisoning (PSP)

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. In more severe cases, infants can have upper airway obstruction and respiratory failure. 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 and abnormal cerebrospinal fluid analysis are uncommon.

16. How does an adult patient with food-borne botulism present?

- Early symptoms are nonspecific, usually begin 12 to 36 hours after ingestion (range, 6 hours to 8 days), and include nausea, vomiting, weakness, malaise, and dizziness.
- Next, prominent anticholinergic symptoms ensue, such as extreme dry mouth, decreased lacrimation, constipation, and urinary retention.
- Then, symmetric cranial nerve palsies follow (up to 3 days after anticholinergic symptoms), including ptosis, diplopia, blurred vision, photophobia, dysphonia, and dysphagia.
- Finally, a descending symmetric flaccid paralysis of the voluntary muscles may follow and lead to respiratory failure.

17. How is botulism diagnosed?

The clinical diagnosis is based on history and examination, with treatment initiated based on clinical suspicion. The most sensitive means of confirmatory diagnosis is via mouse bioassay. Anaerobic cultures of serum, stool, and/or implicated food products may be used. Testing should be performed under the coordination of the state health departments and the Centers for Disease Control and Prevention (CDC).

18. What is the treatment of food-borne botulism?

Treatment is mostly supportive, including early elective intubation of patients at risk for respiratory failure. Heptavalent equine-derived antitoxin is also recommended within 24 hours to arrest the progression of and decrease the duration of paralysis. Hypersensitivity reactions, including anaphylaxis, are less common.

19. What is treatment for infant botulism?

Treatment of infant botulism includes supportive care, including intubation and mechanical ventilation for respiratory failure. Human botulinum immune globulin (BabyBIG) reduces duration of hospitalization, mechanical ventilation, and tube feedings. The California Department of Health Services Infant Botulism Treatment and Prevention Program should be contacted for assistance in management and delivery of BabyBIG (510-540-2646, www.infantbotulism.org). Equine-derived antitoxin is not recommended for infant botulism.

20. 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.

21. Are antibiotics indicated in wound botulism?

In addition to surgical debridement, antibiotics (penicillin G or metronidazole) may be of use in wound botulism, but benefit is unproven.

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 botulinum immune globulin (BabyBIG) is available for infant botulism.

FOOD POISONING

22. Name the causes of food poisoning.

- Viruses (e.g., *Rotavirus, Norovirus, Sapovirus, Enterovirus,* or hepatitis)
- Direct bacterial invasion or endotoxins (e.g., *Escherichia coli, Vibrio* species, *Campylobacter* species, *Salmonella* species, *Yersinia enterocolitica,* or *Listeria monocytogenes*)
- Parasites (e.g., *Giardia lamblia*, *Cryptosporidium* species, or *Entamoeba histolytica*)
- Mushrooms (e.g., *Amanita phalloides*, *Chlorophyllum* species)
- Secreted exotoxins (e.g., *Staphylococcus aureus, Shigella* species, *Bacillus cereus, Clostridium perfringens,* tetrodotoxin or shellfish-associated algal toxins)

23. What is the time course and geographic incidence of traveler's diarrhea?

Onset is 4 to 7 days after arrival, with 90% beginning within 2 weeks. Although about 10% of patients have more than a week of symptoms, traveler's diarrhea usually lasts 3 to 5 days; 2% of patients have more than 1 month of symptoms. High-risk destinations include Latin America, Africa, the Middle East, and Southeast Asia (20% to 90% prevalence), whereas southern Europe, China, Russia, and the Caribbean are lower risk (8% to 20% prevalence). Overall, approximately 20% to 60% of travelers will be affected, and of those, 3% to 30% of individuals will have symptoms of dysentery (e.g., bloody stool, fever).

24. What are some of the more serious complications of traveler's diarrhea? What are the causative agents?

- Postinfectious inflammatory syndromes (e.g., Reiter 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 (O157:H7)*
- Amebic hepatitis and amebic abscesses: *E. histolytica*
- Bacteremia leading to endocarditis, aortitis, septic arthritis, osteomyelitis: *Salmonella*

25. Should antibiotics be used for infectious diarrhea?

Although controversial, the use of antibiotics in acute diarrhea has been shown to reduce the duration of illness 1 to 2 days in selected patient populations. Empiric antibiotics should be considered if the patient is febrile and has signs of invasive disease or if symptoms are severe or require hospitalization. A single oral dose of ciprofloxacin 2 g or 3-day regimen of ciprofloxacin 500 mg orally (per os, PO) twice daily is recommended for severe cases. Azithromycin 1 g (5 to 10 mg/kg) PO as a single dose can be considered in pregnant women and children, for whom fluoroquinolones are contraindicated. In areas where amebiasis and giardiasis are prevalent, consider metronidazole 500 to 750 mg PO three times daily for 10 days for amebiasis or 250 mg PO three times daily for 5 days for giardiasis (Fig. 52-1).

26. In what patient population with infectious diarrhea should antibiotics be avoided?

Antibiotic therapy is not generally recommended in children with bloody diarrhea. Antibiotic therapy in enterohemorrhagic *E. coli* (O157:H7) infections can increase lysis of organisms with release of endotoxin and Shiga-like toxin, increasing the risk of HUS.

27. Which diarrhea-producing agent is associated with febrile seizures in children?

Shigella, in young children, can cause 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.

28. What is scombroid poisoning, and how is it treated?

Scombroid fish poisoning is caused by ingestion of improperly refrigerated fish, with bacterial conversion of naturally occurring histidine to histamine, which persists despite cooking. Fish with high levels of the amino acid histidine, including fish from the scombroid family (e.g., tuna, albacore, bonito, mackerel, and skipjack), mahimahi, and bluefish have been implicated. Affected fish typically retain normal appearance and odor but may taste peppery. Not a true fish allergy, poisoning causes histaminergic symptoms minutes to hours after ingestion, typically an urticarial rash of the face, neck, and upper chest. Other symptoms include:

- Flushing
- Nausea
- Vomiting
- Diarrhea
- Headache
- Metallic taste in the mouth
- Palpitations
- Abdominal cramping
- Dizziness
- Dry mouth
- Conjunctival injection

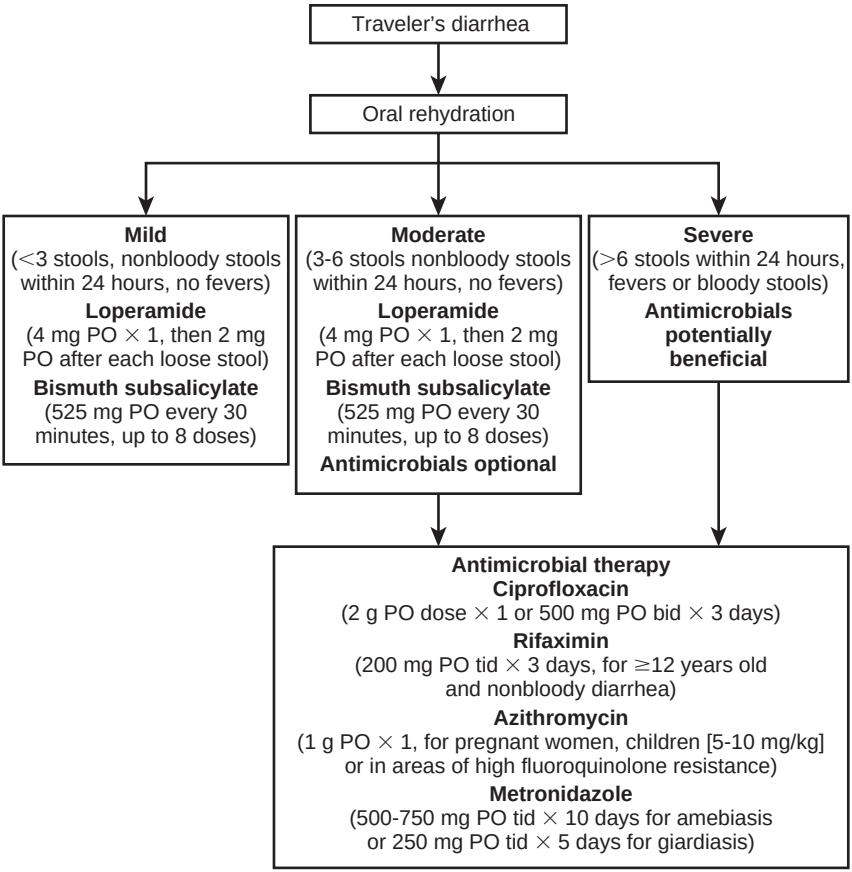

Figure 52-1. Approach to traveler's diarrhea. *bid,* Twice daily; *PO,* orally *(per os)*; *tid,* three times daily.

- Severe hypotension resembling anaphylactic shock and requiring pressor support
- Acute pulmonary edema

Treatment with H_1- and H_2-antagonists is generally effective.

29. What is ciguatera poisoning, and how is it treated?

Ciguatera poisoning occurs after the ingestion of coral reef fish (barracuda, sea bass, parrot fish, red snapper, grouper, amber jack, kingfish, and sturgeon) that contain ciguatoxin, a heat-stable toxin that opens sodium channels. Fish contaminated with ciguatoxin have no distinctive appearance, odor, or taste. Symptoms, starting within hours of ingestion, include diaphoresis, headache, abdominal pain, nausea, vomiting, watery diarrhea, and neurologic symptoms, including reversal of temperature perception, facial/perioral paresthesias, sensation of loose/painful teeth, ataxia, and coma. Other findings include bradycardia, hypotension, arthralgias, myalgias, itching, dysuria, and dyspareunia. Most symptoms resolve within a few days; however, severe neurologic and cardiac symptoms may persist for days to weeks. Treatment is largely supportive care.

30. What is tetrodotoxin poisoning, and how is it treated?

Tetrodotoxin is found in puffer fish, blue-ringed octopus, horse shoe crabs, and rough-skinned newt. It blocks sodium channels in the central and peripheral (including autonomic) nervous systems, interfering with axonal nerve transmission in muscle. Tetrodotoxin is resistant to cooking and is

most concentrated in the viscera and skin of the puffer fish. Safe consumption is predicated on expert chefs removing these areas. 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. Respiratory failure ensues and is followed by cardiovascular collapse, coma, and death within 6 hours on average. Treatment should be focused on airway management, mechanical ventilation, and supportive care.

31. 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 supportive. Syndromes include:

- Amnesic shellfish poisoning (ASP) is caused by domoic acid, a preformed agent with neuroexcitatory glutamatergic activity, found primarily in squid, scallops, mussels, and razor clams. Symptoms start within 24 hours of ingestion and can include nausea, vomiting, dizziness, headache, confusion, respiratory difficulty, and coma with loss of short-term memory that may be permanent.
- Diarrhetic shellfish poisoning (DSP) is caused by okadaic acid found in affected mussels, cockles, scallops, oysters, 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.
- PSP is caused by saxitoxin 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.
- Neurotoxic shellfish poisoning (NSP) is caused by the brevotoxin family of toxins, commonly found in cockles, mussels, and whelks off the coast of Florida and the Gulf of Mexico. Symptoms begin 15 minutes to 18 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, bradycardia, and respiratory difficulty. Mechanism and symptoms are similar to ciguatoxin poisoning but without long-term sequelae.

32. Which population of patients is at risk from eating raw oysters?

Patients with preexisting liver diseases (cirrhosis and hemochromatosis), immunodeficiency, or hematologic disorders with elevated iron levels have an 80 times higher risk of invasive *Vibrio* disease. Consumption of raw oysters, especially from warmer waters between March and November, has a high incidence of *Vibrio vulnificus* and *Vibrio parahaemolyticus*.

33. Describe the four stages of *A. phalloides* mushroom toxicity.

- Stage 1: Patient remains asymptomatic for 6 to 24 hours postingestion, an important distinction from most GI-irritant mushrooms. After the initial latent period, patients experience violent onset of nausea, vomiting, and diarrhea (often bloody), and severe abdominal pain lasting 1 to 2 days, which is often misdiagnosed as viral gastroenteritis. This stage may include acid-base disturbances, electrolyte abnormalities, hypoglycemia, dehydration, and hypotension. Physical examination may be significant for epigastric tenderness and hepatomegaly with normal liver function tests.
- Stage 2: The quiescent stage begins 24 to 48 hours after ingestion, and is characterized by transient clinical improvement despite continued hepatic deterioration.
- Stage 3: Beginning suddenly 2 to 4 days postingestion, the patient develops hepatic and renal failure, with marked rise in liver function tests, cardiomyopathy, hepatic encephalopathy, convulsions, coma, and death.
- Stage 4: Recovery stage for survivors.

KEY POINTS: FOOD POISONING

1. Antibiotics are not recommended in children with bloody diarrhea with concern for toxin release and development of HUS.
2. Patients with liver disease are at highest risk for invasive *Vibrio* disease from eating raw oysters.
3. Patients with *A. phalloides* toxic poisoning may appear to be improving before experiencing rapid deterioration.

Acknowledgment

The editors gratefully acknowledge the contributions of John E. Houghland, MD, Kevin Dean, MD, and Scott Rudkin, MD, MBA, authors of this chapter in the previous edition.

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QUESTIONS

1. Which of the following patients with full childhood immunizations will require an update of tetanus immunization?
   a. A 28-year-old man with a clean cut to his left index finger, who had his last immunization 3 years ago
   b. A 35-year-old man who was a deep puncture wound from a rusty nail, with his last immunization 4 years ago
   c. A 59-year-old female who has a superficial cut to her scalp with her last immunization 8 years ago
   d. A 24-year-old female who is 8 weeks pregnant with a cut to the right arm by a kitchen knife, who had her last immunization 2 years ago during her previous pregnancy.

   The correct answer is *d*.
2. Which of the following is true regarding *C. botulinum* toxin?
   a. Most common cause of death in the United States from botulism and is secondary to autonomic dysfunction.
   b. Associated with the physical examination finding of *risus sardonicus*
   c. Prevents the release of acetylcholine from the presynaptic neuronal membrane in the neuromuscular junction
   d. Infants in developing countries acquire the disease from lack of maternal immunization and lack of umbilical stump hygiene

   The correct answer is *c*.
3. A patient with facial erythema 15 minutes after eating mahimahi, which he states tasted peppery, most likely has which of the following?
   a. Scombroid poisoning
   b. Tetrodotoxin poisoning
   c. Neurotoxic shellfish poisoning
   d. Ciguatera poisoning

   The correct answer is *a*.

CHAPTER 53

TRAVEL MEDICINE AND VECTOR-BORNE DISEASES

Jennifer W. Bellows, MD, MPH

1. What is travel medicine?

Travel medicine refers to the prevention and management of health problems in travelers.

2. Why is travel medicine important?

International tourism and business travel are growing industries; more than 1 billion international arrivals occurred worldwide, and nearly 62 million international trips were taken by Americans in 2013. Approximately 8% of 50 million travelers from high-income countries to developing ones become ill enough to seek care. Health care providers and medical students are traveling internationally at record levels to provide charitable health care and to participate in clinical rotations. For example, greater than 30% of graduating medical students in 2013 took part in an overseas medical elective before graduation, compared to 6% in 1984.

3. Should I visit a travel clinic before my trip?

Fewer than half of patients treated after travel report a pretravel clinic visit. All travelers, especially those going to developing countries, should visit a travel medicine clinic 4 to 8 weeks before departure to obtain antimalarial prophylaxis and required vaccinations. Dental, gynecologic, and primary health care visits are also recommended, particularly before prolonged travel to areas with few health care resources. The traveler should assemble a medical kit with the basics: first-aid supplies, antidiarrheal medicines, contraceptives, water disinfectant, sunscreen, insect repellent, prescribed medications and their generic names, and antihistamines.

4. What other pretravel preparation should take place?

Travelers should have a basic understanding of health risks particular to their journey, activities, and destination, and take relevant precautions before, during, and after travel. The Centers for Disease Control and Prevention (CDC) *Yellow Book* provides information regarding country and region-specific health risks, and is an invaluable resource for both travelers and health professionals. Travelers should register with the Smart Traveler Enrollment Program of the U.S. Department of State to receive up-to-date information on travel warnings and to facilitate seeking medical care or repatriation if needed. The U.S Department of State also provides health-related information, including detailed country-specific safety concerns, instructions for finding hospitals and arranging medical evacuation while abroad, and links to travel insurance plans. Travel insurance is highly recommended and should cover hospitalizations and acute care in the destination country, as well as repatriation if necessary.

5. How should I prepare professionally before embarking on volunteer clinical work overseas?

Physicians should volunteer with organizations that have a long-standing and integrated role within the local health care system. Volunteers should have expertise treating diseases common in the region, speak the local language, and understand the cultural and social influences on health care provision. Participation in a tropical and/or wilderness medicine course is advised.

6. What clinical history should I obtain from the ill returned traveler?

Find out about the location of travel, including layovers, type of transportation, dates of travel, accommodations, activities, and exposures. Ask specifically about use of insect repellents, bed nets, food and water safety, exposure to fresh water, insect and animal bites, body fluid exposures, and pretravel preparation, including vaccines and use of malarial prophylaxis. Careful investigation of the timing of symptom onset and associated symptoms is crucial as well. The CDC's website is an

Abstract

Travelers are exposed to a wide variety of exotic diseases abroad and in the United States. Because many of these patients will come to an ED, emergency medicine physicians should be familiar with these diseases.

Keywords:

travel, vector-borne, tick-borne, mosquito-borne, emergency medicine, returned traveler, travel medicine

excellent resource for researching common health conditions by geographic region, presenting signs and symptoms, and season, as well as updates on recent outbreaks.

7. What are the most common illnesses affecting returned travelers?

- Diarrhea and other gastrointestinal illness (37%)
- Febrile illnesses (14%)
- Skin conditions (12%)

Nearly 1 in 5 patients with fever is diagnosed with *Plasmodium falciparum* malaria.

8. What causes traveler's diarrhea (TD)?

Acute diarrheal illness, or TD, is the most common illness in persons traveling from high-income to low-income regions. Travelers to developing countries who consume inadequately prepared food, and fruits and vegetables that are not peeled or washed in treated water are at highest risk. Most TD is caused by an unspecified pathogen; a recent surveillance study found that about 23% is bacterial, followed by *Giardia* (13%), amebas (4%), and *Campylobacter* (4%). An overview of the clinical presentation, prevention, and empiric treatment of TD is available in Chapter 52.

9. What are the most common skin conditions seen in returned travelers?

Most skin conditions seen in travelers are similar to those seen domestically and caused by the same organisms, including insect bites, cellulitis, nonspecific dermatitis, and fungal infections. A few notable exceptions include cutaneous leishmaniases, cutaneous larva migrans, and rashes associated with systemic illnesses such as dengue, rickettsial infections, and chikungunya fever. Diagnosis relies heavily on knowing the location and timing of travel; a history of animal, insect, or arthropod bites, stings and scratches; and sexual activity.

10. What is leishmaniasis?

A vector-borne disease transmitted by sand flies, leishmaniasis can present in myriad ways, but most commonly with cutaneous ulcers on exposed skin called *localized cutaneous leishmaniasis (LCL).* It begins as a pink papule that eventually enlarges into a painless ulceration with an indurated border. Symptoms can start weeks or months after initial exposure, and it is most common among travelers to the Middle East, North Africa, and Central and South America. Diagnosis is made via identification of the vector from a suspicious ulcer though culture or polymerase chain reaction (PCR).

11. How is LCL treated?

Treatment of LCL will decrease risk of scarring and disfigurement, as well as the risk of extension to one of the many other manifestations of the disease, such as mucosal and diffuse cutaneous leishmaniasis. Topical agents (e.g., paromomycin) are first-line treatment; thermotherapy or cryotherapy can be performed by an experienced dermatologist.

12. What are the most important tools in diagnosing these vector-borne diseases?

A detailed history and a skin examination are critical. 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.

13. What causes malaria?

There are four species of the protozoan *Plasmodium: P. falciparum, P. vivax, P. ovale*, and *P. 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 most life-threatening type, causes virtually all of the 1 million annual malaria deaths worldwide.

14. Can malaria be prevented?

Although no one method of protection is 100% effective, proper use of prophylactic drugs, bed nets, and insect repellent, as well as proper patient education, would prevent most cases of traveler's malaria seen in the United States.

15. What is the presentation of malaria in ED patients?

Symptoms develop 10 to 14 days after *P. falciparum* infection, but may be latent for 1 year with *P. vivax*. Patients complain of flulike 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 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.

16. 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 parasitemia percentage. Despite recommended repeat blood smears at least three times at 12-hour intervals, false-negative results will still occur. Antigen rapid detection kits are useful in case of limited access to an experienced laboratory technician.

17. 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). Treatment should be initiated according to the CDC, which has a 24-hour hotline for clinicians, as well as comprehensive online resources. 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 empiric 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.

18. What is dengue, and where does it occur?

Dengue is a mosquito-borne flavivirus endemic to tropical regions. The CDC reported a fourfold increase in reported cases from 1989 to 2007 in Central America, South America, Mexico, and the Caribbean. In the United States, eight localized outbreaks on the Texas-Mexico border since 1980, and a 2009 outbreak of 27 cases in Key West were the first cases acquired in the continental United States outside the Texas-Mexico border since 1945. The principal vector, *Aedes aegypti*, is found across the southeastern United States.

19. How does dengue fever present?

Many infections, especially in children, are asymptomatic or go unnoticed as a mild febrile illness. Classic dengue fever occurs 5 to 6 days after incubation and lasts about 1 week, with most patients fully recovering. Presenting symptoms include fever, retroorbital headache, nausea, vomiting, arthralgias, and severe myalgias, earning it the nickname of *breakbone fever.* The characteristic confluent, blanching, macular rash occurs in 50% of cases. A small proportion of patients go on to develop severe dengue, which is characterized by vascular permeability with consequent pulmonary effusions, ascites, hemorrhage, and multiorgan failure. Severe, untreated dengue carries a mortality rate of greater than 40%.

20. How do I diagnose and treat dengue fever?

Dengue fever should be suspected in patients returning from endemic areas with suspicious symptoms and laboratory markers. The diagnosis can be made with enzyme-linked immunosorbent assay (ELISA) serology, but it is often negative during acute illness and may cross react with antibodies to other arboviruses, such as West Nile virus. Laboratory abnormalities include thrombocytopenia, leukopenia, and nonspecific elevation of liver enzymes. Progression to severe dengue is marked by hemoconcentration, hypoproteinemia, and rapid decrease in platelet count.

Treatment is supportive: fluids and analgesics for dengue fever and intensive care for severe dengue, which in experienced centers can reduce the mortality rate to less than 1%. Nonsteroidal antiinflammatory drugs (NSAIDs) and aspirin must be avoided because of their platelet inhibition. A vaccine is undergoing phase III clinical trials, but it is not yet commercially available.

21. What is West Nile virus?

The West Nile virus is a flavivirus acquired through the bite of *Culex* mosquitoes. It is endemic in Africa, the Middle East, southern Europe, Southwest Asia, and Australia. The United States reported its first outbreak in New York City in 1999; the virus spread westward to the Pacific by 2003. A U.S. peak incidence of nearly 10,000 was reported in 2003, decreasing to 2500 cases in 2013.

22. What are the symptoms of West Nile infections?

Only one in five infections leads to symptoms; only 1 in approximately 200 present with central nervous system involvement. Symptomatic infection with the West Nile virus occurs 2 to 14 days after exposure, and 94% of patients in the United States report symptom onset between July and September. Initial symptoms include fever, headache, weakness, nausea, vomiting, and a maculopapular rash that predominates the torso and extremities, sparing the palms and soles. Those with central nervous system involvement most commonly have an encephalitic syndrome that includes 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.

23. How is West Nile encephalitis diagnosed and treated?

Diagnosis is made with enzyme-linked immunosorbent assay (ELISA) antibody assays on serum or cerebrospinal fluid (CSF). Treatment is supportive.

24. 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. 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.

25. List the principal vectors and distribution of tick-borne diseases.

See Table 53-1.

26. 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 and 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.

27. Describe the three clinical stages of Lyme disease.

Like syphilis, another spirochetal disease, there are three phases.

1. Early acute or localized infection: In 80% of infections, the classic skin lesion, erythema migrans (EM), develops at the bite site within 3 to 30 days. Up to 75% of patients with EM will not recall a tick bite. EM expands slowly as an erythematous macule with central clearing that 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.
2. Early disseminated disease: Untreated, this may occur 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. Neurologic manifestations include cranial neuritis, such as unilateral or bilateral lower facial nerve palsy, and aseptic meningitis. Other neurologic manifestations include radiculoneuritis, similar to postherpetic neuralgia, with burning and paresthesias. Carditis, most commonly presenting with atrioventricular blocks, occurs in fewer than 10% of cases.

Table 53-1. Principal Vectors and Distribution of the Tick-Borne Diseases

DISEASE	VECTOR	PATHOGEN	U.S. DISTRIBUTION	FIRST-LINE TREATMENT
Babesiosis	*Ixodes scapularis*	*Babesia microti*	Northeast, upper Midwest	Azithromycin and atovaquone
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 disease	Doxycycline
Lyme disease	*Ixodes* species	*Borrelia burgdorferi*	Northeast, Midwest, West	Doxycycline
Tick-borne relapsing fever	*Ornithodoros* species	*Borrelia* species	Western mountains	Tetracycline
RMSF	*Dermacentor* species	*Rickettsia rickettsii*	Nationwide, mostly Southeast	Doxycycline
STARI	*A. americanum*	*Borrelia lonestari*	South	Doxycycline
Tick paralysis	Multiple	Toxins	Nationwide	Tick removal
Tularemia	*A. americanum* and *Dermacentor* species	*F. tularensis*	West, South Central	Streptomycin

RMSF, Rocky Mountain spotted fever; *STARI*, southern tick-associated rash illness.

3. Late or chronic disease: Late manifestations occur months to years after infection. Lyme arthritis is most common, presenting in 10% of those untreated and affecting one or several large joints, usually the knee. There is typically no joint destruction, and symptoms may spontaneously resolve 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.

28. 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. Blood cultures have low sensitivity and thus are of little diagnostic value.

29. How is Lyme disease treated?

Oral therapy with doxycycline or amoxicillin is effective for early disease and for mild early disseminated disease. Neurologic and cardiac manifestations (with the exception of an isolated Bell 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 milliseconds, given the risk of progression. Temporary pacing may be needed for third-degree blocks. Late Lyme disease may not always respond to treatment.

30. Can Lyme disease be prevented?

The mainstays of prevention remain avoiding tick exposure, 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.

31. An ED patient has 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 *I. 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 disease (pediatric dosage is 4 mg/kg up to maximum dose of 200 mg). 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 EM rash develops at site of tick bite.
3. Heart block, lower motor facial nerve palsy, and arthritis develop in advanced disease.
4. Positive serology test is not diagnostic of infection.
5. Treatment is doxycycline, amoxicillin, or ceftriaxone.

32. What on earth is STARI and what can be done about it?

STARI stands for *southern tick-associated rash illness* and causes EM just like Lyme disease. It is caused by a spirochete, *Borrelia lonestari*, transmitted by the lone star tick, *Amblyomma americanum*. If a patient comes to the ED in the southern United States, an area nonendemic for Lyme disease, the diagnosis is likely to be STARI. Treat it just as you would early localized Lyme disease.

33. What is tick-borne relapsing fever?

Tick-borne relapsing fever (TBRF) 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. 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 disease responds well to doxycycline and erythromycin, but a Jarisch-Herxheimer reaction may occur (malaise and hypotension.)

34. What is Rocky Mountain spotted fever (RMSF)?

RMSF is a life-threatening infection caused by *Rickettsia rickettsii* and transmitted by *Dermacentor*, or dog ticks. Currently, most cases are reported from the southeastern and south-central United States. Abrupt-onset fever, severe headache, and myalgias 5 to 7 days after the tick bite are the most common presenting symptoms. The rash is petechial, occurring initially on the wrists and ankles. It then spreads to the palms and soles, and then the trunk, often progressing into purpuric lesions. Although the rash is rarely present during the first 3 days of illness, about 60% of infections will develop with the classic triad of rash, fever, and tick exposure; 10% of patients never develop a rash.

35. How dangerous is RMSF? What can be done about it?

Untreated, RMSF mortality ranges from 20% to 80%. The rickettsial pathogen induces a vasculitis that leads to end-organ dysfunction, including confusion, respiratory failure, and renal failure. Death is most typically a result of disseminated intravascular coagulation. Appropriate and timely antibiotics can reduce the mortality rate to less than 5%, but delays are common because of the late onset of the characteristic rash. Doxycycline remains the drug of choice. Consider early empiric treatment in the spring and summer in endemic regions.

36. What are ehrlichiosis and anaplasmosis?

Ehrlichiosis and anaplasmosis are tick-borne diseases caused by the rickettsia-like bacteria, *Ehrlichia chaffeensis* and *Anaplasma phagocytophilia.* 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. Both diseases present with fever and flulike symptoms and progress coma, with respiratory and renal failure in severe cases. Rash may occur in ehrlichiosis but not in anaplasmosis.

37. 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. Microscopic examination of buffy coat or peripheral blood may reveal characteristic inclusion bodies in monocytes or neutrophils. Doxycycline is first-line therapy.

38. What is Colorado tick fever?

Colorado tick fever is caused by an RNA *Coltivirus* that is transmitted by *Dermacentor* ticks in the western United States. Patients generally seek treatment 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 in 3 days. Prognosis is excellent, although complications such as encephalitis, meningitis, and pericarditis have been reported. Diagnosis is by serology, and treatment is supportive.

39. What is babesiosis?

Babesiosis is a malaria-like illness, caused by the *Babesia microti* protozoan. It is transmitted by *Ixodes* ticks in the northeastern and upper midwestern United States. Patients have fever, drenching sweats, myalgias, and headache. Although 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 azithromycin plus atovaquone for mild infection, and quinine plus clindamycin for more severe cases.

40. What is tularemia?

Tularemia is a rare disease caused by *Francisella tularensis*, a virulent gram-negative coccobacillus. Transmission occurs through tick bites or contact with infected tissue of rabbits or rodents; cases have been reported in all U.S. states except Hawaii. 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. Untreated, the mortality rate is 30% to 60% from septic shock. Streptomycin remains the drug of choice to treat both forms of the disease.

41. What is tick paralysis?

Tick paralysis is a syndrome caused by neurotoxins in the saliva of gravid female ticks. The syndrome usually presents as an ascending paralysis similar to that of Guillain-Barré disease, with sparing of sensorium and sensory function. Young girls in western North America are at highest risk, especially after prolonged tick attachment. Mortality does occur as a result of respiratory failure. Tick removal usually brings about prompt and complete recovery; treatment is otherwise supportive.

42. What is the proper method for tick removal?

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.

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Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Andrew M. Kestler for his previous contributions to this chapter.

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QUESTIONS

1. Which of the following is true about malaria?
   a. Thin smears are more sensitive for diagnosis.
   b. Smears should be repeated twice if they are initially negative.
   c. Treatment should never be started empirically.
   d. Treatment may cause dysrhythmias.
   The correct answer is *d*.
2. Which is not true regarding RMSF?
   a. Most cases are reported from the southeastern and south-central United States.
   b. Symptoms abruptly start the day after a bite from the dog tick *Dermacentor*.
   c. Rash is petechial, spreading from wrists and ankles to the palms and soles and finally to the trunk.
   d. 10% of patients never develop the rash.
   The correct answer is *b*.
3. One in how many patients infected with West Nile virus will develop central nervous system manifestations?
   a. 10
   b. 50
   c. 100
   d. 200
   The correct answer is *d*.

CHAPTER 54

ARTHRITIS

Nicole M. Dubosh, MD

1. What are the signs and symptoms of arthritis?

Arthritis refers to the inflammation of a joint. The process may be monoarticular (involving a single joint) or polyarticular (involving multiple joints). Common presenting symptoms include 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 have a limp or avoid using the extremity. There are many different etiologies of arthritis, some of which are more serious than others and can result in permanent joint damage and increased mortality. It is important for the emergency physician to identify those types of arthritis that require immediate management.

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, systemic lupus erythematosus [SLE], Reiter syndrome)
- Degenerative conditions (osteoarthritis [OA])

3. What is the difference between an intraarticular and a periarticular process?

An intraarticular process involves inflammation of the synovium. This results in diffuse, generalized joint pain, warmth, effusion, and an increase in pain with range of motion about the joint and with axial loading. Arthritis is an intraarticular process. A periarticular process has a more localized area of tenderness, lack of joint effusion, and pain with stretching muscles and tendons over the affected surfaces, as opposed to with moving the joint throughout its entire range. Bursitis and tendinitis are examples of periarticular processes.

4. What are some examples of diseases that are monoarticular, polyarticular, and periarticular?

See Table 54-1 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 diseases. Examples include genital ulcerations, purulent urethral discharge, and conjunctivitis in Reiter syndrome; urethral or cervical discharge in gonococcal arthritis; tophi or concomitant renal stones in gout; malar rash in SLE; swan-neck deformity in rheumatoid arthritis; erythema chronicum migrans rash in Lyme disease; and evidence of joint surgery or cellulitis overlying a prosthetic joint 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 commonly affects the first metatarsophalangeal (MTP) joint. Rheumatoid arthritis commonly affects the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints. OA often affects the distal interphalangeal (DIP) and the first MCP joints. Septic joints most commonly involve the knee (>50%) and the hip.

7. Is radiography helpful in the diagnosis of arthritis?

Oftentimes the only radiographic evidence of inflammation is soft-tissue swelling. Plain radiographs may reveal foreign bodies, fractures, effusions, osteoporosis, or osteomyelitis. The radiographic

Abstract

Arthritis refers to the inflammation of a joint. Patients with arthritis often have joint pain at rest, swelling, and pain with moving the joint. Etiologies of arthritis include infection, degeneration, trauma, crystal deposition, hemorrhage, neoplasm, and inflammatory conditions. Septic arthritis, or bacterial arthritis, is the most serious of these and requires prompt recognition, because it can lead to permanent joint damage and increased mortality. It is important for the emergency physician to understand how to diagnose and treat various types of arthritis.

Keywords:

arthritis, septic arthritis, gout, pseudogout, osteoarthritis (OA), Lyme arthritis, arthrocentesis

Table 54-1. 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 syndrome	
	Lyme disease	
	Serum sickness	

changes of degenerative arthritis include asymmetric 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 cell (WBC) count useful for the evaluation of acute arthritis?

No, peripheral WBC, ESR, and C-reactive protein (CRP) are nonspecific markers of inflammation and are not useful in acute arthritis. A recent metaanalysis found no reduction in posttest probability of a septic joint based on these findings. The ESR and WBC represent the body's acute-phase reaction to inflammation and infection and 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.

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. An arthrocentesis can drain a tense hemarthrosis and enable a caregiver to inject an analgesic or antiinflammatory medication into the joint. The procedure is simple and safe, and complications are rare when performed under sterile conditions with proper technique. If a prosthetic joint infection is suspected, an orthopedic consultation should be obtained before joint aspiration.

10. What are the general steps of an arthrocentesis?

1. Place the patient in a comfortable position with the joint exposed.
2. Palpate the bony landmarks.
3. Cleanse and prep the skin, and drape the patient with sterile drapes.
4. Provide anesthesia by local infiltration with an anesthetic, such as 1% or 2% lidocaine.
5. Using an 18-gauge needle (or smaller, depending on joint size) attached to a syringe, aspirate gently while carefully advancing the needle into the joint. Avoid puncture of the articular cartilage.
6. Withdraw as much synovial fluid as possible.
7. If necessary, inject anesthetic solution into the joint for pain relief.

Send the synovial fluid for WBC count with differential, crystals, Gram stain, culture, and if possible, synovial lactate tests. If you only retrieve one drop of fluid, it should be sent for culture.

11. What are some causes of arthritis with fever?

Diseases causing arthritis with fever include septic arthritis, Lyme disease, rheumatic fever, Reiter syndrome, and toxic synovitis.

12. How do I interpret the results of the arthrocentesis?

See Table 54-2 for interpretation of synovial fluid analysis.

13. Does a synovial fluid WBC 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) WBCs, and a Gram stain positive for bacteria. However, some patients with septic arthritis had synovial fluid counts of less than 50,000 cells/mm^3.

Table 54-2. 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	50-4000 (600)	<30	Good	NS	—
Group II (Noninfectious, Mildly Inflammatory; SLE Scleroderma)						
	Clear to slightly turbid	0-9000 (3000)	<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*

(From Wyngaarden JB, Smith LH, editors: *Cecil textbook of medicine*, ed 18, Philadelphia, 1988, Saunders, p 1994, with permission.)
LE, Lupus erythematosus; *NS*, not significant; *PMN*, polymorphonuclear cells; *SLE*, systemic lupus erythematosus.

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 should be low if the clinical examination suggests bacterial arthritis.

14. Are there any other synovial fluid tests for arthritis?

There is emerging evidence to suggest that synovial fluid lactate levels greater than 10 mmol/L is highly suggestive of septic arthritis, whereas levels less than 4.3 mmol/L make septic arthritis very unlikely.

15. What is the most serious cause of arthritis?

Nongonococcal 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 preexisting joint disease, including prosthetic joints and rheumatoid arthritis. Almost half of patients with septic arthritis have 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.

16. What organisms cause bacterial arthritis?

Septic arthritis can be grouped into gonococcal and nongonococcal, as the disease process and management differs. *Neisseria gonorrhoeae* is the most common cause of septic arthritis in young, healthy, sexually active adults. The most common cause of nongonococcal septic arthritis is *Staphylococcus aureus,* followed by *Streptococcus* species. Methicillin-resistant *S. aureus* (MRSA) causes up to half of septic arthritis cases, with risk factors including advanced age, comorbid medical conditions, and recent hospitalization. Other causative organisms include *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.

17. How is bacterial arthritis treated?

Patients with bacterial arthritis require admission to the hospital and immediate orthopedic consultation 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. Culture results should not delay initiation of IV antibiotics. Vancomycin and a third-generation cephalosporin is the recommended empiric coverage, and if the patient is allergic to penicillins or cephalosporins, aztreonam or a fluoroquinolone can be substituted. See Table 54-3 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.

18. What causes crystal-induced arthritis?

Crystal-induced arthropathies include gout and pseudogout. They are more common than septic arthritis and often mimic a septic joint. Gout is caused by monosodium urate crystal precipitation into a joint, whereas pseudogout develops when calcium pyrophosphate crystals precipitate into 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 commonly 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 many cases.

20. What medications can be used to treat gout in the acute setting?

Nonsteroidal antiinflammatory drugs (NSAIDs) are the primary agents used to treat gout. For example, indomethacin is given at a dosage of 75 to 200 mg/day for several days, then tapered

Table 54-3. Antibiotic Treatment for Septic Arthritis

ORGANISM	GRAM STAIN	ANTIBIOTICS	DOSAGE
Methicillin-sensitive *Staphylococcus aureus*	Gram-positive cocci clusters	Cefazolin, nafcillin, or oxacillin	Cefazolin 2 g IV q8h Nafcillin 2 g IV q4h Oxacillin 2 g IV q4h
Methicillin-resistant *S. aureus*	Gram-positive cocci clusters	Vancomycin	Vancomycin 15 mg/kg IV q12h
Streptococcus pneumonia	Gram-positive cocci chains	Penicillin G or ampicillin	Penicillin G 12-18 mU IV q24h, divided
Penicillin sensitive			Ampicillin 2 g IV q4h
S. pneumonia, penicillin resistant	Gram-positive cocci chains	Ceftriaxone or cefepime	Ceftriaxone 1 g IV q24h Cefepime 2 g IV q8h
Neisseria gonorrhoeae	Gram-negative cocci	Ceftriaxone or cefepime	Ceftriaxone 1 g IV q24h Cefepime 2 g IV q8h
Pseudomonas aeruginosa	Gram-negative rods	Ceftazidime or cefepime plus gentamicin or tobramycin	Ceftazidime 2 g IV q8h Cefepime 2 g IV q8h Gentamicin 5 mg/kg IV q24h Tobramycin 5 mg/kg IV q24h

IV, Intravenously; *mU,* million units; *q,* every.

off as inflammation decreases. Colchicine is also effective in treating acute attacks and works by inhibiting microtubule formation, resulting in a decreased inflammatory response. It may be administered orally at an initial dose of 1.2 mg at the first sign of a flare followed by 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. Once bacterial infection has been ruled out, oral corticosteroids may also be administered: for example, 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?

Lyme disease, caused by the bacteria *Borrelia burgdorferi,* can cause arthritis as a late manifestation of the disease. Synovial fluid polymerase chain reaction (PCR) testing should be ordered for *B. burgdorferi* if clinical suspicion is high. The Infectious Diseases Society of America recommends treatment with a 28-day course of oral antibiotics (doxycycline 200 mg daily divided into two doses or amoxicillin 1.5 g daily divided into three doses) for patients without neurologic manifestations of the disease.

22. What are the signs and symptoms of OA?

OA, or degenerative arthritis, is the most common joint disease and is more prevalent in the elderly. Symptoms include chronic, progressive joint pain; morning stiffness; crepitus; Heberden nodes at the distal phalangeal joints; and Bouchard nodes at the PIP joints. The joint pain is generally worse with weight bearing and improves with rest.

23. What are the treatment options for OA?

Acetaminophen and NSAIDs are efficacious. Acetaminophen has a safer side-effect profile at 650 mg every 6 hours. Common NSAID regimens include ibuprofen 400 to 600 mg every 6 hours or naproxen 220 to 375 mg once or twice daily. Although efficacy is unproven, alternative therapies include glucosamine and chondroitin, alone or in combination. Intraarticular corticosteroid joint injections with methylprednisolone, triamcinolone, or betamethasone may provide relief.

KEY POINTS

1. Septic arthritis is a medical emergency, requiring prompt diagnosis by arthrocentesis, followed by management with IV antibiotics and orthopedic consultation for joint washout.
2. Abnormal serum inflammatory markers, including WBC count, ESR, and CRP, cannot be used to rule out a septic joint. Synovial fluid WBC count greater than 50,000/mm^3 and predominantly PMN WBCs are commonly seen in septic arthritis but are not absolute.
3. Gout and pseudogout are causes of arthritis caused by crystal precipitation in the joint and can mimic septic arthritis.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Catherine B. Custalow for her previous contributions to this chapter.

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QUESTIONS

1. A 65-year-old male has a swollen, erythematous, painful great toe. He denies any trauma or injury, and review of systems is otherwise negative. Arthrocentesis is performed, and synovial fluid analysis shows the following: 21,000 WBC/mm^3 with 65% PMNs, negative Gram stain, negatively birefringent needle-shaped crystals. What is the best management option?
   a. Start broad-spectrum IV antibiotics and consult orthopedics for emergent washout.
   b. Prescribe colchicine if there are no contraindications, and discharge home with outpatient primary care follow-up instructions.
   c. Discharge home with a short course of narcotics and outpatient primary care follow-up instruction.
   d. Discharge home with instructions for 3 weeks of doxycycline.
   The correct answer is *b*.
2. Which of the following rules out a septic joint?
   a. Less than 50,000 WBCs/mm^3 in the synovial fluid
   b. Normal serum ESR and CRP
   c. Synovial fluid lactate less than 4.3 mmol/L
   d. Negative synovial fluid culture
   The correct answer is *d*.
3. A 32-year-old healthy female exhibits malaise, bulls-eye rash, and joint pain in her knees, wrists, and elbows. She recalls a tick bite 1 month previously while hiking in Massachusetts. Her review of systems is otherwise negative. She takes no medications and has no allergies. Her vital signs are normal, and her physical examination is notable for slight joint tenderness, no evidence of joint effusion or erythema, and pain with range of motion of the joints mentioned. What is the best management of this patient?
   a. Perform arthrocentesis of all the the joints mentioned and treat based on culture results.
   b. Send for a Lyme titer and treat with a 3-week course of doxycycline.
   c. Perform a lumbar puncture, obtain an electrocardiograph (ECG), and admit for IV antibiotics.
   d. Treat with analgesics and refer to rheumatology clinic for further workup.
   The correct answer is *b*.

CHAPTER 55

SKIN DISEASES

Renee A. King, MD, MPH

1. What are the terms used to describe skin lesions?

Use characteristics such as color, contour, depth, distribution, location, presence of scales, and texture. Common terminology for skin lesions is listed in Table 55-1.

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. Which skin lesions could signify an emergent condition?

- Blistered or denuded skin
- Generalized erythema, especially in elderly, chronically debilitated, or febrile patients
- Petechia, purpura, or ecchymosis
- Necrosis
- Urticaria

4. What types of skin diseases result in potentially life-threatening compromise to the skin barrier?

Most are blistering diseases. When the blister breaks, there is 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.

KEY POINTS: DISEASES THAT CAUSE SIGNIFICANT SKIN BARRIER BREAKDOWN

1. Extensive blistering
2. Erythroderma
3. Extensive oral lesions that make decrease oral intake

5. Identify the skin lesions found in meningococcal disease, Rocky Mountain spotted fever, toxic shock syndrome, and necrotizing fasciitis?

- Meningococcemia: Petechiae or purpura with dusky centers, commonly found on the trunk and limbs; lesions may be on palms and soles.
- Rocky Mountain spotted fever: On the fourth day after fever, lesions originate on distal extremities, may include the palms and soles, and spread centrally. After 1 to 2 days, skin

Abstract

This section discusses cutaneous illnesses that are often diagnosed and treated in the ED setting. Definitions of common dermatologic descriptions are included, and should provide the reader with basic terms that describe characteristics of common lesions. Etiologies, common signs and symptoms, and treatments of various cutaneous disorders, including common childhood exanthems, are a significant portion of this discussion. Review questions have been provided at the end, which will allow the reader to review and retain what has been discussed in the chapter.

Keywords:

cutaneous, skin, lesions, exanthem

Table 55-1. 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

findings evolve into petechia or purpura. The rash can be difficult to differentiate from meningococcemia.

- Toxic shock syndrome: Lesions include a scarlatiniform rash, edema of the face and limbs, conjunctival erythema, and mucosal redness within oral or genital areas. Desquamation of hands and feet 1 to 2 weeks after initial lesions may be seen.
- Necrotizing fasciitis: Briskly advancing tender erythema, progressing to duskiness and necrosis with or without blisters; overlying skin findings may belie the necrosis occurring underneath.

6. Describe findings seen in common childhood skin rashes.

See Table 55-2.

7. Describe 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. Lesions are found on the dorsal hands and extensor extremities, and palms and soles commonly are involved. Mucous membranes are usually spared or affected mildly, and significant involvement should raise suspicion for another diagnosis, such as SJS.

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 because of intense necrosis. EM has been linked to infections, medications, malignancy, autoimmune diseases, and immunizations. EM commonly follows herpes simplex virus (HSV) infection; *Mycoplasma* pneumonia is another etiology. The eruption lasts 10 to 14 days and may recur after subsequent episodes of HSV. Management consists of antihistamines to ease significant itching and discomfort. Topical or oral steroids are not indicated.

8. Which illness can mimic EM?

Acute urticaria may appear on palms and soles and are disk shaped with a pale center and surrounding erythema. EM has a darker center. Urticaria often moves around the body, whereas the lesions of EM are fixed. Unlike urticaria, skin findings of EM do not improve with diphenhydramine or epinephrine.

Table 55-2. Childhood Skin Rashes

ILLNESS (PATHOGEN)	AGE OF PRESENTATION	CLINICAL FINDINGS	OTHER
Erythema infectiosum (parvovirus B19)	School-age children	Bright red patches (especially around the cheeks), followed by lacy, reticular-like pattern; rash follows resolution of fever and clinical improvement	Also called *fifth disease* or *slapped-cheek disease*; rash can last for months, precipitated by changes in temperature, sun, exercise, and emotional stress
Hand-foot-mouth (Coxsackie virus)	Young children	Sudden onset of scattered papules on palms, soles, buttocks, and mucosa that develop into vesicles with a red rim	Late summer and fall
Kawasaki disease (KD) (unknown; likely immune phenomenon)	Young children, but can occur at any age; incomplete presentations more likely in infants	High fever for at least 5 days with four of the following: Cervical lymphadenopathy Hands that are edematous or desquamating Exanthema Mucosal changes Nonpurulent conjunctivitis	Coronary artery aneurysms develop in 1:5 patients, leading to myocardial infarction and arrhythmias; treat with IVIG and aspirin Laboratory evidence for incomplete KD: CRP ≥3 mg/dL or ESR ≥40 mm/h WBC count ≥15,000/μL Normocytic, normochromic anemia Platelets ≥450,000/μL after 7 days of illness Sterile pyuria ≥10 WBCs/high-power field ALT >50 U/L albumin ≤3 g/dL
Roseola (herpes virus 6)	Infants and toddlers	Illness begins with 2 to 3 days of persistent fever, followed by sudden defervescence and the development of a pink maculopapular rash	Also called *exanthema subitum*; periorbital edema can be present.

Scarlet fever (group A strep)	Children 2-10 years of age most common	Red macules and papules beginning on the neck, extending to the trunk and extremities; skin can have a coarse sandpaper feel. Intense erythema is located in the axillae, groin, and abdomen.	Pastia lines (characterized by lines of petechiae beside major skin folds) may also be seen on examination. Palms and soles are spared, and an erythematous face with circumoral pallor may develop. Desquamation as the lesions resolve in 1 to 3 weeks.
Staphylococcal scalded skin syndrome (certain strains of *Staphylococcus*)	Children <5 years of age	Begins with upper respiratory infection, followed by redness on the face, neck, and axilla, with crusting around eyes, mouth, and as in skin folds.	Nikolsky sign: Skin detaches from the epidermis with slight rubbing, leaving an erythematous, damp skin; mucous membranes are not affected. Can affect the entire body in neonates; usually only the upper body in older children.
Varicella (varicella zoster virus)	Any age, especially in nonimmunized; milder cases still occur in vaccinated children and adults	Groups of faint macules evolve into papules and then vesicles over 1-2 days; vesicles acquire a moist crusty appearance and eventually erode into shallow lesions. Begins on the trunk and extends peripherally to extremities.	Also called *chicken pox*, a hallmark being lesions in various phases of development (i.e., macules, papules, vesicles, crusts, erosions). Can involve the palms, soles, and mucous membranes; very itchy. Resembles "dew drop on a rose petal"; i.e., vesicle sits atop a larger red macule.

ALT, Alanine aminotransferase; *CRP*, C-reactive protein; *ESR*, erythrocyte sedimentation rate; *IVIG*, intravenous immunoglobulin; *WBC*, white blood cell.

9. How do adverse drug reactions typically present?

Adverse drug reactions typically appear as generalized erythematous macules and papules. Skin findings typically commence 1 to 2 weeks after initiation of a new drug. Management comprises discontinuing the causal medication. Use of antihistamines may relieve itching and discomfort.

10. Which medications are commonly implicated in drug eruptions?

- Aminopenicillins
- Anticonvulsants
- Cephalosporins
- Sulfonamides

11. Which illness commonly mimics drug eruptions?

Drug eruptions and viral exanthems have similar skin and histologic findings, relying on a thorough history to differentiate them. Eosinophilia supports the diagnosis of drug eruption. Ten percent to 20% of exanthematous eruptions in children are medication-related, as are 50% to 70% in adults.

12. What clinical signs should alert concern of a severe adverse medication reaction?

Signs of severe reactions include facial edema, enlargement of the liver and spleen, mucosal petechia, significant eosinophilia, painful dark-colored papules or blisters, and skin sloughing. The most serious and life threatening of these reactions include SJS, TEN, and drug reaction with systemic symptoms (DRESS) (Table 55-3).

KEY POINTS: ONSET OF CUTANEOUS MEDICATION ERUPTIONS

1. Exanthematous drug eruptions: 4 to 14 days
2. SJS or TEN: 7 to 21 days
3. DRESS syndrome: 21 to 42 days

13. What are characteristics of melanoma?

Recognition of melanoma and referral for surgical removal 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. Also important to note is that melanoma may appear different than a person's other nevi. A brown nevus on a fair-skinned patient is concerning if no other lesions appear similar, even if the brown macule appears evenly pigmented, small, and entirely round. A change in nevus appearance is a risk factor, as is a previous episode or family history of melanoma.

KEY POINTS: SKIN FINDINGS SUGGESTIVE OF MELANOMA

1. Areas of pigment regression
2. Change in color, shape, or size
3. Irregular borders
4. Irregular pigmentation (presence of red, white, or blue)
5. Different from patient's other pigmented lesions

14. What other skin findings can mimic melanoma?

- Seborrheic keratosis: Common, benign, darkly pigmented or unevenly colored growths that typically occur in middle age. Growths commonly have scales, but they may not be easy to visualize with the naked eye.
- Venous lakes: 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.

15. Which spider bites cause necrosis?

Bites from the brown recluse *(Loxosceles reclusa)* and the hobo spider *(Tegenaria agrestis)* may lead to necrosis. The brown recluse is found in states ranging from Ohio to as far west as Nebraska and

Table 55-3. Characteristics of Severe Drug Eruptions

	ETIOLOGY	SIGNS/SYMPTOMS	MORTALITY RISK	TREATMENT
SJS	Medications (70% to 90%) Antibacterial sulfonamides Anticonvulsants NSAIDs Allopurinol *Mycoplasma pneumoniae* infection	Prodrome 1-14 days before onset of mucosal involvement. Typical symptoms include fever, malaise, headache, sore throat, rhinorrhea, and cough). Acute rash that begins as erythematous macules that progress to painful papules, then vesicles and bullae that blister and slough (usually <10% of the body surface involved). Mucosal necrosis and sloughing of two membranes (commonly mouth and eyes).	1% to 5%	Prompt withdrawal of offending agent is imperative. Symptomatic management is similar to burn treatment. Oral lesions are managed with mouthwashes and topical anesthetics to tolerate oral rehydration. Areas of denuded skin must be cleansed and covered to prevent infection and dehydration. Tetanus prophylaxis
TEN	Medications (95%): Antibiotics Sulfonamides Anticonvulsants NSAIDs Allopurinol	Onset with fever 1-2 weeks before appearance of painful skin lesions of blistering/sloughing. Painful, red macules appear, progress and become darker, then coalesce. Areas of hypopigmentation may also be interspersed, then blisters and sloughing are seen (usually >30% body surface 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.	25% to 35%	Prompt withdrawal of offending agent is imperative. Symptomatic management is similar to ICU/burn treatment is initiated. Controversies exist in the management of TEN, such as in the use of steroids and other immunomodulators. Early administration of intravenous immunoglobulin may be beneficial.
DRESS	Medications Anticonvulsants Antidepressants NSIADs Antibiotics, including sulfonamides, allopurinol, minocycline, dapsone Gold salts	Fever >38°C (100.4°F) and morbilliform rash are the most common presenting characteristics. Facial edema is a hallmark. Lymphadenopathy, eosinophilia, and atypical lymphocytosis are common features. Involvement of at least one internal organ is also seen.	10% to 20%	Prompt withdrawal of the offending agent is imperative. Systemic steroids are commonly used, although effectiveness is unverified in controlled clinical trials.

DRESS, Drug reaction with systemic symptoms; *ICU*, intensive care unit; *NSAID*, nonsteroidal antiinflammatory drug; *SJS*, Stevens-Johnson syndrome; *TEN*, toxic epidermal necrolysis.

in the South from Texas to Florida. Due to transportation of the spider in moving boxes and other methods, brown recluses may be encountered in states outside of this region. The hobo spider is found in the northwest United States and western Canada. Other *Loxosceles* species, found in deserts of the southwestern United States, may also cause necrosis.

16. Which skin findings may appear similar to a necrotic spider bite?

Necrotizing fasciitis, ecthyma, pyoderma gangrenosum, vasculitis, and clotting disorders may appear similar to spider bites. 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.

17. What kind of skin lesions do MRSA cause?

MRSA infections commonly cause lesions of the skin and soft tissue, including abscesses and furuncles. Certain findings may increase risk factors of MRSA infection, such as a reported history of spider bite, recent antibiotic use, history of diabetes, and increased skin exposure (e.g., athletes or locker rooms), as well as a previous episode of MRSA infection. In immunocompetent patients, antibiotics are not needed unless a surrounding cellulitis is present, fever is noted, or the lesion fails to resolve.

18. What kinds of purpura are associated with benign conditions?

- Actinic purpura: Common in elderly fair-skinned patients with chronic sun exposure, seen on the dorsum of the hands and forearms; characterized by 1- to 5-cm purplish ecchymotic lesions.
- Purpura pigmentosa progressiva: A benign and chronic condition characterized by lower extremity petechiae. Found in all age groups, lesions tend to be pinpoint, nonpalpable, and numerous. Cortisone cream may alleviate itchiness.

19. Which skin lesions mimic cellulitis?

- Stasis dermatitis: Erythema and scaling on the bilateral lower extremities; cellulitis is usually unilateral with pain and warmth over the affected area, rapidly expanding redness, and at times fever.
- Allergic contact dermatitis: Area of skin erythema associated with itchy vesicles and papules that develop within a few days of an allergen exposure. Requires removal of the offending agent along with topical corticosteroids. Systemic corticosteroids for 2 to 3 weeks may be necessary for severe reactions (e.g., severe poison ivy).

20. Debridement is generally contraindicated in which lower extremity eruption?

- Pyoderma gangrenosum

21. Should steroids be used to treat eczema?

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.

22. Should steroids be used in psoriasis?

Patients dealing with psoriasis have different treatment options. In mild disease, topical creams and emollients should suffice. Moderate psoriasis may require the addition of phototherapy. Although systemic treatments may become necessary in severe psoriasis, steroids are not usually recommended because rebound pustular psoriasis may develop once steroid medication is withdrawn.

23. What are the classes of steroids, and on which part of the body should they be applied?

See Table 55-4.

24. Which formulation of topical steroids is most potent?

Ointments have the greatest potency, followed by gels, emollients, creams, lotions, solutions, and sprays.

Table 55-4. Topical Steroids

POTENCY/CLASS	AREAS OF USE	EXAMPLES
Low potency Class 6 and 7	All areas, including thin-skinned areas: Face, breasts, axilla, and groin	1% hydrocortisone 2.5% hydrocortisone 0.05% desonide
Moderate potency Class 4 and 5	Neck and body; not on thin-skilled areas. Most commonly used in the ED setting	0.025% fluocinolone 0.1% triamcinolone 0.2% hydrocortisone valerate
High potency Class 2 and 3	Not on thin-skilled areas; best for areas with thicker skin (e.g., palms and soles). Can cause adverse effects when used >2 weeks	0.05% fluocinonide 0.1% halcinonide 0.25% desoximetasone
Superpotency Class 1	Used to treat prolonged, refractory illnesses, especially on thick skin, including the palms and soles; requires outpatient monitoring	0.05% clobetasol 0.05% betamethasone dipropionate in optimized vehicle 0.05% halobetasol 0.05% diflorasone

Acknowledgment

The editors and author of this chapter would like to acknowledge and thank Drs. Lela A. Lee and Joanna M. Burch for their previous contributions to this chapter.

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QUESTIONS

1. A 9-month-old, otherwise well-appearing infant presents with perioral edema noticed by his mother. He had been sick for 3 days with fever, which has now resolved and was followed by a pink maculopapular rash. What is your suspected diagnosis?
   a. Roseola
   b. Fifth disease
   c. Varicella
   d. Scarlet fever
   The correct answer is *a*.
2. What is a distinguishing feature of erythema nodosum?
   a. Rash changes locations on skin
   b. Dark scaling plaques with red center
   c. Does not respond to diphenhydramine
   d. Intensely itchy
   The correct answer is *c*.
3. Which is a sign of melanoma?
   a. Blanching erythema with pressure
   b. Smooth borders
   c. Rapidly changing lesion
   d. Dark brown color
   The correct answer is *c*.

CHAPTER 56

LIGHTNING AND ELECTRICAL INJURIES

Andrea Stember, MD, and Tracy Cushing, MD, MPH, FACEP

LIGHTNING INJURIES

1. What causes lightning?

Lightning is a large discharge of electrical energy. When warm and cold air meet within the atmosphere, the warm air rises and the cold air descends, forming cumulonimbus clouds, more commonly known as *thunderheads.* The air within a cloud contains water molecules. Where the air is warm, the molecules are in a liquid state; cold air forms ice crystals. The movement of these molecules within a convectively active cloud creates an electric charge and separation of polar (oppositely charged) ends of the cloud. Generally speaking, the upper portion of the cloud retains a positive charge, and the lower portion is more negatively charged. Air acts as an insulator between the opposite charges within the clouds, as well as between the cloud and the ground. Once the charge builds beyond the insulating capacity of the air, lightning is discharged, which equalizes the charged regions within the atmosphere. Lightning can discharge between opposite charges within a cloud, between clouds (cloud flashes), or between the opposite charges of the cloud and the ground (cloud-to-ground lightning). The potential between the cloud and ground can be up to 7500 volts per inch. When a cloud-to-ground strike occurs, it is initiated by the formation of a stepped leader, a path of negatively charged electricity descending from the cloud, in a series of short, zigzagged spurts, while simultaneously, a positively charged upward streamer is generated from the ground. When these currents meet at approximately 50 to 100 meters above ground, the return stroke is initiated, commonly known as the *lightning bolt.* Usually there are four to five return strokes, which are high-voltage, high-current, and high-velocity electrical discharges that strike objects within 30 to 50 m from the point of the upward streamer.

2. What is a "bolt from the blue"?

There is a phenomenon called *bolt from the blue,* which is a cloud-to-ground strike that travels a relatively large distance, sometimes through an apparently cloudless sky, to strike an object up to 25 miles away from the thunderstorm.

3. What causes thunder?

Thunder is an acoustic wave caused by lightning. The energy created in the electrostatic discharge of a bolt of lightning heats the surrounding air to greater than 50,000°F (27,760°C) within a few milliseconds, which creates a high-pressure region within the column of air. The pressurized air then expands outward, causing an acoustic sound wave that creates thunder.

4. How does lightning cause injury?

A bolt of lightning is a massive discharge of electrical energy with currents ranging from 30,000 to 110,000 A, which fortunately is only applied for milliseconds, thus limiting the energy transfer to the body. There are five types of lightning injuries.

1. Direct strike: An uninterrupted connection between the victim and the bolt of lightning
2. Contact injury: A transfer of electrical energy from touching an object that is struck directly
3. Side splash: An injury from current "splashing" or jumping from nearby objects to a victim's body
4. Ground current: When lightning strikes the ground or an object nearby and travels through the ground from the strike point to the victim
5. Upward streamer: When positively charged current passes up from the ground through the victim that does not connect with the pilot strike or stepped leader

Abstract

This chapter summarizes lightning and electrical injuries commonly encountered by emergency medicine providers.

Keywords:

lightning injuries, electrical injuries, lightning, electricity, ferning, ground, splash

In addition to the electrical injury induced from a lightning strike, there are also injuries from blunt or blast trauma and secondary burns. Common findings associated with blunt and blast trauma include tympanic membrane rupture, pulmonary contusions, loss of consciousness, fractures or dislocations, and clothing/shoes being blown off.

5. What types of injuries does lightning cause?

Injuries caused from lightning vary from minor to catastrophic. Nearly all organ systems can be affected (Table 56-1). The most catastrophic is sudden cardiac death, usually from a direct strike. Lightning electricity travels through the path of least resistance through body tissues. Nerves have lowest resistance, followed by blood, muscle, skin, fat, and then bone. See Table 56-2 for some common presentations of lightning victims based on injury severity.

Table 56-1. Lightning Injuries by Organ System

ORGAN SYSTEM	INJURIES
Cardiovascular	Injuries range from benign ECG changes to sudden cardiac death. Direct strikes are more commonly associated with asystole and sudden cardiac death. ECG changes include ST elevation, T-wave inversion, PR depression, QT prolongation, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation. Severe cardiomyopathy and cardiogenic shock may occur, as well as labile blood pressures and autonomic instability.
Respiratory	Pulmonary contusion, pulmonary hemorrhage, hemothorax, pneumothorax, apnea, hypoxia
Nervous	Injuries range from transient to permanent, and can have immediate or delayed onset. Transient symptoms include loss of consciousness, seizure, headache, paresthesias, weakness, confusion, disorientation, memory loss, autonomic dysfunction (including pupillary dysfunction), and keraunoparalysis (transient paralysis) after lightning strike. Keraunoparalysis is thought to be secondary to overstimulation of autonomic nervous system, which causes vascular spasm. Permanent symptoms include hypoxic encephalopathy, intracranial hemorrhage, and basal ganglia or brain stem injury. Delayed neurologic injuries include myelopathy and neuropathy.
Dermatologic	Lichtenberg figures (“feathering” or “ferning” pattern) are pathognomonic for lightning strike. Linear burns (partial-thickness burns from the vaporization of sweat into steam), or flashover, punctate burns (small, <1 cm, full-thickness circular burns from exit current), and thermal burns may be secondary to burning clothing, fabric, and surrounding environment.
Musculoskeletal	Fractures, dislocations, muscle necrosis, and compartment syndrome
Renal	Myoglobinuria can be seen, but rarely.
Ophthalmologic	Lightning strike may affect both anterior and posterior chambers, and can occur from current injury, blunt and blast trauma, vasoconstriction, and heat. The lens is the most commonly injured portion of eye, with cataracts being the most common injury. There may also be mydriasis, loss of light reflex, anisocoria, and Horner syndrome.
Otologic	Tympanic membrane rupture (secondary to blast trauma and electrical injury), tinnitus, hearing loss
Psychiatric	Depression, posttraumatic stress disorder, memory impairment, personality changes, storm apprehension, phobias

ECG, Electrocardiogram.

Table 56-2. Common Presentations of Electrical Injuries by Organ System

ORGAN SYSTEM	INJURY
Skin	Skin may show a variety of burns, usually thermal. Typically, in AC burns, the entrance and exit burns are similar in size and shape. In DC burns, the exit is larger than the entrance wound. Flexor crease burns and mouth commissure burns (this carries the risk of delayed bleeding from the labial artery when the eschar separates) may also be seen.
Cardiovascular	Patient may experience asystole or VF. Usually VF is caused by low-voltage AC, whereas asystole is caused by high-voltage AC or DC. Can also see atrial and ventricular ectopy, atrial fibrillation, first-degree and second-degree heart block, QT prolongation, bundle-branch blocks, and acute myocardial infarction (rare). Long-term cardiac complications are rare.
Vascular	Hemorrhage, venous and/or arterial thrombosis, vasospasm and ischemia, necrosis (presumed to be from skip lesions where the current skips from the blood to the vessel wall)
Nervous	Electrical burn can cause central or peripheral injuries, including transient amnesia, confusion, or loss of consciousness; and seizure, apnea, respiratory depression, paralysis, and paresthesias. Peripheral nerve damage (motor nerves injured more commonly than sensory nerves) has a poor recovery rate.
Musculoskeletal	Compartment syndrome, fractures, dislocations, muscular pain, muscle necrosis (leading to rhabdomyolysis), tendon rupture, "electroporation" (formation of cell membrane pores in bone), aseptic necrosis, and periosteal burns
Respiratory	Chest wall muscle spasm from tetanic contractions may cause respiratory arrest. Can also see respiratory arrest from inhibition of respiratory center within brain stem.
Gastrointestinal	Hollow organ and solid organ injury (rare); stress ulcers
Renal	Acute tubular necrosis (from rhabdomyolysis and myoglobinuria), renal failure, hyperkalemia, hypocalcemia, acidosis
ENT	Ruptured tympanic membranes, facial burns, cataracts (develop in approximately 6% of victims, usually 6-24 months after incident), corneal burns, intraocular hemorrhage, retinal edema, retinal detachment, uveitis, optic nerve atrophy
Genitourinary	Can see spontaneous abortion in pregnant victims (fetal death rate is 73%), oligohydramnios, and IUGR. (Amniotic fluid and fetal tissues conduct 200 times better than dry, intact adult skin.)

AC, Alternating current; *DC*, direct current; *ENT*, ear, nose, and throat; *IUGR*, intrauterine growth retardation; *VF*, ventricular fibrillation.

6. Is lightning direct current (DC) or alternating current (AC)?

Technically, it is neither, but it can behave in a manner similar to both DC and AC, depending on circumstance. It has a very high voltage (100 million to 2 billion V), very large current (20,000 to 300,000 A), and very high energy (1 billion J to 280 kW hours); however, it has a very short duration, lasting only 0.1 to 1 millisecond. It behaves most like DC.

7. Is it true that lightning never strikes twice in the same place?

No, contrary to popular belief, lightning can and does strike the same place twice. The Empire State Building in New York is struck around 23 times per year. In the United States, within the contiguous 48 states, an average of 20,000,000 cloud-to-ground strikes have been detected annually since 1989.

8. Is it true that I am safe from a lightning strike if I am in my car, because the rubber tires act as an insulator.

No, it is true that being inside a metal-topped car (rather than a convertible) with the windows and doors closed is safer than no shelter at all; however, you are not entirely protected. The metal body, rather than the rubber tires, offers some protection, by acting as a Faraday cage, which allows the flow of electricity around the exterior of the car, rather than through it. This does not, however, protect occupants from splash current or induced electromagnetic currents through the interior.

9. Am I safe from lightning if I am indoors.

No, again, being indoors is safer than no shelter at all, but a significant number of lightning injuries actually occur in people who are inside buildings. Side flash through plumbing, telephone wires, and electrical appliances that are connected to the exterior of the building account for this.

10. Does lightning ever hit airplanes? What are the consequences?

Yes, but fortunately, the consequences are minimal. On average, commercial airlines report that each aircraft is struck 1 to 2 times per year. Most strikes occur at an altitude of around 10,000 to 15,000 feet. Most aircraft skins are composed primarily of aluminum, which conducts electric current very well. The lightning flashes over the aircraft, leaving only minor, if any, damage. By ensuring that no gaps exist in the exterior, aircraft engineers are able to ensure that lightning flashes over the plane. The bright flash of lightning can temporarily blind pilots, and the electromagnetic effect produced by the flashover can temporarily interrupt aircraft lighting and aviation controls. The last confirmed commercial plane crash in the United States directly attributable to lightning occurred in 1967, when lightning ignited the fuel tank and caused a catastrophic explosion.

11. What happens to the ground when lightning strikes it?

When lightning strikes the ground, it tends to fuse dirt and clay into silica. This results in a black, glassy rock called a *fulgurite,* which usually takes the shape of a convoluted tube. The shape of the tube is the shape of the path the lightning current followed within the ground. When lightning strikes a tree, it turns the water within the wood to steam, often causing an explosion of bark and wood pieces from the rapidly expanding steam.

12. How common is lightning? How common are injuries or deaths?

Lightning strikes occur approximately 50 times per second worldwide; approximately one fifth of the strikes result in cloud-to-ground strikes. Within the contiguous 48 states of the United States, there are an average of 20,000,000 cloud-to-ground strikes annually. Internationally, there are around 24,000 fatalities per year as a result of lightning. Within the United States, the incidence of lightning-related fatalities has been declining over the past 50 years. Currently, lightning causes approximately 40 deaths per year, and there are an estimated 400 lightning-related injuries.

13. Who tends to get struck by lightning? Where do most strikes occur?

Males are at a higher risk of being struck by lightning than females. One demographic study revealed that greater than 80% of lightning-strike victims are male. Most victims are between the ages of 20 and 45 years, and more than 90% of deaths occur between May and September. If you live or recreate in Florida, Colorado, or Texas you are at the highest risk. Within the United States, the lifetime risk of getting struck by lightning is around 1 in 10,000. Activities commonly associated with lightning strikes include hiking, boating, fishing, swimming, golfing, farming, operating heavy equipment, and land-line telephone use.

14. What factors predispose someone or something to be struck by lightning?

Several factors predispose victims and/or objects to lightning strikes, including:

- Vicinity to a thunderstorm (essentially if you can hear thunder, you are at risk)
- Isolation
- Height of person or object
- Shape of object (objects with pointed tops are at increased risk)

15. I am treating a hiker who was found unconscious on the trail after a thunderstorm had passed. The patient has no memory of what happened. How can I tell if he was struck by lightning?

Several physical examination findings can help you make the diagnosis. First, examine his skin, looking for "fern" or "feathering" patterns, known as *Lichtenberg figures*. These skin findings are

pathognomonic for a lightning strike. The skin findings will usually be present within 1 hour of the lightning strike and resolve over several hours. Unfortunately, Lichtenberg figures are only present in about 20% of confirmed lightning strikes. However, if they are present, you have made the diagnosis. Next, you should examine for evidence of other burns on his skin, as well as his clothing, shoes, and equipment. Examine ears for evidence of tympanic membrane rupture, which occurs in approximately 50% of lightning strikes. See Table 56-1 for additional physical examination findings.

Regarding the victim's memory, it is reported that 100% of direct-strike victims have no recollection of the event. Victims of indirect strikes may initially have some recollection of the events; however, they often develop anterograde amnesia.

16. The hiker has regained a palpable carotid pulse, but he does not appear to be breathing on his own. Why? What should I do?

The primary cause of death in lightning-strike victims is cardiac arrest, caused by a sudden depolarization of the entire myocardium. Often, the intrinsic cardiac automaticity will spontaneously restore organized cardiac activity along with a perfusing cardiac rhythm. However, there is simultaneous respiratory arrest as a result of spasm of the thoracic muscles and stunning of the central nervous system (CNS) respiratory center. You should continue to give ventilatory support, in the form of rescue breathing, or a secondary hypoxic cardiac arrest will occur.

17. The hiker is also tachycardic, hypertensive, and has cool, pale skin with diminished peripheral pulses. He is awake, but unable to move his extremities. Why?

The victim is most likely experiencing keraunoparalysis, which is a transient paralysis after a lightning strike. It is to be secondary to overstimulation of the autonomic nervous system, which causes vasospasm and hypoperfusion, leading to the symptoms mentioned in the question. The symptoms usually resolve within several hours; however, remember to be wary of occult injuries, including traumatic spinal cord injuries, in the workup of patients who have been struck by lightning.

18. During mass casualty training, responders are taught to allocate resources to victims who are not breathing or moving only after they have taken care of victims with obvious signs of life. Should this same practice apply when there are multiple victims of a lightning strike?

No, lightning strikes are the exception to the mass casualty triage principle. In the situation of a lightning strike, first priority should go to victims who are not breathing or moving. This is because for victims who are in cardiac arrest, the risk of death is the highest. For victims without cardiopulmonary arrest, there is little chance of dying, which means in this situation, the first priority is patients in cardiopulmonary arrest.

19. Is prolonged cardiopulmonary resuscitation (CPR) beneficial in lightning-strike victims?

No, there is no evidence to support prolonged CPR or to suggest that it improves survival. If reversible causes of cardiopulmonary arrest have been ruled out, it would be reasonable to stop CPR after 20 to 30 minutes, just as you would do in a non–lightning-strike situation.

20. I am performing CPR on a lightning-strike victim when one of my colleagues notices that the victim has fixed and dilated pupils. Should I stop CPR based on these findings?

No, in the case of lightning strikes, fixed and dilated pupils cannot be used as an indicator of brain death or to gauge prognosis. Lightning strikes can cause autonomic dysfunction, including an abnormal pupillary response.

21. Do victims of lightning strike typically suffer extensive burns?

No, contrary to popular folklore, victims do not burst into flames and turn into a pile of ash. In fact, of the victims who do suffer thermal burns, only 10% require skin grafting. Lightning usually flashes over a victim, with few, if any, deep-tissue burns. Victims with burns usually have linear, punctate, or partial-thickness thermal burns. Of note, victims with cranial burns have a threefold increase in mortality rate and are twice as likely to have cardiac arrest as victims with burns elsewhere.

22. What are the best ways to prevent lightning-related injury or death?

- Behavioral strategies: Choose safer places when there is a risk of lightning strike. "When thunder roars go indoors" is the current recommendation from the National Weather Service.

Table 56-3. Comparison of Lightning with High-Voltage and Low-Voltage Electrical Injuries

	LIGHTNING	HIGH-VOLTAGE INJURY	LOW-VOLTAGE INJURY
Voltage (V)	$>30 \times 10^6$	>1000	<600 (<240)
Current (A)	>200,000	<1000	<240
Duration	Instantaneous	Brief	Prolonged
Type of current	DC	DC or AC	Mostly AC
Cardiac arrest (cause)	Asystole	Ventricular fibrillation	Ventricular fibrillation
Respiratory arrest (cause)	Direct CNS injury	Indirect trauma or tetanic contractions of respiratory muscles	Tetanic contractions of respiratory muscles
Muscle contraction	Single	DC: single; AC: tetanic	Tetanic
Burns	Rare, superficial	Common, deep	Usually superficial
Rhabdomyolysis	Uncommon	Very common	Common
Blunt injury (cause)	Blast effect; shock wave	Muscle contraction, fall	Fall (uncommon)
Mortality (acute)	Very high	Moderate	Low

AC, Alternating current; *CNS*, central nervous system; *DC*, direct current.

- Shelter: Seek shelter in the center of the largest, most substantial building available. Avoid use of land-line telephones and electrical appliances. You can also seek shelter in a metal-topped vehicle; be sure to close the windows and doors. If outdoors and no shelter is available, seek shelter deep in a cave, far into a dense forest, or deep ravine. Avoid shallow caves, open picnic shelters, or solitary trees because of the risk of ground current and side splash. Wait a minimum of 30 minutes after hearing the last thunder clap before resuming outdoor activities.
- Lightning position (to be used only if a strike is imminent/unavoidable): Sit or crouch with knees and feet close together, creating only one point of contact with the ground.
- Avoid groups: Separate members of a group by at least 20 feet to limit possibility of ground current and side splash

23. What are the differences between lightning and low- and high- voltage injuries?

See Table 56-3.

ELECTRICAL INJURIES

24. What are the basic physics of electricity?

Simply put, electricity is the flow of charged particles, known as *electrons.* The electric potential difference between two points is known as *voltage.* The number of electrons flowing past a specific point is known as *electric current,* and it is measured in amperes. The resistance to electron flow is a property of the material through which the electrons are flowing, and it is measured in ohms. These factors are related with Ohm's Law, which states that current equals voltage divided by resistance.

25. What is an easy way to classify electrical injuries? Does this help determine the nature and severity of electrical injuries?

Electrical injuries can be divided into two categories: high voltage (>1000 volts) and low voltage (<1000 volts). The most harmful effect of any type of electrical injury is the thermal effect, causing thermal burns. Heat generated by electricity is related to the current, tissue resistance, and duration of contact. Current contributes most to tissue injury; however, in reality, voltage is often used as a surrogate for amperage (high voltages are usually associated with high amperages).

26. How does the type of circuit relate to injury?

Electrical current flows in two types of circuits: DC and AC. AC is the most common type of electricity in homes and is supplied in either 120 V or 240 V. High-voltage (>1000 V) DC can cause large single-muscle contractions, resulting in the victim being thrown away from source. This results in a short duration of contact. High-voltage AC is much more dangerous, because the cyclic flow of current causes muscle tetany and prolonged exposure to the source.

27. What types of electrical burns are there?

Injury patterns depend on voltage, current, pathway of the current, duration, and type of circuit involved.

28. What is the epidemiology of electrical injuries?

There are approximately 500 to 1000 deaths per year in the United States from electrical injuries. High-tension electrical injuries account for approximately 7% of burn unit admissions. In the pediatric population, electrical injuries cause 3% to 5% of burn unit admissions and 2% to 3% of ED visits. The mortality rate of all electrical injuries is between 3% and 15%. Serious electrical burns have a mortality rate of up to 40%. In victims of low-voltage electrical injuries who do not suffer immediate cardiac death, mortality is low, but there may be significant morbidity, including oral trauma in children and hand trauma in adults.

In general, there is a bimodal age distribution for electrical injuries, with a peak in young children, and another around age 20. The peak in children is secondary to developmental stages in which children are exploring electrical outlets and cords. The peak around age 20 is because of occupational exposures and accidents, with electrical and construction workers accounting for a large percentage of these incidents. Electrical injury is the fourth leading cause of occupational deaths in the United States.

29. What should I do if I am a responder to the scene of an electrical injury?

The first priority is to not become a victim yourself. Take time to ensure scene safety. Be sure to turn off all power sources that pose a threat to the rescuers or the victim. As with any trauma, airway, breathing, and circulation (ABCs) are the mainstay of initial resuscitation. Assume trauma has occurred, and consider immobilizing the spine if there is a high suspicion for spinal injuries. Cardiac arrest from electrical injury is treated with CPR and general resuscitation principles. Additionally, given the propensity for large burns and extensive tissue damage, fluid resuscitation should begin early.

30. How does tissue resistance relate to electrical injury?

Within the body, different tissue types have different resistance, which affects how electric currents travel through them, resulting in different patterns of injury. Nerves have the least amount of resistance, meaning that electrical currents penetrate the deepest through nerves but cause them the least amount of heat injury. Blood and blood vessels have the next least resistance, followed by mucous membranes, muscle, skin, tendons, fat, and then bones. With the highest resistance in bone, it has the least penetration but the most thermal injury.

Of note, skin resistance is variable depending on thickness, moisture content, and vascularity. Thick, dry, calloused skin, as on feet and hands, is much more resistant than thin, wet skin. Skin that is immersed in water has even lower resistance. When exposed to electrical energy, the thick skin of the hands and feet, with its high resistance, experiences thermal burns. As the skin is burned and charred in these areas, resistance drops, allowing for deeper penetration of electrical current and more extensive injury.

31. Which organ systems are affected by electrical injury? What types of injuries occur?

See Table 56-2.

32. What are the most common long-term complications of electrical injuries?

Electrical injury can cause serious long-term sequelae, including neurologic symptoms such as numbness, weakness, paresthesias, memory problems, and chronic pain. Psychiatric symptoms include anxiety, poor concentration, depression, nightmares, insomnia, and flashbacks or posttraumatic stress disorder (PTSD). Victims of high-voltage injury have significantly more complications related to contact burns.

33. An ambulance arrives at the ED with a 22-year-old, 75-kg man who was working on a ladder near a high-voltage electrical line when he received a shock. He has two burns: one on his palm, where it contacted the wire, and one on his shin, where he was touching the ladder. Together, these burns are about 2% of his total body surface area (TBSA). How much intravenous (IV) fluid should I give him?

Burn victims are usually resuscitated using a formula, such as the Parkland formula (4 mL × weight in kg × TBSA) to determine fluids; however, these equations do not apply in situations of electrical burns. This is because the surface damage does not reflect the degree of deeper tissue damage. In this scenario, fluid replacement should be administered at a rate that produces 1 to 2 mL/kg/h of urine output (75 to 150 mL/h for this patient). Fluids should be given early and aggressively in the resuscitation of patients with electrical burn, to prevent renal failure secondary to rhabdomyolysis.

34. I am caring for a patient after she suffered an electric shock. She has no recollection of the event, and she complained briefly of paresthesias. Should I obtain a computed tomography (CT) scan of the head, or is close observation enough?

You should consider a head CT scan in any victim of high-voltage electrical injury who has CNS symptoms on presentation or shortly after the event. Neural tissue has low resistance, and thus conducts electricity well. Subarachnoid hemorrhage can occur as a result of high-voltage shocks and needs to be ruled out. Additionally, every patient with electrical injury should be evaluated as a trauma patient; thus CT of the head may be indicated to rule out other intracranial injuries as a result of blunt head trauma.

35. Can I think of victims of high-voltage electrical injury as similar to lightning-strike victims?

No, lightning strikes and high-voltage electricity cause different injury patterns and thus require a different approach. High-voltage injuries often cause deep burns that may lead to rhabdomyolysis and renal failure, require aggressive fluid repletion, and may need further interventions, such as fasciotomies, if a compartment syndrome develops (Table 56-4). Victims of high-voltage electrical injury, if in cardiac arrest, will often exhibit ventricular fibrillation. In contrast, victims of lightning rarely have deep or severe burns, rarely need aggressive fluid resuscitation, and generally do not require fasciotomies. Patients who have been struck by lightning also rarely develop kidney failure, and if in cardiac arrest, it is more likely to be asystole from massive cardiac depolarization.

36. Are there any medications to consider for electrical or lightning injury victims?

There is no pharmacotherapy indicated specifically for lightning or electrical injuries. In patients with electrical injury who have rhabdomyolysis, aggressive fluid resuscitation with normal saline is beneficial. Forced diuresis with mannitol or loop diuretics may be beneficial to help clear myoglobin in the setting of rhabdomyolysis as well. Sodium bicarbonate can also be used to alkalize the urine, which in turn increases myoglobin clearance.

37. How should I triage victims of lightning-strike or electrical injury on scene?

All victims of lightning strike should be transported to an ED on a cardiac monitor.

38. Who needs to be admitted for lightning-strike or electrical injuries?

Any patient with cardiac abnormalities (detected in the field or the ED), neurologic findings, or significant burns warrants hospital admission. See Table 56-5 for admission and cardiac monitoring indications. Patients who meet admission criteria and who have suffered a high-voltage or lightning strike injury should receive cardiac monitoring for 24 hours. Patients who suffered low-voltage injuries and demonstrated electrocardiogram (ECG) abnormalities initially should also receive cardiac monitoring. Patients with burns should be stabilized and then considered for transfer to a burn unit. Patients should be treated appropriately for any associated traumatic injuries. Asymptomatic patients with a responsible party willing to take them home may be discharged from the ED with appropriate follow-up instructions as needed. Patients with lightning-strike injury should be referred for an ophthalmologic follow-up appointment within 6 months, as well as otolaryngologic, neurologic, and psychiatric follow-up care as needed.

Table 56-4. Type of Exposure and Initial Presentation of Electrical Injuries

TYPE OF EXPOSURE	PRESENTATION
Low-voltage AC without LOC and/or cardiac arrest	<1000 V exposure; usually in home/office setting. Children typically are seen after biting cord; generally suffer oral burns. Adults come to ED with hand burns after working on home appliances. May have significant injuries if prolonged exposure with tetanic muscle contractions.
Low-voltage AC with LOC and/or arrest	Consider respiratory arrest secondary to thoracic muscle spasm and/or cardiac arrhythmias; consider whenever unwitnessed arrests occur.
High-voltage AC without LOC and/or arrest	Devastating thermal burns
High-voltage AC with LOC and/or arrest	Rare; usually no LOC or arrest
DC injury	Typically single-muscle contraction that throws victim from source. Rarely associated with LOC, unless secondary head trauma. Victims can usually remember what happened.
Conducted electrical devices/weapons (CEWs)	Example: Taser gun used in law enforcement. Delivers high-voltage current, is neither true AC nor DC, but is more like a series of low-amplitude DC shocks. No evidence of cardiac arrhythmias or death in healthy volunteers.

AC, Alternating current; *DC*, direct current; *LOC*, loss of consciousness.

Table 56-5. Criteria for Admission and Cardiac Monitoring Versus Discharge

ADMIT (CONSIDER TRANSFER TO BURN OR TRAUMA CENTER)	DISCHARGE
Cardiac arrest/required CPR	Asymptomatic low-voltage injury
Documented loss of consciousness	No ECG findings
Abnormal ECG; dysrhythmia observed in prehospital or ED setting	No significant burns
Presence of significant risk factors for cardiac disease	*Ophthalmology follow-up examination in 6 months for lightning injury
Concomitant injury severe enough to warrant admission	*ENT, neurology, psychiatry follow-up examinations as needed
Suspicion of conductive injury: High-voltage injury, especially with transthoracic current path	*Close ENT or plastic surgery follow-up monitoring and counseling about eschar formation in mouth commissure burns in pediatric patients
Hypoxia	
Chest pain	
Neurologic abnormalities	
Major burns; circumferential burns, hand/face/groin burns	
Myoglobinuria	
OB/GYN consult for pregnant patients	

CPR, Cardiopulmonary resuscitation; *ECG*, electrocardiogram; *ENT*, ear, nose, and throat; *OB/GYN*, obstetric, gynecologic.
*If discharged, these referrals and follow-up arrangements should be made.

39. What laboratory result can be used to determine patients who may benefit from early fasciotomy, in an attempt to prevent future amputations from electrical burns?

Elevated serum creatinine kinase levels can be used as prognostic factor and is one laboratory result that can help determine the need for early surgical decompression via fasciotomy to prevent future amputation and limb loss in high-voltage burn victims.

40. What about children who get injured by a household electrical cord or appliance? Should I admit them to the hospital for observation, or can I discharge them home from the ED?

Most often, children sustain electrical injuries in the home. These injuries are usually associated with electrical cords (60% to 70%), with contact to an extremity or the mouth, or with wall outlets (15% to 20%). For healthy children who are exposed to common household currents (120 to 240 V) without water contact, you may discharge them home as long as they were asymptomatic at presentation, with no evidence of ventricular arrhythmia or cardiac arrest in the field. Children who are asymptomatic at presentation are at very low risk of developing cardiac arrhythmias, and those with nonfatal arrhythmias or nonspecific ECG abnormalities typically resolve within 24 hours without intervention. Therefore the recommended practice is that healthy children may be discharged home after exposure to common household electrical currents without initial screening ECG or admission for cardiac monitoring.

41. What about pregnant patients who sustain electrical injuries?

When a fetus is exposed to electrical current, just as in adults, it is at risk of cardiac arrest. The risk may be higher in a fetus, however, because fetal tissue offers less resistance than postnatal tissue. Fetal skin is 200 times less resistant than postnatal skin, and according to one study, exposure of the fetus to 100 to 380 V for 0.3 seconds may be lethal. When considering the risk to the fetus, the critical consideration is the current path, and whether the current passed through the uterus. This is most often seen in hand-to-foot passage. When caring for a pregnant patient after an electrical injury, care for the mother as you would in any other scenario. Regarding the fetus, it is recommended that fetal heart Doppler monitoring should be done. An ultrasound examination should be performed if the patient has not previously had one, to document an intrauterine pregnancy and for heartbeat if Doppler monitoring is not available. Pregnant patients should be monitored for 24 hours if there are any signs of fetal distress or decreased fetal movement/fetal demise, and upon discharge should be counseled about close follow-up observation with their obstetrician.

KEY POINTS: LIGHTNING AND ELECTRICAL INJURIES

1. Traditional triage rules do not apply to lightning victims; remember to reverse triage and concentrate on victims who appear to be in cardiopulmonary arrest.
2. Secure the scene; rescuers should not become victims.
3. Assume occult trauma and remember that entrance/surface burns may be indicators of underlying tissue damage. It is not possible to predict the degree of underlying tissue damage based on the extent of cutaneous injury.
4. AC exposure is far more dangerous than DC exposure of the same voltage, secondary to the potential for tetanic muscle contractions and prolonged contact.

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Acknowledgment

The authors and editors gratefully acknowledge the contributions of Gabrielle A. Jacquet and Timothy R Hurtado, authors of this chapter in previous editions.

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QUESTIONS

1. What is one way to prevent lightning-related injuries and fatalities?
   a. When outdoors with a group of people, remain in close contact, with less than 10 feet separating group members.
   b. If a lightning strike is imminent, lay flat on the ground, in the lightning position.
   c. Ensure that at least 10 minutes has passed after the last lightning strike before returning outside.
   d. Seek shelter in the center of the largest, most substantial building available.
   The correct answer is *d*.
2. What laboratory result can be an indicator that early fasciotomy may be necessary in patients with electrical burn?
   a. Elevated serum potassium
   b. Elevated serum creatinine kinase
   c. Elevated serum creatinine
   d. Elevated serum phosphorous
   The correct answer is *b*.
3. Which of the following is true regarding lightning and/or electrical injuries?
   a. You can treat victims of high-voltage electrical injury the same as you would treat victims of lightning strike.
   b. Patients who suffer low-voltage injuries and demonstrate ECG abnormalities should receive cardiac monitoring.
   c. When in cardiac arrest, patients with high-voltage electrical injury will often exhibit asystole as the presenting arrhythmia.
   d. Victims of lightning strike commonly develop kidney failure as a result of their injuries
   The correct answer is *b*.

DROWNING

Andrew Schmidt, DO, MPH, and Jedd Roe, MD, MBA, FACEP

CHAPTER 57

1. Define *drowning.*

In 2002, the World Congress on Drowning developed the following standard definition for drowning: *The process of experiencing respiratory impairment due to submersion or immersion in a liquid.* From this definition, there are three possible outcomes: no morbidity, morbidity, or mortality. In addition, recommendations were made to discontinue the use of the following descriptive terms: *near, dry, wet, active, passive,* and *secondary.* These terms only serve to add confusion and do not hold any clinical value.

2. How many people drown each year?

Each year in the United States, more than 4000 people die from drowning (an estimated 500,000 worldwide), and it is the second leading cause of injury death in children and teenagers ages 1 to 19 years. Most important, there are an estimated 8 million nonfatal drownings worldwide each year, many of which may lead to severe morbidity.

3. Who drowns, and why?

Drowning is primarily a disease of youth, and the incidence peaks in two groups: toddlers and teenagers. The most vulnerable group is toddlers (ages 1 to 4 years); they are at risk because of their inherently inquisitive nature and 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 years. In people ages 15 to 24 years, nearly 80% of drowning victims are male. Young males are often victims because of risk-taking behavior during swimming, boating, diving, or other water-related activities, and alcohol is a contributing factor in more than 60% of all teenage and young adult drownings.

Other risk factors in all age groups are as follows:

- Inability to swim
- Seizure disorder
- Cardiovascular disease
- Long QT syndrome
- Substance abuse
- Trauma (diving in shallow water, boating)
- Hypothermia
- Freediving training (hyperventilation)

4. What kills a drowning victim?

The primary cause of morbidity and mortality in drowning is hypoxia. Historically, a misguided focus has been placed on the type of water aspirated (salt versus fresh), the volume of water aspirated, and whether laryngospasm occurred. In reality, the end point is always hypoxia, no matter what pathophysiologic events lead up to it. In addition, the actual volumes of water commonly aspirated are much smaller than originally postulated. Therefore a resuscitative strategy focused on expelling water from the lungs, instead of reversing hypoxia, will do more harm than good.

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 inhales 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 (CNS) damage begins within minutes.

Abstract

Drowning is a leading cause of accidental death around the world, especially in children and young adults. More important, it is a disease that can be prevented through improved public education and treated appropriately through a simple understanding of the underlying pathophysiology. The primary cause of systemic injury in drowning patients is hypoxemia, and its correction should be the focus of prehospital and ED resuscitation and treatment. Once the condition is stabilized, the disposition of a patient can be determined by physical examination findings and response to treatment, with patients displaying mild symptoms able to be safely discharged after a short period of observation. This chapter provides a simple overview of what drowning is and is not, the role of hypoxemia and initial steps to reverse it, myths surrounding drowning that get in the way of effective treatment, and evidence to help guide disposition and predict outcome.

Keywords:

drowning, submersion, immersion, hypoxemia, hypothermia

6. Describe the presenting symptoms of 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 CNS 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 partial pressure of oxygen (PO_2) decreases, the partial pressure of carbon dioxide (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) caused by 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 (most commonly asystole or pulseless electric activity [PEA]) are caused by hypoxemia and complicated by the ensuing acidosis. The heart is relatively resistant to hypoxic injury, and with proper resuscitation resumption of cardiac activity is common, although severe CNS 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 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 can be avoided, and prompt and full recovery may be possible. 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 as rapidly as possible. Establish a patent airway and administer oxygen with positive pressure ventilation as indicated by the patient's condition. Although cervical spine immobilization is important for suspected spinal injury, the incidence of this in drowning cases is very low and most often suggested by facial trauma, intoxication, or eye-witness accounts. Cervical spine immobilization should never delay proper ventilation in the critical patient. If the patient is pulseless, cardiopulmonary resuscitation (CPR) (with ventilations) should be initiated using advanced cardiac life support (ACLS) protocols.

Note: There is no evidence supporting the use of abdominal thrusts or 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. Early airway management with positive pressure ventilation and positive end-expiratory pressure is appropriate to decrease intrapulmonary shunting. If there is high suspicion for cervical spine injury (witnessed diving or fall from height, known ethanol ingestion, facial trauma), appropriate precautions should be taken with in-line stabilization, as long as it does not delay 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 initial chest radiograph does not predict extent of injury or clinical course. Findings on chest radiograph vary, but patients with severe injury often display a pattern similar to that seen in ARDS.

13. Is there a role for prophylactic antibiotics?

When highly contaminated water is involved (e.g., sewage), prophylactic antibiotics may be considered. In all other instances, prophylactic antibiotics are of no proven benefit. Their use is indicated after initial resuscitation in the presence of clinical evidence suggesting pneumonia, and should be guided by bronchoalveolar sampling.

14. Is there an indication for the use of sodium bicarbonate during resuscitation?

There is no good evidence to support using sodium bicarbonate during resuscitation.

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, as long as the treatment of such does not delay airway support. 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 been shown to be of benefit in cardiac arrest, and case reports have suggested similar outcomes for victims of submersion. This should be initiated only after proper resuscitation, with a focus on ventilation, and only in facilities with the proper policies, equipment, and training to initiate and maintain the hypothermia for 24 to 48 hours.

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 drowning victims should be continued until patients are adequately rewarmed or to the level required for therapeutic hypothermia (see Chapter 58). Of note, extracorporeal membrane oxygenation (ECMO) has received increased attention in the literature for the treatment of drowning patients. If available, this may be considered for severe or refractory hypothermia or hypoxemia.

18. When should resuscitative efforts be withheld?

In general, all patients should receive initial resuscitative efforts. There are numerous reports of survival after prolonged submersion, especially in children who have drowned in cold water, although these are by no means the usual circumstance. In general, a submersion time longer than 10 minutes or resuscitation time longer than 25 minutes has been shown to correlate with death or survival with poor neurologic outcome. Historically, the philosophy has been to resuscitate the victim until the core temperature is normalized. Whereas this may hold some value, it is important to acknowledge the often devastating neurologic outcomes patients and families have to live with after prolonged submersion and resuscitation.

19. What is the disposition of a submersion victim?

All submersion victims with cardiac arrest deserve aggressive prehospital and in-hospital resuscitation efforts, with a focus on reversing hypoxia. All other submersion victims require close observation. Some respiratory complications of drowning are delayed in presentation and usually appear within 4 to 6 hours. After initial resuscitation and stabilization, any patient with continued respiratory complaints or symptoms, altered mentation, chest radiograph abnormalities, or a demonstrated oxygen requirement should be monitored closely in a hospital for at least 24 hours. Patients without any symptoms and completely normal evaluation may be discharged after 4 to 6 hours of observation with instructions to return immediately if respiratory distress ensues.

Table 57-1. 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

20. What are the most important factors in estimating prognosis?

The most consistent prognostic indicator found in the literature is duration of submersion, and this highlights the pivotal role that hypoxia plays in the injury process. Other factors that have been found to have some prognostic value are:

- Delay in initiation of CPR
- Delay in arrival of emergency medical services (EMS)
- Need for prolonged resuscitation
- A Glasgow Coma Scale score less than or equal to 5
- pH less than 7
- Asystole on arrival to the ED

Dr. David Szpilman has proposed a clinical classification based on the analysis of 1831 cases of submersion seen in Brazil over 19 years. The classification is based on clinical findings in the field, and the mortality rates are shown in Table 57-1.

21. Can we prevent drowning?

Many of the factors contributing to death by drowning are preventable and can be directed at those groups at risk, particularly children. Efforts include:

- Participating in swim lessons
- Fencing of private and public swimming pools
- Using 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.

KEY POINTS: SUBMERSION INCIDENTS

1. Toddlers and teenagers are most at risk for death from submersion.
2. Prehospital treatment is critical and directed at correcting underlying hypoxia.
3. A normal chest radiograph does not rule out pulmonary injury.
4. Asymptomatic drowning victims can often be safely discharged after 4 to 6 hours of observation.
5. Most drownings are preventable.

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QUESTIONS

1. What is the most important goal the resuscitation of a drowning patient?
   a. Reversal of hypothermia
   b. Reversal of hypotension
   c. Reversal of hypoxemia
   d. Reversal of hyperkalemia
   The correct answer is *c*.
2. In a drowning patient who comes the ED with altered mental status, vomiting, and an arterial oxygen saturation level (SaO_2) of 85% on room air, which is the preferred method for oxygen delivery?
   a. Nasal cannula
   b. Nonrebreather mask
   c. Noninvasive positive pressure ventilation
   d. Mechanical ventilation through endotracheal tube
   The correct answer is *d*.
3. Which of the following statements about the ED treatment of drowning is true?
   a. A normal initial chest radiograph alone has little prognostic value.
   b. Steroids and antibiotics have proven survival benefit.
   c. A patient with a normal initial head computed tomography will have a good neurologic outcome.
   d. The most common initial pulseless dysrhythmia is ventricular fibrillation.
   The correct answer is *a*.

CHAPTER 58

HYPOTHERMIA AND FROSTBITE

Martin R. Huecker, MD, and Daniel F. Danzl, MD

HYPOTHERMIA

1. What is accidental hypothermia?

Accidental hypothermia is 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 rate 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 cannot 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 to 35°C [90°F to 95°F]) depresses the central nervous system (CNS) and increases the metabolic rate, pulse, and amount of shivering thermogenesis. Dysarthria, amnesia, ataxia, and apathy are common findings.
- Moderate hypothermia (27°C to 32.2°C [80°F to 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 factors predispose a patient to hypothermia?

- Decreased heat production
- Increased heat loss
- Impaired thermoregulation

6. What decreases heat production?

Decreased heat production is common with the following conditions:

- Age extremes
- Inadequate stored fuel
- Endocrinologic or neuromuscular inefficiency

Abstract

Accidental hypothermia is described as an unintentional decrease in core temperature to less than 35°C. Body temperature is regulated by many mechanisms, and heat is dissipated by radiation, conduction, convection, respiration, and evaporation. Symptoms vary from apathy and amnesia to coma, as patients progress from mild to severe hypothermia. Rewarming measures must be initiated as early as possible, with active rewarming (including extracorporeal membrane oxygenation [ECMO]) indicated for more severe cases. Frostbite occurs when tissue temperature decreases to 0°C. Acral tissues are affected first, with multiple tissue changes culminating in cellular death. Frozen tissues should be thawed in 40°C to 41°C circulating water and should not be exposed to refreezing. Demarcation of tissue death may take months.

Keywords:

accidental hypothermia, heat loss mechanisms, rewarming techniques, frostbite, nonfreezing cold injury

Neonates are poorly adapted for cold, even without being subjected to emergent deliveries and resuscitations. The elderly have progressively impaired thermal perception. Anything from 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 heat stroke treatment.

8. How is thermoregulation impaired?

Impairment is via central, peripheral, metabolic, or pharmacologic mechanisms. A variety of CNS 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 core 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 CNS 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, alcohol intoxication, or CNS 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 hemagglutination 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 (INR), 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 a partial pressure of carbon dioxide (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 less than 32.2°C (90°F)
- Age extremes
- Neurologic or endocrinologic insufficiency

16. What is core temperature afterdrop?

Core temperature afterdrop is the commonly observed continued drop in core temperature after initiation of rewarming. There are two causes.

1. Temperature equilibration between tissues
2. 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?

Active core rewarming involves 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 method 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 patients who are severely hypothermic and unstable, or for 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. Preexistent 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 (32°F). Ice crystal formation damages the cellular architecture, and stasis progresses to microvascular thrombosis.

27. Which factors predispose a patient 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 a person 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 postinjury may demonstrate soft-tissue swelling or frank bony destructions. 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. However, liquefaction gangrene is a more common sequela 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 to 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 circulating water at 40°C to 41°C 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 aspirated under sterile conditions. After debridement, apply antibiotic ointment or a specific thromboxane inhibitor, such as 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 beneficial?

A variety of vasodilatory treatment regimens, including medical and surgical sympathectomies, dextran, heparin, and a variety of antiinflammatory 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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QUESTIONS

1. Accidental hypothermia is defined as an unintentional depression of the core temperature below:
   a. 33°C
   b. 34°C
   c. 35°C
   d. 36°C
   The correct answer is *d*.
2. Which of the five mechanisms of heat loss is usually the greatest?
   a. Radiation
   b. Conduction
   c. Respiration
   d. Convection
   e. Evaporation
   The correct answer is *a*.
3. The correct technique to treat both superficial and deep frostbite is:
   a. Slow, gradual thawing with dry radiant heat
   b. Rapid thawing with radiant heat source
   c. Vigorous massage during room ambient temperature thawing
   d. Rapid thawing in a tepid water bath at 40°C to 42°C
   e. Rapid thawing in a hot bath at 46°C
   The correct answer is *d*.
4. Frostnip, pernio, and immersion foot are examples of:
   a. Frostbite
   b. Chilblain
   c. Trench foot
   d. Nonfreezing cold injury
   e. Cold-induced vasodilation
   The correct answer is *d*.

HEAT ILLNESS

Christopher B. Colwell, MD, and Elena Garcia, MD

CHAPTER 59

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. Information on body temperature is transmitted from cutaneous and core receptors to the hypothalamus, which then sends signals to regulate increase or decrease blood flow to the periphery (for cooling) or to the core (for warming). This is a critical function, because human body systems operate under a narrow range of temperatures (35°C to 38.2°C [95°F to 100.8°F]). In addition, the sympathetic system regulates sweat production, which helps to cool the body.

2. What are the four mechanisms for heat dissipation?

1. Conduction: Passive transfer of heat from one object (the body) to another through direct contact via a temperature gradient
2. Convection: Heat loss to air or water molecules circulating around the body
3. Radiation: Heat transfer by electromagnetic waves; accounts for 65% of heat loss in cool environments and is a major source of heat gain in hot climates
4. Evaporation: Conversion from a liquid to a gaseous phase (e.g., sweat)

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. At humidity levels exceeding 75%, sweat evaporation ceases.

5. Is there any way to predict heat stress?

Wet bulb globe temperature (WBGT) accounts for absolute temperature, radiant heat absorption, and humidity when recording a heat index. This and other measurements are used on a larger scale to predict hyperthermia occurrence in a given environment and can inform whether an activity should be attempted or not (i.e., whether a competitive event, such as a marathon, should be postponed).

6. How does heat harm 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 syndrome (SIRS). In addition, heat directly injures the vascular endothelium, causing increased vascular permeability, activation of the coagulation cascade, and disseminated intravascular coagulation (DIC). Heat also may accelerate biochemical reactions, which in turn may cause metabolic abnormalities.

Temperatures greater than 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 can cause the same degree of damage as higher temperatures over shorter periods, and increases in humidity can both worsen the heat index and impair the body's ability to dissipate heat.

7. Why is this epidemiologically important?

In the United States, there are on average more than 600 deaths annually attributed to excessive heat exposure. Heat-related illness is the leading cause of morbidity and mortality among high school athletes in the United States.

Abstract

This chapter reviews heat illness and the various types of heat-related problems that may be encountered in the ED. Pathophysiology and treatment options are also reviewed.

Keywords:

heat illness, heat stroke, evaporative cooling

8. Why might someone be unable to dissipate heat appropriately?

- Increased energy production (e.g., exercise, delirium, seizures, fever, sympathomimetic drugs, thyroid storm)
- Damaged conducting system (e.g., atherosclerosis, diabetes)
- Thermostat malfunction (e.g., hypothalamic hemorrhage)
- Pump malfunction/decreased cardiac output (e.g., cardiac disease, β-blockers)
- Low coolant levels/dehydration (e.g., inadequate intake, diarrhea, vomiting, diuretics)
- Radiator malfunction (e.g., skin disease, anhidrosis, occlusive clothing, anticholinergic drugs)

9. What risk factors can lower the threshold for heat stroke?

In addition to the previously mentioned factors, age (infants, young children, and the elderly), inability to care for oneself, alcohol abuse, obesity, and nonacclimatization can limit the ability to respond to changes in temperature.

10. Why are young children at higher risk for heat illness?

They have a higher surface to mass ratio, which increases their absorption of heat. In addition, they have a lower proportion of sweat glands to regulate heat loss.

11. List the spectrum of heat illnesses and briefly describe them.

- Heat edema: Transient swelling of hands, feet, and ankles because of dependent pooling of interstitial fluid via peripheral vasodilation, commonly occurring in nonacclimatized individuals. Usually resolves spontaneously, and may be amendable to compression stockings and elevation. Diuretics are not useful and should be avoided as they may worsen heat illness.
- Prickly heat (also known as *heat rash, miliaria rubra,* or *lichen tropicus*): Pruritic vesicular rash on an erythematous base are caused by excessive sweating and blockage of sweat ducts, primarily occurring on parts of the body covered by tight clothing. Blocked ducts can become infected and can be treated with chlorhexidine cream and small amounts of salicylic acid 1% to localized areas. Routine use of talcum or baby powder should be avoided.
- Heat cramps: Painful involuntary spasms of large muscle groups occurring after exertion and copious sweating, thought to be caused by excessive salt depletion. If seen in a patient, heat cramps should trigger additional evaluation for volume depletion and other heat-related illness. Treated with oral salt solutions (not salt tablets, which can delay gastric emptying) or intravenous fluids.
- Heat syncope: Loss of consciousness caused by a precipitous drop in blood pressure as a result of peripheral vasodilation shunting blood from the core, characterized by rapid return to normal mental status and functional ability. Worsened by dehydration and prolonged standing.
- Heat exhaustion: Excessive dehydration and electrolyte depletion, leading to fatigue and other symptoms (e.g., nausea, headaches, muscle cramps). Patient may be hypotensive but is typically not hyperthermic.
- Heat stroke: A medical emergency caused by neurologic dysfunction and hyperthermia at a temperature greater than 40°C (104°F). Neurologic changes may range from confusion and delirium to seizures and coma, and the patient may or may not be diaphoretic.

Some have advocated for describing heat illness in basically two categories: heat stroke, and everything else that can occur in a hot environment that may or may not be directly caused by heat exposure (e.g., electrolyte repletion and exercise leading to cramps, syncope, and exhaustion).

12. How are heat-related illnesses treated?

General management includes removing the patient from the hot environment into a shady or air-conditioned area and replacing fluid loss with oral electrolyte and salt solutions and, in more severe cases, intravenous fluids.

Immediate cooling should be initiated, especially in the case of heat stroke. Active cooling is most efficiently achieved by removing all of the patient's clothing, wetting the patient, and using a fan to circulate air over the patient (allows for both convective and evaporative heat loss). Cold packs to the groin and axillae may also be applied but have limited effect. Immersion in ice water baths is a very effective way of cooling patients (takes advantage of water's 25 times greater conduction rate as air); however, this is not practical in many settings. Cooling blankets are of limited use and should not replace evaporative cooling methods. Invasive cooling techniques, such as iced gastric, peritoneal, or rectal lavage and cardiopulmonary bypass, do not have a clear advantage over evaporative cooling and can cause delays in initiating treatment.

Core temperature should be monitored (rectal thermometry is considered the gold standard). Free water deficits should be replaced slowly over 48 hours, as overcorrection of hypernatremia can lead to cerebral edema.

13. Tell me more about heat exhaustion.

Heat exhaustion is caused by either water or salt depletion, or both. With water depletion, there is progressive dehydration resulting from inadequate water consumption when working in a hot environment (termed *voluntary dehydration,* because individuals commonly only replace two thirds of their water loss). This can progress to heat stroke. In the salt depletion form, excessive sweating is replaced by free water, which leads to hyponatremia and hypochloremia with near-normal body temperature.

Regardless of the primary cause, symptoms can be variable, and include fatigue, weakness, headache, impaired judgment, vertigo, nausea/vomiting, and occasionally muscle cramps (if salt depletion). Orthostatic dizziness and syncope is possible, and sweating may be profuse. Heart rate may be normal or tachycardic. Body temperature will be modestly elevated (<40°C [104°F]) or may be normal. Signs of major central nervous system (CNS) dysfunction (seizures, coma) or temperature greater than 40°C (104°F) portend to a worse diagnosis (heat stroke).

14. Why is heat stroke so bad?

Heat stroke is a life-threatening medical emergency characterized by an elevated core body temperature (>40°C [104°F]), and CNS dysfunction with varying degrees of shock and multiorgan collapse. Skin is hot, and the patient may or may not be sweating. Symptoms include muscle twitching, confusion, drowsiness, disorientation, ataxia, anxiety, psychosis, seizures, and coma. In addition, vasoconstriction of the renal and splanchnic circulation leads to acute renal failure, hepatic damage, and intestinal ischemia. SIRS and/or DIC may develop, resulting from hepatic injury-mediated coagulopathy, activation of clotting factors, thrombocytopenia, fibrinolysis, and direct thermal injury of the vascular endothelium

15. Describe the two types of heat stroke.

Classic heat stroke (CHS) occurs during periods of sustained high ambient temperatures and humidity, and particularly affects individuals who are in poorly ventilated areas (no air-conditioning). People who are particularly vulnerable to this condition include those of low socioeconomic status, the elderly, infants, alcoholics, and patients with chronic medical conditions or taking medications altering their ability to adapt to the hot environment. Anhidrosis is common, and laboratory abnormalities are generally mild.

Exertional heat stroke (EHS) is observed in active, young, healthy individuals (such as athletes and military recruits) whose normal adaptive responses to a hot climate are overwhelmed by endogenous heat production (strenuous activity). Sweating is preserved. Lactic acidosis, rhabdomyolysis, and renal failure is common, as is hypoglycemia resulting from depleted glycogen stores and increased glucose metabolism.

16. How do I treat a patient with heat stroke?

Primary attention should be given to the airway, breathing, and circulation (ABCs). Immediate and rapid cooling must be initiated, with a goal temperature of lower than 38.9°C (102°F) within 30 minutes of presentation to minimize organ damage. Cooling should not be delayed to determine exact temperature, although core temperature should be monitored during treatment. You cannot cool too quickly. Cold water immersion has been shown to be effective for patients with EHS. Evaporative cooling is effective for both CHS and EHS.

In addition, intubation may be necessary if the patient is unable to protect his or her airway; hypoxemia may occur as a result of aspiration, pulmonary infarction, or edema.

Resuscitation with intravenous fluids is necessary for concomitant dehydration, although cooling can singularly increase blood pressure via peripheral vasoconstriction. Electrolyte repletion should be appropriately tailored to specific laboratory derangements. Hemodynamics and urine output should help guide resuscitation efforts. Central venous pressure (CVP) may be a deceptive marker of resuscitation, because patients may have high cardiac index, low peripheral vascular resistance, and transient right-sided heart failure caused by a distributive shock state. Overresuscitation should be avoided, because it may exacerbate pulmonary edema.

Rhabdomyolysis is typically treated with fluid resuscitation but may warrant urinary alkalinization. In oliguric patients, mannitol should be avoided, and hemodialysis should be considered in severe cases.

Tachyarrhythmias that may develop as a result of heat stroke commonly resolve with cooling, and electrical cardioversion should be avoided, because it may worsen myocardial damage.

17. Are any medications indicated in the treatment of heat stroke, and are there any that should be avoided?

As mentioned, evaporative cooling and supportive care is paramount. However, benzodiazepines may be used to control shivering. Chlorpromazine, although efficacious for shivering, has anticholinergic properties and should be reserved for only severe cases where cooling is not adequate because of violent shivering. Dantrolene, which is used to treat malignant hyperthermia, is not effective in the treatment of heat stroke.

Antipyretic agents such as acetaminophen or salicylic acid have not been shown to be beneficial, and may in fact cause harm by worsening hepatic damage (in the case of acetaminophen) or worsening hyperthermia and coagulopathy (salicylates). α-Adrenergic agents, such as norepinephrine, should be avoided, because they can worsen vasoconstriction and decrease cutaneous heat exchange (therefore compromising cooling treatment). Atropine and other anticholinergics that inhibit sweating should also be avoided.

18. What laboratory abnormalities are seen in patients with heat illness?

Patients with severe heat exhaustion and heat stroke may have evidence of acute renal failure (elevated creatinine), hemoconcentration (elevated hemoglobin/hematocrit), rhabdomyolysis (elevated creatinine kinase), hypernatremia or hyponatremia, hyperkalemia or hypokalemia, leukocytosis, and DIC (prolonged prothrombin time and partial thromboplastin time, elevated D-dimer). Importantly, elevated liver enzymes can help distinguish severe heat exhaustion from heat stroke, because transaminases may be in the tens of thousands with heat stroke but may take more than 24 hours to develop.

19. What is the differential diagnosis for the etiology of heat stroke?

- Infection
  - Generalized (e.g., sepsis, malaria, typhoid, tetanus)
  - CNS (e.g., encephalitis, meningitis, brain abscess)
- Drugs/toxins
  - Intoxication (e.g., phencyclidine, amphetamines, cocaine, anticholinergics, salicylate, diuretics, antipsychotics phenothiazines)
  - Withdrawal (e.g., ethanol)
  - Serotonin syndrome
  - Neuroleptic malignant syndrome
  - Malignant hyperthermia
  - Drug-induced fever
- Endocrine derangements
  - Thyroid storm, pheochromocytoma
- Neurologic conditions
  - Status epilepticus, cerebral hemorrhage
- Blood clots
  - Deep vein thrombosis, pulmonary embolism, deep-seated hematomas

20. What is the mortality rate associated with heat stroke?

The mortality rate varies because of a number of factors, such as age, underlying comorbidities, and, most important, the degree and duration of hyperthermia; it can range from 20% to 60%. Poor prognostic indicators include advanced age, persistent hypotension requiring vasopressor therapy, and respiratory failure requiring intubation.

21. How do I prevent heat-related illness?

Maximize heat loss with lightweight, loose fitting clothing (which allows for enhanced convection and evaporation), and ensure adequate hydration and electrolyte repletion before and during any strenuous activity in a hot environment. Decrease heat stress by limiting exertion as much as possible.

22. What about acclimatization?

Acclimatization is the physiologic adaptation a normal person develops as a result of repeated exposure to heat stress, which allows for earlier onset of sweating (at a lower core temperature), increased sweat volume, and decreased electrolyte concentration of sweat. Additionally, heart rate

generally lowers, and stroke volume becomes more robust as the cardiovascular system adapts to the hot environment. The process can take a week or more, and involves limiting exertional activity during that period.

KEY POINTS: HEAT ILLNESS

1. Heat stroke is life threatening and is defined by temperature greater than 40°C (104°F) and altered mental status. Heat stroke victims may or may not be sweating.
2. Cooling should be initiated immediately upon suspicion of heat stroke, and you cannot cool a patient too quickly.
3. Evaporative cooling is quick, safe, and effective.
4. Antipyretics are not effective for environmental hyperthermia.

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QUESTIONS

1. What is paramount in treatment for heat illnesses?
   a. Hydration
   b. Electrolyte repletion
   c. Cooling
   d. Antidotes
   The correct answer is *c*.
2. What is the correct description of heat stroke?
   a. Cerebrovascular accident as a result of elevated temperatures
   b. Loss of consciousness when temperature is greater than 40°C (104°F)
   c. Altered mental status because of heat
   d. Body temperature greater than 40°C (104°F) and altered mental status
   The correct answer is *d*.
3. What is the most effective and readily available method of cooling?
   a. Ice water immersion
   b. Misting with tepid water and fanning
   c. Ice packs to the groin and axilla
   d. Lavage and cardiopulmonary bypass
   The correct answer is *b*.

CHAPTER 60

ALTITUDE ILLNESS AND DYSBARISMS

Jeffrey Druck, MD, and Alex Badulak, MD

1. What are the three disease states that comprise high-altitude illness?

High-altitude illness comprises three distinct clinical entities:

1. Acute mountain sickness (AMS)
2. High-altitude pulmonary edema (HAPE)
3. High-altitude cerebral edema (HACE)

2. What are the symptoms of AMS?

AMS is 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
- Difficulty sleeping

It is an entirely clinical definition.

3. How quickly do symptoms of AMS develop, and what is the minimum elevation at which AMS occurs?

Usually, symptoms begin within 6 to 12 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 to 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 [bid]), 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?

The strongest independent risk factor for developing AMS is a history of AMS during previous ascents. Other major independent risk factors include fast rate of ascent (>625 m per day above 2000 m elevation) and a lack of acclimatization (fewer than 5 days at elevations >3000 m in the 2 months preceding ascent).

Other possible risk factors include:

- Exertion on arrival
- Age younger than 46 years
- Female sex
- History of migraines
- Elevation attained

Abstract

This chapter reviews the key elements of altitude-related illnesses, as well as their diagnosis and treatment.

Keywords:

altitude illness, high-altitude pulmonary edema (HAPE), high-altitude cerebral edema (HACE), dysbarisms, arterial gas embolism (AGE), acute mountain sickness (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?

Data support nonpharmacologic approaches to preventing AMS, including acclimatization with 1 week of residence, physical activity from 2000 to 3000 m elevation, and slow ascent of no more than 300 to 500 m per day above 2500 m with a rest day every 3 to 4 days. Pharmacologic therapy includes nonsteroidal antiinflammatory drugs (NSAIDs; acetylsalicylic acid [ASA] 325 mg every 4 hours starting 1 hour before ascent or ibuprofen 600 mg three times daily starting a few hours before ascent), acetazolamide 125 mg twice daily starting the day before ascent and continuing for 2 days at maximum altitude, or dexamethasone 4 mg twice daily for the same period. High-risk patients should be offered acetazolamide 250 mg two or three times daily and/or dexamethasone 4 mg three times daily. The data on *Gingko biloba* for the treatment of AMS remain controversial. However, 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. Acclimatization
2. Slow ascent
3. Acetazolamide or dexamethasone

7. What is HACE?

HACE is a clinical diagnosis distinguished by a change in consciousness and associated truncal ataxia, and usually mild fever in the setting of ascent to altitude (usually >4000 m). Magnetic resonance imaging (MRI) findings in patients with HACE include vasogenic edema and microhemorrhages in the corpus callosum, and death results from brain herniation.

8. When does HACE occur?

The onset of HACE usually occurs 2 to 5 days after arrival at 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 HACE are the same as those for AMS.

11. What is HAPE?

HAPE is 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 2 to 3 days of arrival at altitude.

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 being researched and show promise in small randomized controlled trials (RCTs).

14. Is there any preventive therapy for HAPE?

Nifedipine has been shown to decrease the recurrence of HAPE in patients with previous HAPE. New data suggest that acetazolamide does not prevent HAPE, although it may have a role in preventing reentry HAPE in children who reside at altitude, go to low elevation, and are prone to developing HAPE upon return to altitude. High-dosage dexamethasone may also have a role in HAPE prevention. 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. Will I ever see HAPE, HACE, or AMS at the same time?

HACE is thought to be end-stage AMS, so you will 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, and 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 50% when untreated

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 [AGE], pneumomediastinum, and barosinusitis), as well as disease related to bubble formation (e.g., pulmonary decompression sickness [DCS], spinal decompression sickness, and the bends).

18. How much pressure does a diver experience at 10 m (33 ft) underwater?

Each 10 m (33 ft) is equivalent to 1 atm. Because sea level is equivalent to 1 atm, 10 m (33 ft) underwater is 2 atm, which is equal to 29.4 psi or 1520 mm Hg.

19. What are the bends?

The bends, also known as *caisson 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 I 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. Add into this mixture Henry's law, which 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 be 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 inhaled gas 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 (Valsalva maneuver). 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 could a diver get a pneumothorax with ascent?

If a diver held his or her breath to go underwater to 10 m (33 ft, or 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, if he or she does not continue breathing normally but holds his or her breath, the diver's lung volume would double, resulting in rupture of the lung parenchyma, thus causing a pneumothorax.

25. What is 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 atm underwater (effectively 2 atm), one third at an effective 3 atm, 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 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 central nervous system (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 arterial circulation.

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 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), thus entering the cerebral arterial circulation.

32. How do I 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 utility 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 rates. 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. Victims of facial barotrauma 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. First aid oxygen therapy may result in better outcomes while transport to a hyperbaric oxygen chamber is being arranged. NSAIDs, such as tenoxicam, and air mixtures, such as heliox (a mixture of helium and oxygen), may reduce the number of patients requiring recompression therapy but do not appear to change 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. It is also recommended that one should not fly within 24 hours of diving, because aircraft cabins are pressurized to about 8000 ft, and this lower pressure may cause DCS or the bends. Air mixtures such as nitrox, a nitrogen and oxygen gas mixture with greater than 21% oxygen, or heliox, a mixture of helium and oxygen particularly for deep dives, will also decrease the risk of DCS.

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QUESTIONS

1. Which of the following is not a treatment for AMS?
   a. Descent
   b. Supplemental oxygen
   c. Acetazolamide
   d. Sildenafil
   The correct answer is *d*. Supplemental oxygen, acetazolamide, dexamethasone, and immediate descent to a lower elevation are all possible treatments for AMS. Sildenafil is being used as an experimental medication for the treatment of HAPE.
2. Which of the following is not a pressure-related complication?
   a. MEBT
   b. The bends
   c. Hermansky-Pudlak syndrome
   d. AGE
   The correct answer is *c*. *MEBT* is middle ear barotrauma; the bends is caused by rapid ascension with nitrogen precipitating in the tissues; and *AGE* is an arterial gas embolism, resulting from the rupture of a alveolus. Hermansky-Pudlak syndrome is a genetic tyrosinase deficiency.
3. How do you treat DCS?
   a. Hyperbaric chamber
   b. Dexamethasone
   c. Acetazolamide
   d. Sildenafil
   The correct answer is *a*. Only hyperbaric therapy helps with DCS.

CHAPTER 61

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°C (100.4°F). Be aware that parents often consider a fever to be below the 38°C mark, such as when a parent says that a child had a fever of 99.2°F.

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. Oral temperatures are generally not attempted in young children for obvious logistic 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 the following:
   - Bacteremia
   - Urinary tract infection (UTI)
   - Bacterial meningitis
   - Pneumonia (established by a focal infiltrate on chest radiograph)

5. **Does it matter how much fever the child has?**
   Hyperpyrexia (temperature of 40.5°C [104.9°F]) 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 *appearing toxic*?**
   Children who appear toxic 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

Abstract

Fever is a common presenting complaint in children in the ED. Although most fevers are the result of a viral infection, serious bacterial infections (SBIs), such as bacteremia, urinary tract infection (UTI), meningitis, and pneumonia, must be considered. Febrile seizures are very common but do not place the child at higher risk for SBI and have no long-term morbidity. Neonates with a fever should undergo a complete sepsis evaluation and be admitted for antibiotics. Infants between the ages of 1 and 3 months may undergo risk stratification to decide disposition. Among infants and children older than 3 months, UTI is the most common SBI.

Keywords:

fever, febrile seizure, Rochester criteria, Philadelphia criteria, bacteremia, pneumococcal vaccine

7. Which antipyretics work best for children?

Studies show that acetaminophen (15 mg/kg; suspensions 160 mg/5 mL) and ibuprofen (10 mg/kg; suspension 100 mg/5 mL) have similar efficacy, and both work well for getting febrile children to defervesce. Because household measuring spoons measure volume inaccurately, parents should be encouraged to use syringes for medication dosing.

Note: Most children's elixirs contain half the amount of an adult tablet per 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 dosage, or be unfamiliar with units of measure (such as *mL*). It is also common for parents to believe that antipyretics should cure the fever, and a parent may say, "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 often help with this issue.

9. What is wrong with baby aspirin?

Aspirin administration to children with certain viral infections has been associated with Reye syndrome (encephalopathy and acute liver failure). This syndrome, although very 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. However, fever by itself is not harmful.

11. What are febrile seizures?

Febrile seizures, the most common seizure disorder seen in children (incidence 4%), are typically associated with high fevers early in the course of illness. There is no evidence to support the myth that febrile seizures are associated with the rate of rise of the fever rather than the absolute temperature. There is no increased risk of SBI in patients with simple febrile seizures as compared with children who have fever alone. A simple febrile seizure is characterized as a generalized tonic-clonic seizure that does not recur within 24 hours, is associated with fever, lasts less than 15 minutes, and occurs in an otherwise neurologically normal child between the ages of 3 months and 5 years. There are excellent long-term data that show 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°C [100.4°F]) younger than 1 month should receive a complete sepsis evaluation.

- 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 premature infant of 32 weeks' gestation who was born 6 weeks ago is still considered to be younger than 1 month.

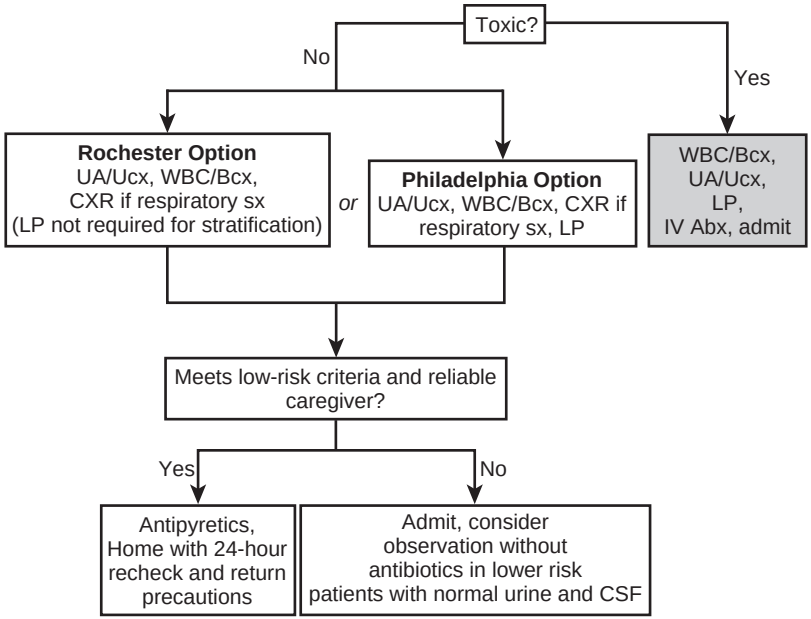

Figure 61-1. Algorithm for infants 1 to 3 months of age. *Abx,* Antibiotics; *Bcx,* blood culture; *CSF,* cerebrospinal fluid; *CXR,* chest x-ray; *IV,* intravenous; *LP,* lumbar puncture; *sx,* symptoms; *T,* temperature; *UA,* urinalysis; *Ucx,* urine culture; *WBC,* white blood cell count.

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 the algorithm in Figure 61-1.

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 61-2 (note different temperature cut-off for this older age group).

16. How do I 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 sign, Kernig sign, neck stiffness, or bulging fontanel) are commonly absent and unreliable in young children.

17. What if the child has a fever source or one is found during the workup?

It depends on the source and the age group. A viral source makes the risk of bacteremia and meningitis lower. 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 UTIs, and rarely bacteremia, may coexist with viral infections, respiratory infections such as bronchiolitis, and gastroenteritis.

18. Must I always follow the guidelines, or is there room for clinical judgment in there somewhere?

A study of more than 3000 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 identifying and treating SBI as rigid adherence to clinical guidelines. Their findings suggest that if close follow-up monitoring 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 care.

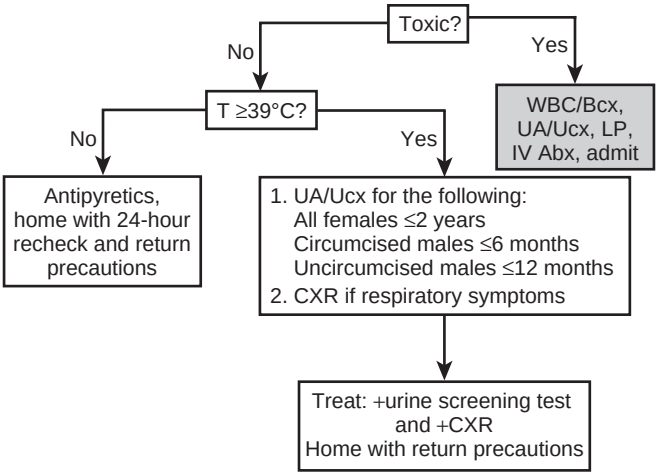

Figure 61-2. Algorithm for children 3 to 36 months of age. *Abx,* Antibiotics; *Bcx,* blood culture; *CSF,* cerebrospinal fluid; *CXR,* chest radiograph; *IV,* intravenous; *LP,* lumbar puncture; *sx,* symptoms; *T,* temperature; *UA,* urinalysis; *Ucx,* urine culture; *WBC,* white blood cell count.

19. What if the child looks great; can he or she go home?

Children who do not appear toxic, are older than 1 month, and meet low-risk criteria may be discharged home with return precautions and closely monitored follow-up care. This, of course, presumes a child has 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 appears well at the time of evaluation. If a patient meets low-risk stratification criteria, discharge without antibiotics is recommended with a 24-hour follow-up examination.

Rochester criteria

- WBC between 5000 and 15,000
- Urine WBC count less than 10/high-power field (hpf)
- Stool WBC count less than 5/hpf (in infants with diarrhea)
- **Note:** LP not required for stratification

Philadelphia criteria

- WBC count less than 15,000, with band-to-neutrophil ratio less than 0.2
- Urine WBC count <10/hpf and no bacteria on Gram stain
- Cerebrospinal fluid (CSF) WBC count less than 8/hpf
- Negative CSF Gram stain
- Stool and chest radiograph negative (if obtained)

21. What is the risk of occult bacteremia (presence of bacteria in the blood stream with no apparent focus of 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 pneumococcal vaccine (13 valent) suggests that the current rate of bacteremia is significantly 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. The current ratio of contaminant to true positive blood culture is 8:1.

22. What antibiotic should be used for empiric coverage of bacteremia?

Younger than age 28 days
- Cefotaxime 50 mg/kg IV (may consider substituting with gentamicin 2.5 mg/kg IV in first week of life to treat group B streptococcal infection)

plus
- Ampicillin 50 mg/kg IV (to cover for *Listeria*)

Age 28 days to 3 months
- Cefotaxime 50 mg/kg IV

Age 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 the following:
- Maternal HSV infection at delivery (although two thirds are asymptomatic)
- Maternal history of HSV or other sexually transmitted diseases
- Vesicular rash
- Seizures
- CSF pleocytosis (WBC count of 20 to 100 is typical.)

Patients are usually younger than 22 days of age. Patients with risk factors should be given acyclovir pending an 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 dosage is 20 mg/kg IV every 8 hours (dosage 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 proven otherwise. The condition of a 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

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QUESTIONS

1. Which of the following is not a criteria for a simple febrile seizure?
   a. Lasts less than 15 minutes
   b. Patient is between the ages of 3 months and 6 years
   c. May have 1 to 2 seizures within 24 hours
   d. Normal after the seizure
   The correct answer is *c.*
2. The rate of bacteremia in otherwise healthy children who have a fever and look well is
   a. Less than 1%
   b. 1%
   c. 2%
   d. 4%
   The correct answer is *a.*
3. Which of the following is not a part of the Rochester criteria to low-risk stratify a 6-week-old infant with a fever?
   a. WBC count greater than 5000 and less than 15,000
   b. Urine less than 10 WBC/hpf
   c. Stool less than 5 WBC/hpf
   d. LP less than 5 WBC/hpf
   The correct answer is *d.*

SEIZURES IN INFANCY AND CHILDHOOD

Andrew M. White, MD, PhD

CHAPTER 62

1. How does one determine if an event in a child is actually a seizure?

Many events that appear to be seizures are actually nonepileptic. These events can be classified by the age at which the event occurred (Fig. 62-1).

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. Side-to-side movements of the head are also usually not seizures. It is always good to have a witness demonstrate the movements, or better yet to get a video.
- Are the events always provoked? Seizures are generally not provoked; breath-holding spells are always provoked; the child is upset or injured, begins to cry, and stops breathing. There may be associated jerks.
- Is there tongue biting during the event and, if so, where? During a true seizure, the tongue is usually bitten on the side. During syncope, the tongue is usually not bitten, but if so, usually bitten on the tip.
- Was there urination or loss of stool? This favors a diagnosis of true seizure.
- Does it only happen during exercise? If so, it is more likely to be a cardiac event.
- What do the eyes do during the seizure? During most seizures, the eyes remain open. They may roll up or deviate to one side or the other.
- Does it happen only during certain times of day? Seizures will happen at all times of day, but there are certain types that are much more common in sleep (benign rolandic) and others that are more common during waking hours (benign infantile partial seizures).
- Does it only happen upon standing? The event is likely to be orthostatic syncope or cardiac in origin.
- What is the child like after the event? Except for absence-type or myoclonic seizures, children are postictal (tired, confused) after seizures.
- Does the child retain consciousness during the event? If it is a generalized tonic-clonic seizure (convulsion), this is impossible, because both halves of the brain are involved. Retaining consciousness is possible for focal seizures or brief absences.
- Does the child remember the event? Typically, the child will not remember the entire complex partial (focal) seizure or any part of a generalized tonic-clonic seizure (convulsion). Depending on the length of an absence spell, it is possible that there will be recollection (<6 seconds).

2. What can be learned about the child's history?

- Get a step-by-step description of the events before, during, and after the seizure.
- Learn about activities before the seizure (e.g., provoked breath-holding spells).
- Get 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.
- Ask additional questions to establish the cause of the seizure, including any recent illnesses, trauma, and overall progression in development.
- Obtain past medical history, as well as a family history of seizures.
  - In patients with known epilepsy, inquiries about medication compliance are necessary.

Abstract

Seizures are defined as an excessive, nonphysiologic, synchronized discharge of neurons. Epilepsy is defined as an increased tendency to have unprovoked seizures. Operationally, it is defined as two or more unprovoked seizures. This chapter discusses the diagnosis, evaluation, and management of seizure disorders.

Keywords:

pediatric epilepsy, seizures, febrile seizures, antiepileptic medications

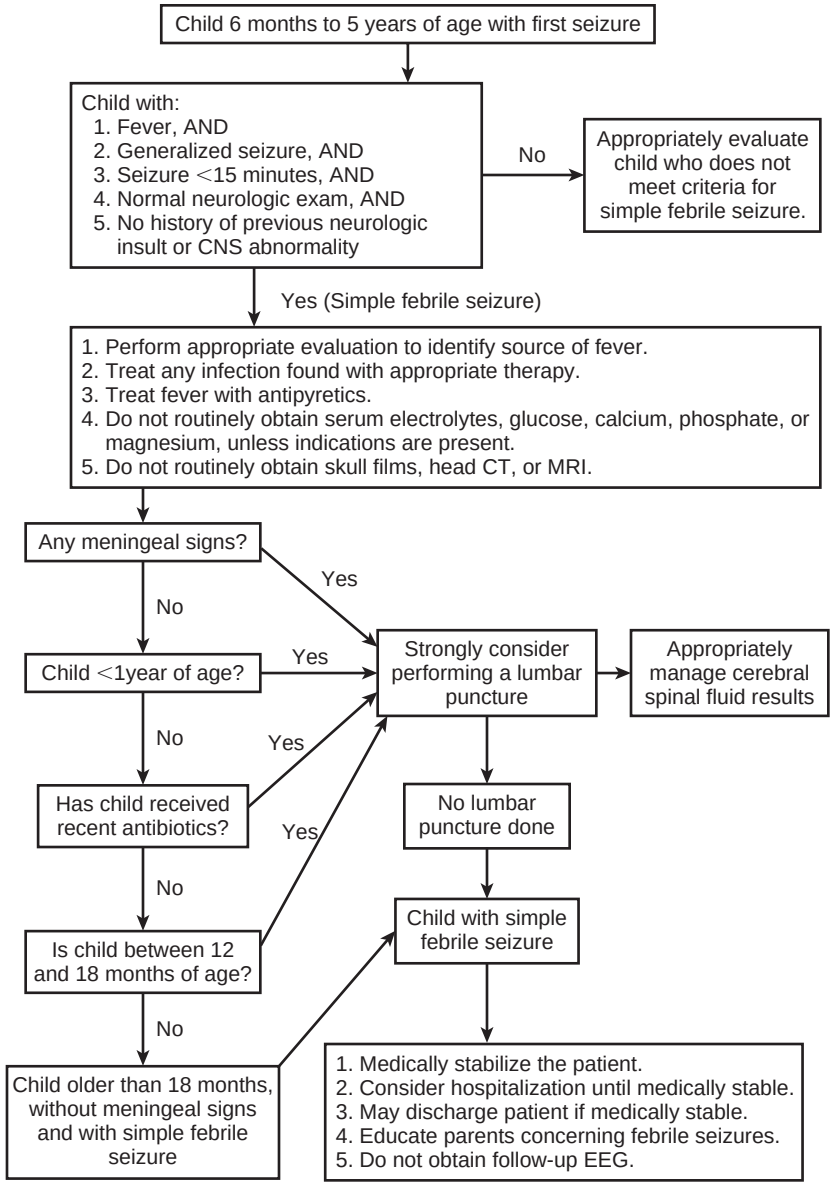

Figure 62-1. Evaluation of child with simple febrile seizure. *CNS,* Central nervous system; *CT,* computed tomography; *EEG,* electroencephalogram; *MRI,* magnetic resonance imaging. *(Modified from Committee on Quality Improvement, Subcommittee on Febrile Seizures, American Academy of Pediatrics: Practice parameter: the neurodiagnostic evaluation of the child with a first simple febrile seizure.* Pediatrics *97:769–775, 1996.)*

3. What things should be sought on physical examination?

Perform a complete neurologic 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 abusive head trauma (retinal hemorrhages, bruising, fractures) should be performed.

4. How are pediatric seizures classified?

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 before the seizure. This can help significantly in the localization of the epileptic focus. Seizures can also be classified by syndrome, prognosis, and cause. Classification systems have recently been updated. The latest classification system (2010) describes generalized, focal, and unknown seizure types. They are also classified based on seizure etiology (genetic, structural/metabolic, or unknown), and can be further categorized based on whether they are part of a syndrome, and, if so, the particular age group that corresponds to that syndrome.

5. What are common reasons for a seizure in the neonate?

The most common cause for neonatal seizures is hypoxic-ischemic encephalopathy. Additional causes include:

- Intracranial hemorrhage (subarachnoid in full-term infants, germinal matrix in preterm neonates)
- Metabolic disturbances (hypoglycemia, hypocalcemia, drug withdrawal, amino acidemias, organic acidurias, urea cycle defects)
- Infection: TORCH infections (toxoplasmosis, rubella, cytomegalovirus, herpes 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?

Determine serum electrolyte levels, including glucose, calcium, and urine toxicology reports. Request an ammonia level (free-flowing collection), looking for a urea cycle defect. Unless another cause is found, perform a lumbar puncture (LP) for an infectious etiology. Cerebrospinal fluid (CSF) studies 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. There is an infantile epilepsy panel available, but it is quite expensive. Cerebral imaging studies include ultrasound, computed tomography (CT) scan, or magnetic resonance imaging (MRI). Although requiring sedation, MRI is the gold standard and 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. Newer medications for neonates include topiramate (Topamax) and levetiracetam (Keppra).

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)
- Structural abnormalities (malformation of cortical development)

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 afebrile 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 sibling 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%), prolonged for more than 15 minutes (8%), or 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. Simple febrile seizures have none of the complex features.

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 of whether 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 neurologic examination
- Lower temperature at onset of seizure
- Brief period of recognized fever
- More frequent illnesses (daycare)

13. What tests should be done after a febrile seizure?

Children with simple febrile seizures should be evaluated and treated based on fever alone, because the concomitant presentation of the seizure does not increase the risk of serious bacterial illnesses above baseline. For complex febrile seizures without an obvious source, consider the following:

- Complete blood count (CBC), glucose, electrolytes, calcium, magnesium, urinalysis (UA), and stool culture, particularly for complex febrile seizures. (See Chapter 61 for further discussion on pediatric fever.) Strongly consider LP if the patient is younger than 18 months with no fever focus.
- Head CT for patients with focal neurologic features or altered mental status
- EEG (as outpatient if patient returns to baseline neurologic status) if complex features or strong family history of epilepsy
- Nonemergent outpatient MRI with pediatric neurology follow-up consultation

14. Under what conditions should a child having febrile seizures be treated, and what treatments should be used?

The two medications shown effective at decreasing subsequent febrile seizures are phenobarbital and valproate. However, phenobarbital may impact cognitive ability in children. Neurologists also use levetiracetam. Divalproex (Depakote) is not a good drug for children younger than 2 years because of the potential for fatal hepatotoxicity. Medication should only be started (in consultation with a pediatric neurologist) with the following features: high frequency, prolonged, or a family remote from medical care.

Attempts at controlling the fever with agents such as acetaminophen or ibuprofen usually fail; the seizure is typically the first sign of the illness. Use rectal diazepam or intranasal diazepam for children with prolonged (longer than 5 minutes) febrile seizures to treat the seizure and help alleviate parental anxiety.

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 1/3000 of infants, typically developing 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, incontinentia 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 at first, but multiple clustered jerks lasting several minutes may subsequently occur. The jerking can be unilateral or bilateral, and are often accompanied by a cry. Developmental regression is a poor prognostic sign. The pattern on the EEG associated with the syndrome is high amplitude and chaotic, termed *hypsarrhythmia.* The actual spasm is associated with an "electrodecrement," or flattening of the EEG.

17. What is the standard treatment for infantile spasms?

The standard treatment for infantile spasm is adrenocorticotropic hormone (ACTH). ACTH can have severe side effects, including hypertension, osteoporosis, and decreased resistance to infection. ACTH has become exceedingly expensive ($100,000 per course), and practitioners are now using steroids and other newer drugs such as zonisamide or topiramate. Vigabatrin is a drug that is available in the United States under a special program and is the preferred drug in tuberous sclerosis. It is less expensive ($10,000) and only slightly less effective in studies. A possible side effect of this drug is permanent loss of peripheral vision.

18. What is the prognosis for infantile spasms?

The prognosis for infantile spasms is quite poor, with the mortality rate reported as high as 33%. Only 12% of children who survive to adulthood have normal intelligence. There is no currently proven treatment.

19. What is epilepsy?

Epilepsy is described as the tendency to have unprovoked recurring seizures. Operationally, an individual who has had two or more unprovoked seizures is said to have epilepsy.

20. What are some common forms of childhood epilepsy?

- Childhood absence: Begins from 4 to 8 years of age. 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, valproic acid. In adolescent women, lamotrigine is a consideration. Additional drugs used more recently include topiramate and clobazam.
- Benign rolandic epilepsy: Begins from 6 to 10 years of age and involves facial and arm twitching, slurred speech, and drooling. It will occasionally generalize. No treatment is necessary unless seizures generalize during daytime. If that occurs, then carbamazepine or oxcarbazepine are reasonable.
- Juvenile myoclonic epilepsy: Begins from 12 to 18 years of age and consists of a 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 valproic acid or lamotrigine.

21. What workup should be done after an afebrile seizure in an asymptomatic child?

If the child has returned to normal, he or she can go home and then have a follow-up examination with an outpatient EEG and MRI. A neurologic 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. An LP should be performed based on clinical suspicious for meningitis. Occasionally, an acute EEG should also be performed after cessation of clinical symptoms to rule out subclinical status, which is especially common in children younger than 1 year.

22. Under what conditions should afebrile seizures be treated using antiepileptic drugs?

Treatment of seizures with antiepileptics balances the risk of recurrence with the risks of the medication. Antiepileptics are typically not started until after the second seizure. The risk of having a second afebrile seizure after a first is slightly less than 50%. Patients with a dramatically abnormal EEG, very strong family history, or abnormal neurologic examination should receive seizure prophylaxis in consultation with a pediatric neurologist.

23. What are the older and newer antiepileptics, and how do they vary?

- Older antiepileptics
  - Phenobarbital
  - Primidone
  - Phenytoin
  - Carbamazepine
  - Ethosuximide
  - Acetazolamide
  - Clonazepam
  - Valproate
- Newer antiepileptics
  - Topiramate, lamotrigine
  - Levetiracetam
  - Felbamate
  - Gabapentin
  - Oxcarbazepine
  - Zonisamide
  - Pregabalin
  - Clobazam
  - Tiagabine
  - Eslicarbazepine
  - Lacosamide
  - Vigabatrin
  - Perampanel
  - Ezogabine
  - Rufinamide

Older drugs have the advantage of lower cost and greater experience. Newer drugs have the advantages of better side-effect profiles, decreased monitoring requirements, less frequent dosing regimens, and decreased interaction with other drugs.

24. What are important side effects of the different antiepileptic drugs?

Almost all antiepileptics have been linked to suicidal behavior. Specific side effects include:

- 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
- Perampanel (Fycompa): Somnolence, headache, fatigue, and irritability
- Topiramate (Topamax): Sedation, glaucoma, and kidney stones
- Felbamate (Felbatol): Aplastic anemia (can be fatal), 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
- Gabapentin (Neurontin): Somnolence, dizziness, ataxia, nystagmus, headache, tremor, weight gain
- Rufinamide (Banzel): Somnolence, nausea, and headache
- Clobazam (ONFI): Somnolence
- Vigabatrin (Sabril): Peripheral vision loss

25. If an individual stopped taking antiepileptic drug because he or she was not having seizures, and then started to have them again, at what dosage should the medication be restarted?

If it has been longer than 1 week, the drug must be restarted (often tapered up) as first prescribed. This is especially important for a drug such as lamotrigine, which can cause a rash or Stevens-Johnson syndrome to occur if it is started too quickly.

26. When should antiepileptic drugs be discontinued?

The general rule is that an antiepileptic drug can be stopped after 2 seizure-free years. Exceptions to this rule would occur for a dramatically abnormal EEG or abnormal neurologic examination; a shorter time can be considered for neonatal seizures. Approximately two thirds of patients remain seizure free after drug withdrawal. Eighty percent of recurrent seizures present within the first 6 months of treatment cessation.

27. What happens if a dose of antiepileptic drug is missed?

If it is time for the next dose, simply continue on without giving additional medication. If it is not time for the next dose, give the missed dose, and slightly delaying the next dose.

28. What if vomiting occurs shortly after taking an antiepileptic drug?

If it is more than 1 hour since the drug was taken, no action is necessary. If it was from 30 to 60 minutes between the dose and vomiting, give a half dose. If less than 30 minutes, repeat the dose.

29. What is status epilepticus?

Status epilepticus is continuous or intermittent seizure activity lasting longer than 30 minutes.

30. What is the treatment for status epilepticus?

Stabilize airway, breathing, and circulation (ABCs) first, of course. Obtain a bedside glucose level, chemistry panel (with calcium, magnesium, and phosphorous), urine toxicology report, and anticonvulsant levels, along with intracranial imaging (usually CT). If possible, a continuous EEG (performed in consultation with a neurologist) is helpful and should establish a burst-suppression pattern if the seizure cannot be stopped.

If there is no intravenous (IV) access, consider either rectal diazepam or intranasal (or intramuscular [IM]) midazolam. After establishing IV access, give a benzodiazepine, such as lorazepam, repeating the dose multiple times if necessary. If seizures persist, give phenobarbital, phenytoin, or levetiracetam. 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?

- Place the child on his or her side.
- Clear anything near the mouth.
- Remove anything that may cause injury.
- Place something soft under the head.
- Time the event.
  - If less than 5 minutes, no action is likely needed.
  - If longer than 5 minutes, administer rectal diazepam or intranasal (or IM) midazolam, or take the patient to the ED.
- If rectal diazepam or midazolam has been given and the seizure has lasted longer than 10 minutes, go to the ED.

32. What cautions should I give to parents of children who have seizures?

- No bathing or swimming alone
- No ladders or activities above shoulder height

- 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 allow for the differentiation 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 epilepticus or not returning to baseline neurologic state requires consultation with a pediatric neurologist for treatment and EEG monitoring.

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QUESTIONS

1. All of the following are characteristics of seizures except:
   a. Stereotyped movements
   b. Eye closure
   c. Biting the tongue on the side
   d. Poor or lack of responsiveness
   The correct answer is *a*.
2. Which of the following is a feature of a simple febrile seizure?
   a. Focal features
   b. All extremities involved
   c. More than one in a day
   d. Prolonged seizure
   The correct answer is *b*.
3. Which of the following medications has been proven effective in the treatment of febrile seizures?
   a. Topiramate
   b. Divalproex
   c. Phenytoin
   d. Ethosuximide
   The correct answer is *b*.

ACUTE RESPIRATORY DISORDERS IN CHILDREN

CHAPTER 63

Cortney Braund, MD, and Genie E. Roosevelt, MD, MPH

1. What are the signs and symptoms of respiratory distress in a child?

The progression of respiratory distress is shown in Figure 63-1. 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 as fast as 60 breaths per minute, whereas a 12-month-old infant averages 30 breaths per minute. Until respiratory failure leads to hypoventilation and hypercarbia, arterial blood gas levels 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 (approximately one third the diameter of an adult's at birth) 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), whereas intrathoracic lesions (e.g., asthma, bronchiolitis) produce prolonged expiratory wheezing (i.e., high-pitched). Regardless of the location, severe pathologic disruption can produce both inspiratory and expiratory sounds.

4. What are common causes of upper airway obstruction in children?

See Table 63-1.

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 virus [RSV]) with erythema and swelling of the trachea just below the vocal cords, and patients classically have a "barky" or "seal-like" cough. The mean age of affected patients is 18 months, with an age range of 6 months to 3 years. There is 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; radiography is not indicated unless diagnosis is unclear or foreign body obstruction is a consideration.

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 to maximum of 5 mL) 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

Abstract

Respiratory disorders are a common complaint of children in the ED. It is essential to localize and differentiate the etiologies of pediatric respiratory complaints for appropriate management. This chapter discusses the presentation, evaluation, and management of the most common acute respiratory disorders in children.

Keywords:

respiratory distress, asthma, pediatric asthma, croup, laryngotracheitis, laryngotracheobronchitis, nebulized treatments, albuterol, racemic epinephrine, hypertonic saline, corticosteroids, prednisone, dexamethasone, bacterial tracheitis, epiglottitis, retropharyngeal abscess, inhaled foreign body, bronchiolitis, hypoxia

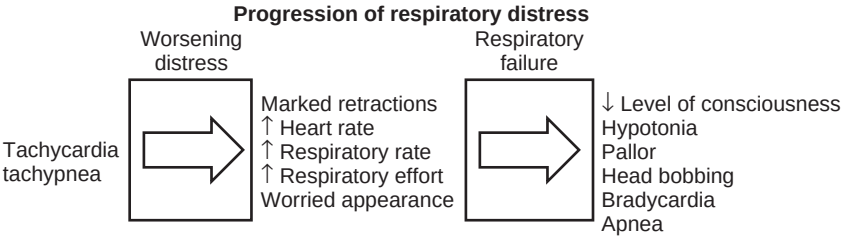

Figure 63-1. Signs and symptoms of respiratory distress in children.

for admission include continued stridor at rest, cyanosis, signs of respiratory distress, dehydration, or questionable adherence to follow-up instructions. 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 comes to the ED with croup. A single oral dose of dexamethasone decreases the need for hospitalization and return ED visits. Dexamethasone has traditionally been given orally at a dose of 0.6 mg/kg (maximum of 10 mg); however, studies have suggested similar efficacy with doses of 0.15 mg/kg and 0.3 mg/kg. There is no evidence to suggest that repeat dosing is indicated, but this might be considered for young patients who seek treatment before the peak of illness (i.e., day 1). Nebulized budesonide does not provide benefit over dexamethasone but may be considered in a patient unable to tolerate oral steroids.

KEY POINTS: CROUP

1. Treatment is with oral dexamethasone 0.6 mg/kg (maximum of 10 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 display such symptoms as drooling, dysphagia, stridor, 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 have croup-like symptoms but are toxic in appearance, with significant respiratory distress. Radiographs may show shaggy subglottic narrowing and clouding of the trachea. Airway management and broad-spectrum antibiotics (third-generation cephalosporin plus an antistaphylococcal agent, such as vancomycin or clindamycin) are the mainstay of therapy for both disorders.

9. What is the appropriate initial management of a patient with suspected epiglottitis?

Immediately set up for an emergent airway and 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. If epiglottitis is suspected, keep the child in a position of comfort (often on a parent's lap) and defer examination of the pharynx, because direct examination or manipulation of the oropharynx can cause contraction of the pharyngeal muscles and worsen airway obstruction. If the patient will tolerate it without agitation, start high-flow oxygen via a nonrebreather bag reservoir mask (where available, heliox can also be used). Radiographs, blood work, intravenous (IV) lines, and antibiotics can wait; if a child's airway becomes obstructed, bag-valve-mask ventilation should be attempted first.

Table 63-1. Causes of Upper Airway Obstruction

	ETIOLOGY	TYPICAL AGE RANGE	ONSET	TOXICITY	DROOLING	TREATMENT
Croup	Parainfluenza type 1, influenza A and B, RSV, rhinovirus, human metapneumovirus, adenovirus, 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	Common	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*, *Moraxella catarrhalis*, (often occurs after viral insult)	Infancy-6 yr	"Croup" prodrome	Moderate	Usually absent	Airway management, antibiotics
Foreign Body		5 mo-3 yr	Acute	Variable	Common	Airway management, endoscopic removal of foreign body

RSV, Respiratory syncytial virus; *URI*, upper respiratory infection.

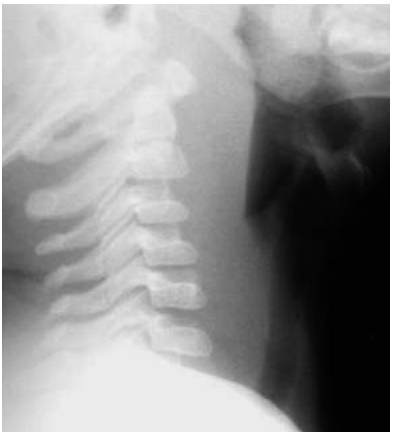

Figure 63-2. Radiograph of lateral neck showing thickening of prevertebral space.

10. What are retropharyngeal space infections?

The retropharynx is a potential space located immediately posterior to the pharynx, larynx, and trachea. Infection may arise by direct penetrating trauma or 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. About 90% of patients are younger than 6 years; the affected child usually appears alert (but mildly toxic), with fever. They may have limited range of motion of the neck (particularly unable to extend), upper respiratory symptoms, dysphagia, odynophagia, or neck swelling.

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. To avoid false-positive findings, the film should be obtained during inspiration with the neck held in normal extension. 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. 63-2). Computed tomography (CT) scanning is highly sensitive and used to differentiate abscess from phlegmon or soft-tissue cellulitis.

12. How are retropharyngeal infections 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 with foreign-body aspiration are males between 5 months and 3 years of age. Although a history of an aspiration event (found in 50% to 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. Expiratory or lateral decubitus films have been shown to lack adequate sensitivity or specificity to be useful. 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's airway cannot be intubated, perform a needle cricothyroidotomy.

15. What is bronchiolitis, and who does it affect?

Commonly found in children younger than 2 years, 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.

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 copious 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. Findings of more severe disease include hypoxemia, inability to feed, irritability, and lethargy. Young infants may experience periods of apnea.

17. Do patients with bronchiolitis need chest radiographs?

Infants with bronchiolitis do not need any laboratory or radiologic evaluation. Chest findings on radiograph 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 radiography to rule out other causes of first-time wheezing, including foreign body, congenital airway anomalies, congestive heart failure, or bacterial pneumonia.

18. When are laboratory tests needed for bronchiolitis?

Laboratory tests are not 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 (<1 month) with bronchiolitis is unchanged and includes a complete sepsis evaluation.

19. What is the treatment for bronchiolitis?

Bronchiolitis treatment is supportive, and involves supplemental oxygen, nasal suctioning, and hydration. There is no evidence to support the routine use of bronchodilators, steroids, or antivirals in the ED. Recent review of bronchodilators (albuterol or salbutamol) for bronchiolitis for infants with first-time wheezing showed no significant benefit in hospitalized patients, no improvement of oxygen saturation, no reduction in the need for hospitalization, and no reduction in the length of stay in hospital or illness at home. Additionally, albuterol may worsen ventilation/perfusion mismatch and exacerbate hypoxemia. Studies addressing the use of nebulized hypertonic saline suggest that it may improve the clinical severity score, but does not reduce need for hospitalization. Nebulized epinephrine may be slightly more effective, likely because of vasoconstrictive effects. When compared with placebo, there was no difference found for length of hospital stay in admitted patients, but it was found that epinephrine may be effective for reducing hospital admission.

KEY POINTS: BRONCHIOLITIS

1. Bronchiolitis treatment is supportive, involving supplemental oxygen, nasal suctioning, and hydration.
2. Nebulized epinephrine may be helpful in reducing the need for hospital admission.
3. Radiographs and laboratory evaluations are not routinely indicated.

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 63-2). Some centers use home oxygen therapy protocols for otherwise well-appearing patients requiring less than 0.5 L of oxygen per hour. These patients require a 24-hour follow-up visit, as well as reliable caretaker oversight.

Table 63-2. 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

21. How are bronchodilators used in the management of acute asthma?

Selective β_2-agonists (e.g., albuterol) 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 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, with the R isomer having the greatest effect on bronchodilation compared with the S isomer. 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 similar to 5 days of prednisone in children with mild to moderate asthma symptoms.

23. When should a chest radiograph be obtained, and what are the typical findings?

Chest radiography 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. Radiography commonly shows 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.

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 63-3).
2. Initial treatment
   - Oxygen as needed to keep saturation in the normal range
   - Three consecutive treatments of nebulized albuterol (2.5 to 5 mg or 0.15 mg/kg per treatment) plus ipratropium 250 to 500 μg
   - Steroids
3. Repeat assessment
   - Patient should be assessed after initial treatment to determine whether additional treatments are needed or if the patient can be observed for potential discharge. The full effect of albuterol may take 15 minutes.
   - 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, he or she can be safely discharged (Step 4a). If symptoms continue, the patient should be given additional therapy (Step 4b).

4a. Discharge
   - Discharge home with a reliable caretaker, patient education, medications, and follow-up instructions.

Table 63-3. Pediatric Asthma Score

	Score		
	1	2	3
Respiratory Rate			
2-3 yr	≤34	35-39	≥40
4-5 yr	≤30	31-35	≥36
6-12 yr	≤26	27-30	≥31
>12 yr	≤23	24-27	≥28
Oxygen requirement	>90% on room air	85% to 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.

- Discharge medications should include albuterol, nebulized or inhaled, every 4 hours as needed for wheezing and oral steroids.

4b. Continued therapy
- Continuous albuterol by nebulization (7.5 to 10 mg/h, can increase to 20 mg/h)
- Ipratropium 500 μg every 4 hours
- Frequent reassessment of patient

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?

Magnesium's effect in asthma is likely the result of the counteraction of calcium ions to prevent bronchial smooth muscle contraction. It may have a role in reducing inflammatory mediators, depressing muscle fiber excitability, and stimulating nitric oxide and prostacyclin synthesis. The benefit in patients with mild to moderate exacerbations is unclear, and its use is often reserved for those patients in severe distress or nonresponsive to albuterol and steroids. In a recent metaanalysis, IV magnesium sulfate improved respiratory function and reduced the number of pediatric hospital admissions. The dosage is a range of 25 mg/kg to 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 patients in the pediatric intensive care unit (PICU).

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, subcutaneous or IV, may be given as an initial bolus of 2 to

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 (at a dosage of 1 to 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 before auscultation helps localize and differentiate etiologies of pediatric respiratory complaints.
2. Foreign bodies should be suspected in any child with signs of airway obstruction.
3. Laboratory tests and radiographs are not routinely indicated in many childhood respiratory disorders.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Drs. Kelly Flett and Joan Bothner for their previous contributions to this chapter.

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QUESTIONS

1. A previously healthy 6-month-old infant is brought to the ED for evaluation of cough, congestion, and fever for 4 days. His vital signs are as follows: T 38.4°C (101.1°F), HR 140, RR 60 with room air saturations of 91%, BP 100/70. He is nontoxic in appearance. His examination is notable for mild intercostal retractions, diffuse expiratory wheezing, and crackles in all lung fields. Which of the following is indicated in the treatment of this patient?
   a. Chest radiography
   b. Oral corticosteroids
   c. Inhaled β_2-agonist
   d. Nasal suctioning
   The correct answer is *d*.
2. A previously healthy 9-month-old infant is brought to the ED with "noisy breathing." He has had a "barky" cough for the last 3 days, which seems to be worsening, and he is now refusing all oral intake. On examination, he appears ill with audible stridor and appears to be drooling. His vital signs are as follows: T 40°C (104°F), HR 140, RR 45, BP 100/80, pOx 87% in room air. What is the most likely diagnosis?
   a. Foreign body
   b. Bacterial tracheitis
   c. Epiglottitis
   d. Laryngotracheitis
   The correct answer is *b*.
3. A 12-year-old girl, with a history of moderate persistent asthma, comes to the ED with a moderate to severe asthma exacerbation. She has received three combination nebulizers of albuterol and ipratropium, oral corticosteroids, and continuous albuterol for 2 hours, but she continues to have wheezing and tachypnea. Her vital signs are as follows: T 37.6°C (99.7°F), HR 120, RR 40, BP 110/80, pOx 92% in room air. What is the next best choice in additional treatment for this patient?
   a. IV epinephrine
   b. Aminophylline drip
   c. IV magnesium
   d. Intubation
   The correct answer is *c*.

PEDIATRIC GASTROINTESTINAL DISORDERS AND DEHYDRATION

CHAPTER 64

Joshua S. Easter, MD, MSc

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, physical examination, and occasionally diagnostic studies (Table 64-1).

2. What are the strongest indicators of dehydration in children?

Physical examination findings, such as delayed capillary refill time (>2 seconds), abnormal skin turgor, and hyperpnea are the strongest indicators of severe dehydration. Historical factors, such as the number of wet diapers, frequency of vomiting or diarrhea, and amount of oral intake are less predictive. Laboratory studies, such as elevated serum blood urea nitrogen (BUN) and creatinine levels, anion gap, and urine specific gravity are also poor predictors of dehydration.

3. How do you manage the different levels of dehydration?

- Mild dehydration (minimal clinical signs): Use oral rehydration with as much fluid as the child desires. Breast milk, formula, Pedialyte, and World Health Organization (WHO) solution are appropriate for fluid replacement. Straight water can lead to hyponatremia; highly sugared beverages, such as apple juice and soda, can exacerbate diarrhea.
- Moderate dehydration (normal or delayed capillary refill, normal or abnormal skin turgor, fatigue): Oral rehydration is the ideal treatment. Using WHO solution or Pedialyte, the patient drinks 50 to 100 mL/kg over 2 to 4 hours in the form of 5-mL aliquots administered via a medicine dropper or syringe every 5 minutes. An alternative 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 (IV) hydration and shorter times to initiation of therapy and ED lengths of stay. Consider IV hydration if the child has persistent vomiting or is refusing to drink.
- Severe dehydration (ill appearing): These children often require multiple boluses of IV normal saline to compensate for their dehydration and should receive 30 mL/kg over the first hour. In children without signs of shock, rapid IV hydration (60 mL/kg/h) is not more efficacious than standard bolus rehydration. Addition of dextrose to IV fluids reduces ketone levels more rapidly compared with normal saline but does not reduce the incidence of hospitalization.

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. For example, a 32-kg child should receive (4 mL/kg $\times$ 10 kg) + (2 mL/kg $\times$ 10 kg) + (1 mL/kg $\times$ 12 kg) = 72 mL/h of maintenance fluids. Most commonly, dextrose 5% in 0.45% normal saline ($D_5\frac{1}{2}NS$) with 20 to 30 mEq/L of potassium is used. Potassium should only be given after ensuring the patient is not in renal failure or hyperkalemia.

5. What role do antiemetics play in the management of pediatric vomiting?

Children who are vomiting and undergoing oral rehydration therapy should receive an antiemetic medication. Ondansetron reduces vomiting, need for IV fluids, and hospital admission. It can be given orally via a pill, liquid, or disintegrating tablet. It is well tolerated by children, has a limited side-effect profile, and does not mask other diagnoses such as appendicitis or intussusception.

6. What are potential causes of vomiting without diarrhea in children?

The differential of isolated vomiting is extensive and includes early gastroenteritis, urinary tract infection, appendicitis, diabetic ketoacidosis, otitis media, pneumonia, streptococcal pharyngitis, testicular or ovarian torsion, toxic ingestions, meningitis, and head injury.

Abstract

This chapter discusses pediatric gastrointestinal emergencies.

Keywords:

gastroenteritis, appendicitis, intussusception, pyloric stenosis, malrotation

Table 64-1. Differential of Nontraumatic Abdominal Pain by Age

Neonate	2 Months-2 Years
Malrotation	Incarcerated hernia
Necrotizing enterocolitis	Intussusception
Testicular torsion (may be undescended)	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
Streptococcal pharyngitis	Pelvic inflammatory disease
Henoch-Schönlein purpura	Gallbladder disease

7. **How do I 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. Red flags include bilious, bloody, and projectile emesis; focal tenderness in the abdomen; high fever; and tachycardia that does not improve with rehydration.

8. **What diagnostic studies should be obtained on children with gastroenteritis?**
   Most patients require no tests. Ill-appearing infants may have depleted their glycogen stores and require bedside glucose testing. In the setting of hypoglycemia, the infant or child should be given 4 mL/kg of 10% dextrose if younger than 3 months or 2 mL/kg of 25% dextrose if older than 3 months. Electrolyte studies, looking for hypernatremia or renal insufficiency, should be reserved for ill-appearing children.

9. **How do I differentiate between bacterial and viral causes of diarrhea?**
   Viruses cause the majority of diarrhea in children. Since the introduction of the rotavirus vaccine, norovirus is the most common pathogen. 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 historical and physical examination findings cannot reliably differentiate bacterial from viral diarrhea.

10. **Which children with diarrhea require diagnostic studies?**
    Stool cultures should be considered in patients with significant comorbidities, ill appearance, bloody stools, severe cramping, recent antibiotic use (also obtain *Clostridium difficile* toxin assay), travel, diarrhea lasting longer than 1 week (also obtain ova and parasite screen), or exposure to a patient with a known bacterial diarrhea.

11. **Are antimotility agents and antibiotics recommended for children with diarrhea?**
    - Antimotility agents should be avoided because of adverse events, particularly in young children.
    - Antibiotics should also be avoided, except for children with traveler's diarrhea or infections in which the pathogen identified is known to be amenable to treatment.
    - Eating solid food does not increase diarrhea. Lactose avoidance in young children may reduce the duration of diarrhea, because some will develop transient lactose intolerance.
    - Probiotics may reduce the duration of diarrhea.

12. **How does hemolytic uremic syndrome (HUS) typically present?**
    Within 2 weeks of a bloody diarrheal illness caused by enterohemorrhagic *Escherichia coli,* children develop anemia, thrombocytopenia, and acute renal failure. Several studies suggest treatment with antibiotics when children have diarrhea caused by enterohemorrhagic *E. coli* may increase the risk of HUS.

KEY POINTS: DEHYDRATION AND GASTROENTERITIS

1. Young children with gastroenteritis can become dehydrated easily.
2. Vomiting in the pediatric population has a broad differential, and gastroenteritis should not be assumed.
3. Most children can be successfully managed without diagnostic tests or IV fluids.

13. Should narcotics be withheld from children with acute abdominal pain while awaiting a surgical evaluation?

No, multiple studies have shown that diagnostic accuracy from physical examination increases when patients' pain is controlled. Pain management may also improve the diagnostic utility of ultrasound (US).

14. How does appendicitis present in younger children?

Appendicitis is the most common nontraumatic surgical emergency in children. The diagnosis of appendicitis is commonly missed in younger children, who often show nonspecific symptoms. Vomiting, abdominal pain, fever, diarrhea, irritability, and right hip pain are some typical presentations, often attributed to other causes. Similarly, children's physical examinations commonly reveal diffuse abdominal tenderness or abdominal distention, whereas pain localized to the right lower quadrant is less common. Appendicitis in the infant is typically recognized only after perforation, which occurs in 70% to 95% of these cases.

15. What physical examination findings are found in older children with appendicitis?

The most common findings are tenderness in the right lower quadrant over the McBurney point (two thirds of the distance along a line from the umbilicus to the anterior superior iliac spine). Rovsing 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 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.

16. 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 count of greater than 10,000/mm^3 has a sensitivity of 88%, but specificity of 53%; a WBC count of greater than 15,000/mm^3 improves specificity to greater than 60%, but the sensitivity declines to 19%. An elevated C-reactive protein level has similar sensitivity and specificity to a WBC count greater than 10,000 mm^3. A positive urinalysis cannot exclude appendicitis; 30% of children with appendicitis have pyuria or bacteriuria.

17. What are the advantages and disadvantages of the different radiographic tests for appendicitis?

- Plain films: Radiography is insensitive and nonspecific, normal in 82%, and should not be routinely obtained.
- US: If available, US should be the initial study of choice in children; no radiation and lack of peritoneal fat in children favors ideal imaging with this modality. With experienced sonographers, US provides a high sensitivity (92%) and specificity (98%). Appendicitis will show an appendiceal diameter greater than 6 mm, wall thickness greater than 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 modality. If the appendix is not visualized on US, this does not rule out appendicitis. These patients should be observed (in the ED or as outpatients) or undergo computed tomography (CT) depending on the level of concern.
- CT: CT is more sensitive than US for appendicitis, but has higher cost, potential need for sedation, and radiation exposure. In adolescents, CT without contrast is equivalent to CT with contrast for diagnosing appendicitis.

18. What is the treatment for appendicitis?

Appendectomy; for uncomplicated appendicitis, laparoscopic appendectomy reduces the risk of postoperative wound infection and shortens recovery time compared with open appendectomy. In contrast, for perforated appendicitis, open appendectomy reduces the risk of postoperative abscess formation. Broad-spectrum perioperative antibiotics should be administered in the ED, because they reduce the risk of operative complications. There is growing evidence that uncomplicated appendicitis may be managed nonoperatively with antibiotics.

KEY POINTS: APPENDICITIS

1. Appendicitis is rare in young children. It presents atypically, resulting in delays in diagnosis and high perforation rates.
2. Laboratory tests are rarely helpful in evaluating a patient with appendicitis.
3. US should be the first imaging study in children with suspected appendicitis. CT should only be used if US is equivocal and there is high clinical suspicion for appendicitis.

19. 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 irritability, during which children may pull their knees up toward their chest, is often the only symptom. Children may appear comfortable between these episodes. Currant jelly stools are a late, rare, and ominous finding from bowel ischemia; although earlier in the disease course, the stool is often guaiac positive. Younger children may exhibit nonspecific findings, such as altered mental status or lethargy.

20. How do I diagnose intussusception?

The classic crescent sign on plain radiography from the intussuscepting mass is rarely seen. Nevertheless, abdominal radiographs 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), the likelihood of intussusception is substantially reduced. US may identify a donut or target sign and has a sensitivity and specificity of nearly 100%. Air enema may be utilized to both diagnose and treat intussusception.

21. How should intussusception be treated?

Air enemas provide equivalent success rates (nearly 90%) to contrast enemas, with less radiation exposure. Lower rates of successful reduction occur with age younger than 3 months and older than 5 years, symptoms lasting longer than 48 hours, hematochezia, or signs of obstruction on radiography. Because there is a slight risk (<1%) of perforation with enema reduction, a surgeon should be available. Recurrence can develop, but less than 5% of recurrences arise within the first 24 hours. Therefore outpatient observation of well-appearing patients after reduction is appropriate. Patients with recurrence can undergo a second air enema. Patients with free air or peritonitis should undergo surgical reduction.

22. 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 midgut volvulus. Congenital malrotation of the midgut predisposes the bowel to twisting on itself, leading to bowel obstruction and vascular compromise, with bowel necrosis of the entire involved segment developing in as little as 2 hours.

Midgut volvulus classically presents with sudden onset of bilious emesis, abdominal pain, and later bloody stools; however, early in the course of illness, more than half of patients have only bilious emesis with normal abdominal examinations. Thus all infants with bilious emesis should undergo diagnostic testing regardless of their abdominal examinations. Although radiography can show small bowel obstruction, a double-bubble sign, or paucity of distal bowel gas with volvulus, plain films are often normal. An upper gastrointestinal (UGI) series with contrast is the gold standard and should be performed in all patients before malrotation is ruled out. 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, IV fluids should be given, a nasogastric tube inserted, and broad-spectrum antibiotics administered. Surgical consultation should be obtained immediately, because patients require emergent detorsion of the bowel.

23. 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 2 to 8 weeks of age. Infants vomit at the end of feeds or within 30 minutes. Unlike more severe conditions, such as malrotation, emesis is usually nonbilious because the stenosis is proximal to the duodenum. In addition, the patient will remain hungry and continue attempting to feed. The classically described palpable "olive" on right upper quadrant examination arising from the hypertrophied pylorus is rarely identified early in the disease or when the child is awake.

24. 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, 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 3 mm wide or more than 14 mm long. If the US is equivocal, then obtain UGI radiography, which will show a string sign as contrast travels through the narrowed pylorus.

Patients with pyloric stenosis require rehydration and surgical correction with pyloromyotomy, although in developing countries patients often can be supported long enough to allow the hypertrophy to resolve without surgery.

25. Are inguinal hernias dangerous?

One percent to 2% of children develop inguinal hernias; indirect inguinal hernias with bowel herniating into the inguinal canal or scrotum are the most common. Nearly 10% of hernias will ultimately incarcerate (become irreducible), and 60% of incarcerations occur in children younger than 1 year. If the incarceration persists, the bowel can strangulate, cutting off its blood supply and leading to bowel obstruction or necrosis. Patients with incarceration often have vomiting, irritability, and inguinal or scrotal swelling.

Unless bowel necrosis is already suspected, manual reduction can be attempted on incarcerated hernias to prevent strangulation. With the patient in Trendelenburg position and after administering appropriate analgesia, apply constant slow pressure to the internal inguinal ring, while the other hand milks fluid or gas in the hernia back into the abdominal cavity. Eighty-five percent of inguinal hernias can be reduced, and these patients can be safely discharged for outpatient surgical repair. If reduction is unsuccessful or strangulation suspected, consult a surgeon.

26. 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 and are typically painless. If you cannot differentiate a hydrocele from a hernia on examination, obtain a scrotal US. Hydroceles are benign and often resolve spontaneously.

KEY POINTS: SURGICAL EMERGENCIES IN YOUNG CHILDREN

1. Intussusception often presents with only intermittent irritability or vomiting. US is the diagnostic study of choice, although air enema is both diagnostic and therapeutic.
2. Bilious emesis in a neonate is a surgical emergency until proven otherwise, requiring a UGI radiographic series or surgical consultation.
3. Infants with pyloric stenosis have projectile nonbilious emesis but remain hungry and interested in feeding. US is the diagnostic study of choice.

27. Why is jaundice concerning in a neonate?

Although 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 myriad causes, including Rh or ABO incompatibility,

Table 64-2. Differential of Lower Gastrointestinal Bleeding by Age

Neonate	**2 Months-2 Years**
Swallowed maternal blood	Anal fissure
Allergic colitis	Allergic or infectious colitis
Infectious colitis	Intussusception
Volvulus	Meckel diverticulum
Necrotizing enterocolitis	Inflammatory bowel disease
2-5 Years	**6-18 Years**
Anal fissure	Anal fissure
Infectious colitis	Infectious colitis
Intussusception	Hemorrhoids
Polyps	Inflammatory bowel disease
Meckel diverticulum	Polyps
Inflammatory bowel disease	Angiodysplasia

prematurity, polycythemia, intestinal obstruction, sepsis, or dehydration. All patients with visible jaundice need a documented bilirubin level, and, if elevated, a search made for the etiology. This may include a blood type, Coombs test, and complete blood count (CBC). Patients with elevated age-specific bilirubin levels may require phototherapy, with the threshold for therapy based on the American Academy of Pediatrics phototherapy nomogram. Exchange transfusion should be considered for marked elevations (>25 mg/dL).

28. Is it normal for a child to have constipation?

It is normal for infants to strain during bowel movements and have varied amounts of time between bowel movements. 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 with constipation have more serious conditions. A thorough history and physical examination can give clues to some of these entities and should include onset of symptoms in the first week of life, emptying of the rectal vault on rectal examination, or noting failure to pass meconium within 24 hours of birth (Hirschsprung disease). Note abnormal tone, lethargy, and weak cry (botulism and hypothyroidism), as well as a 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. 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.

29. How can you treat constipation in the ED?

Most patients with constipation arising from a nonemergent 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 to 3 mL/kg/day), lactulose (1 to 2 mL/kg/day), magnesium hydroxide (Milk of Magnesia; 1 to 3 mL/kg/day), or polyethylene glycol (MiraLax; 1 g/kg/day) can be administered in the short term. Long-term management includes increasing fluid intake and adding fiber to the diet.

30. What are the most common causes of lower gastrointestinal bleeding in children?

See Table 64-2.

31. What is a Meckel diverticulum?

It is a remnant of the omphalomesenteric duct in children. It is the most significant cause of painless rectal bleeding in children and can lead to massive bleeding, diverticulitis, or intussusception. If suspected, a technetium-99m Meckel scan can identify ectopic gastric mucosal tissue, thus making the diagnosis.

Table 64-3. Management of Gastrointestinal Foreign Bodies

EMERGENT ENDOSCOPY	CONSULTATION WITH GASTROENTEROLOGIST
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 >24 hours	Multiple magnets

Table 64-4. Radiographic Findings in Pediatric Abdominal Pain

FINDING	RADIOGRAPHIC 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 or gastric bubble	Wall of pylorus >4 mm thick Canal >14 mm	Pyloric stenosis

32. What is Meckel's rule of twos?

Meckel 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; and average age at presentation is 2 years.

33. How do I 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 (Table 64-3).

34. What are the possible complications of an esophageal foreign body?

Airway obstruction, esophageal stricture, esophageal perforation, aorta-esophageal fistula, mediastinitis, or paraesophageal abscess can arise with esophageal foreign bodies.

35. How can I determine the location (trachea versus esophagus) of a coin in the pharynx?

Coins in the esophagus typically appear in the coronal plane (face on or round) on anteroposterior radiograph, whereas coins in the trachea appear in the sagittal plane ("on end" or "on fos"). However, this is not always accurate. If there is concern that the coin is in the trachea and operative intervention is intended, a lateral view should be obtained to confirm the tracheal position of the coin.

36. How can I differentiate a button battery and coin on radiograph?

Button batteries are round, up to 20 mm in diameter, and thus may be similar in size to pennies and nickels. It is easy to confuse these objects on radiograph. This can have devastating consequences, because a button battery lodged in the esophagus can lead to erosion and esophageal perforation. Unlike coins, button batteries are bilaminar and thus have a double ring or halo on radiograph. On lateral radiograph, button batteries have a step-off that is not present with coins.

37. What diseases are associated with classic findings on radiograph?

See Table 64-4.

Acknowledgment

The editors gratefully acknowledge the contributions of Mark Anderson, MD, author of this chapter in the previous edition.

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QUESTIONS

1. A 4-week-old male has bilious emesis. He is not interested in feeding but is well appearing, with a soft and distended abdomen. He is afebrile, with a heart rate in the 160s and unremarkable abdominal radiographic findings. What is the next most appropriate step in the management of this patient?
   a. Oral challenge
   b. Ultrasound of the pylorus
   c. Upper gastrointestinal series
   d. Admission to the hospital for observation
   The correct answer is *c*. This patient has bilious emesis and therefore may have malrotation with volvulus. This is a surgical emergency until proven otherwise. Abdominal radiographs are not sufficiently sensitive to rule out this diagnosis. As a result, patients with bilious emesis but normal radiographic findings still require surgical consultation and/or UGI series. Observation is not appropriate, because the bowel often will infarct within 2 hours of onset. Pyloric stenosis typically does not lead to bilious emesis.
2. A 5 year-old female comes to the ED after four episodes of nonbilious, nonbloody emesis in the last 12 hours. She is not interested in eating but has drunk 1 cup of juice throughout the day; her last bowel movement was slightly watery earlier today. On examination, she has a temperature of 38.3°C, heart rate of 120, blood pressure of 90/45, and weight of 19 kg. Her abdomen is soft and nontender, and her skin has normal turgor with a capillary refill of less than 2 seconds. What is the most appropriate initial management strategy for this patient?
   a. Administer 570 mL of normal saline and 2 mg of ondansetron IV.
   b. Obtain a CBC and basic metabolic panel.
   c. Administer 4 mg of oral ondansetron and encourage the patient to drink 20 mL of Pedialyte every 5 minutes.
   d. Obtain an US of the right lower quadrant to evaluate for appendicitis.
   The correct answer is *c*. This patient demonstrates signs of moderate dehydration (fatigue) but not shock. She should attempt oral rehydration therapy after receiving an oral antiemetic. Only if this regimen fails should an IV be placed and IV fluids given. Most children with vomiting do not require laboratory testing. This patient does not have abdominal pain or tenderness in her abdomen to suggest appendicitis, and therefore a US is not needed at this point.
3. A 4-year-old girl is brought to the ED after she told her mother that she swallowed a coin. She is refusing to eat but does not have any trouble breathing or swallowing her secretions. Her vital signs are normal, and an anteroposterior chest radiograph demonstrates an approximately 2-cm circular radiopaque object without a halo in the sagittal plane over her thoracic inlet. What is the next best step?
   a. Immediate consultation with a surgeon
   b. Obtain a lateral neck radiograph
   c. Use of a laryngoscope to attempt visualization in the ED
   d. No further action is required, because the object will likely pass spontaneously
   The correct answer is *b*. Although the depiction of the object in the sagittal plane on the anteroposterior view suggests it is in the trachea, a lateral neck image should be obtained to confirm this location. Most swallowed foreign bodies in children are in the gastrointestinal tract and lateral neck. Radiography often demonstrates that objects in the sagittal plane on anteroposterior view are in the esophagus. Consultation with a surgeon is appropriate if the object is in the trachea. Attempts to visualize the object with a laryngoscope are not recommended in the ED.

PEDIATRIC INFECTIOUS DISEASES

Roger M. Barkin, MD, MPH, FACEP, FAAP

CHAPTER 65

1. Are infectious diseases important to recognize in pediatric patients?
Infectious diseases account for a significant percentage of pediatric visits to the ED for acute illness. Although most conditions are self-limited and uncommon, 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)?
Measles is spread by direct contact with infectious droplets or airborne dissemination.

3. What is the incubation period for measles?
From exposure to the onset of symptoms, incubations lasts for 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 C's: 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 spots: 1- to 3-mm bluish-white spots on a bright red surface 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.
Complications include otitis media and bronchopneumonia. Encephalitis may occur as well.

6. What is subacute sclerosing panencephalitis?
Subacute sclerosing panencephalitis is a very 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, and explain why rubella is 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 are seen early in rubella; however, in contrast to Koplik spots, they are not pathognomonic.

9. What is the incubation period for mumps, and when is the patient contagious?
The incubation period 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.

Abstract

Unique infectious diseases are often encountered in the care of children. Many are less commonly seen with the effective immunization of children. Others remain prevalent and must be recognized, some requiring specific evaluation and life-saving management

Keywords:

rubella, roseola, mumps, diphtheria, pertussis, Reye syndrome, Kawasaki disease, erythroderma, infectious mononucleosis

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.
Erythema infectiosum is characterized by 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) has 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 to 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; 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 antibiotics are 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 more 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?
Heterophil antibodies are 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 then 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.

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 because of tonsillar hypertrophy
  - Sinusitis
  - Pneumonia
- Hematologic
  - Thrombocytopenia
  - Hemolytic anemia
  - Granulocytopenia
- Neurologic
  - Encephalitis
  - Cerebellar ataxia
  - Guillain-Barré syndrome
  - Transverse myelitis
  - Bell 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. They are 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, and how are they treated?

See Chapter 52.

25. 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 medium and tellurite agar cultures and Gram stain.

26. What is the therapeutic approach to management of diphtheria?

After ensuring stability of the airway and confirming 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 patient who is allergic to penicillin. Carriers should be treated with antibiotics.

27. What clinical findings must be present to make the diagnosis of Kawasaki disease?

Kawasaki disease is a multisystem disease occurring predominantly in children younger than 5 years. It 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.

28. What is the most significant complication of Kawasaki 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 high dose aspirin and intravenous (IV) immune globulin.

29. What infectious conditions should be considered in a child with diffuse erythroderma?

Several acute infectious entities may present with diffuse erythroderma, several of which may be potentially life threatening.

- A scarlatiniform rash caused by group A *Streptococcus*
- A viral illness
- Scalded skin syndrome *(Staphylococcus aureus)*
- Toxic epidermal necrolysis or erythema multiforme caused by a variety of infections and drugs
- Kawasaki disease
- Toxic shock syndrome *(S. aureus)*
- Leptospirosis

See also Table 55-3.

30. 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.

- In the initial catarrhal phase, patients have respiratory complaints of fever, rhinorrhea, and conjunctivitis lasting 2 weeks. Treatment is more efficacious if started during this stage.
- The paroxysmal phase follows, with unremitting coughing paroxysms accompanied by vomiting that 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.

31. What are the typical stages of Reye syndrome?

Reye 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 *Reyelike syndrome*. Salicylate ingestion has been incriminated, especially when occurring in association with chickenpox or influenza. Clinically, patients have a respiratory or gastrointestinal prodrome followed in several days by 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 common cause of ED visits for children. It is important to differentiate self-limiting 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.

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QUESTIONS

1. Which of the following is not a sign of measles?
   a. Bluish-white spots on a red surface near the molars
   b. Rash spreads from trunk, migrating peripherally
   c. Associated with conjunctivitis, coryza, and a high fever
   d. Two weeks from exposure to onset of rash
   The correct answer is *b*.
2. Which of the following must be present to make a clinical diagnosis of Kawasaki disease?
   a. Fever
   b. Desquamation
   c. Lymph adenopathy
   d. Perioral findings
   The correct answer is *a*.
3. Which of the following statements is false?
   a. In IM, splenic rupture typically occurs in the second or third week of illness.
   b. Mumps can lead to sterility.
   c. The monospot test is more sensitive in infants.
   d. Pertussis is most effectively treated in the catarrhal phase.
   The correct answer is *c*.

CHAPTER 66

EMERGENCY DEPARTMENT EVALUATION OF CHILD ABUSE

Kathryn Wells, MD, FAAP, and Daniel Lindberg, MD

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 community norms and values. Importantly, this definition distinguishes child abuse from maltreatment by someone without responsibility for the child (e.g., strangers). This distinguishes sexual abuse from rape, and physical abuse from assault. Whereas this distinction may have minimal impact on the medical evaluation of injured children, it can become important when it comes to reporting the situation to child protective services (CPS) or law enforcement.

- Physical abuse 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 that 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.

PHYSICAL ABUSE

2. When should I think about physical abuse?

Because one cannot rely solely upon a caregiver's history, and because physical examination findings can be subtle, physical abuse can be remarkably difficult to detect and is commonly missed. Although the short answer of "think about abuse in any child" is both correct and easy to remember, it is not particularly helpful. Whereas physical abuse can occur at any age, both fatal and nonfatal abuse is exponentially more common in younger children. Risk is very high in children who are not old enough to provide their own history (<3 years old), especially in children who are immobile (<6 months old). Young parents, poverty, time of emotional stress, and an unrelated caregiver living in the home are other risk factors.

3. What are some red flags that suggest child abuse?

- Injury unexplained by history or inconsistent with child's developmental age
- Absent, changing, or evolving history
- Unreasonable delay in seeking care
- 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

Abstract

Child maltreatment is both more common and more difficult to identify than almost any other pediatric illness with similar morbidity and mortality rates. Caregivers are unlikely to provide an accurate history, and clinical signs are both subtle and insensitive. Evaluations of potentially abused children are often far more intense than in children with accidental injuries, because even injuries that are clinically mild may be important forensically. At the same time, it is rarely possible in the course of an ED stay, and almost never necessary, to ultimately determine whether a child has been abused. This chapter outlines the role of the emergency physician in the care of potentially abused children and describes best practices for testing and treatment.

Keywords:

child abuse, physical abuse, sexual abuse

4. Are there injuries that are particularly concerning for physical abuse?

The following list includes those presentations that should always prompt a consideration of abuse if they seem to have occurred without a severe mechanism (e.g., motor vehicle collision [MVC]):

- Any bruising in a child younger than 6 months or in an infant that is not "cruising"
- Bruising to the ears, neck, torso, cheek, or eyelid
- Oral/pharyngeal injury without a clear mechanism (e.g., running with a toothbrush)
- Intraabdominal injury
- Long bone fracture in an infant (<12 months old)
- Intracranial hemorrhage
- Rib fractures
- Classic metaphyseal fracture
- Multiple fractures of different ages
- Patterned bruises or burns (bite marks, injury looks like an implement)

Any injury that does not have a history or does not fit the history provided should raise your suspicion for abuse. Remember the developmental stages of infancy and childhood, and ask yourself if the child could have done what the caregiver is reporting. For example, a 2-month-old infant cannot roll over, so he or she likely did not roll off the couch and sustain a fracture.

5. Why do we do so much more testing for abusive injuries than we do for noninflicted trauma?

- Forensic significance: Identifying a single rib fracture in a child that is abused (especially if it is healing) can dramatically affect the recognition of abuse.
- Escalating abuse: Children in whom abuse is missed are commonly returned to dangerous environments where abuse may continue or worsen.
- Additional victims: Violence is a disease that affects households. Identifying abuse in a child can prevent abuse for their siblings, parents, or even pets.
- Long-term effects: For survivors, abuse can have serious, pervasive effects on future health.

6. Once I think about abuse, what parts of the physical examination are most important?

Especially for young infants, even a thorough physical examination can miss serious injuries. Nevertheless, all children who raise a concern for abuse should have a careful examination, including:

- Oropharynx: Examine the lips, frenula (labial and lingular), teeth, palate, tongue, and pharynx.
- Ears: Look inside and behind the pinna.
- Fontanel, when present: Look for bulging as a sign of increases intracranial pressure.
- Eyes: Look for subconjunctival hemorrhage, blue sclera (a sign of osteogenesis imperfecta if there are fractures), and retinal hemorrhages.
- Skin: Examine all surfaces of the body.
- Genitalia: Take off the diaper.
- Growth chart, if available, especially the head circumference: Make sure to chart a good height, weight, and head circumference, because poor growth can be a sign of neglect.

7. Which children need a skeletal survey?

A skeletal survey is a series of radiographs designed to evaluate each bone individually, and is used in cases of suspected abuse in preverbal children. A "babygram" (single anteroposterior view of the infant's body) is not an adequate skeletal survey. A skeletal survey is mandatory for all children younger than 2 years when there is concern for abuse. Keep a low threshold for obtaining this study for children younger than 3 years. Skeletal surveys are not very useful in children older than 5 years, unless the child has decreased mobility or communication. A quality skeletal survey is around 20 films (at least two views of the skull and spine; anteroposterior, lateral, and oblique views of the ribs; and at least one view of the humeri, forearms, hands, femora, tibias, and feet) and should be read by an experienced radiologist. Repeat radiographs (particularly rib films) in 2 weeks often show healing fractures not seen acutely on the original skeletal survey; close follow-up observation of infants undergoing workup for abuse is therefore important.

8. Why are metaphyseal fractures suggestive of abuse?

Also called *bucket handle*, *corner*, or *metaphyseal chip fractures,* metaphyseal fractures in young children are strongly suggestive of 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. 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.

9. Which children need neuroimaging (computed tomography [CT] or magnetic resonance imaging [MRI])?

Brain injuries are common in abused children and can be easy to miss. Several studies have shown high rates (37%) of occult brain injury in young children being evaluated for abuse. Neuroimaging should be obtained for infants younger than 6 months and considered for infants younger than 12 months who have the following indicators:

- Facial bruising
- Multiple fractures
- Rib fractures
- Signs of head injury on examination or altered mental status, seizure, coma, or a history of shaking or trauma involving the head.

CT is the most commonly used imaging technique, but MRI is a reasonable alternative if there is a concern for radiation. Ultrasound is not an acceptable substitute.

10. Which children need a dilated retinal examination?

Although it is noninvasive to look at a child's retina, significant retinal hemorrhages are rare for children without brain injury. If the head CT is normal (or reveals a simple linear parietal skull fracture) it is almost never necessary to have a dedicated retinal examination performed by a pediatric ophthalmologist.

Note: The opposite is not true; retinal examination should not be used to determine which children need neuroimaging; an important fraction of children with head trauma have no retinal hemorrhages.

11. Which children need screening for occult abdominal injury?

Abdominal injuries are a distant second to head injuries as a source of death for abused children, but not so distant that they can be ignored. Clinical signs are very specific, but not sensitive for abdominal injuries. The current recommendations are to obtain aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels for all children with concern for physical abuse, and abdominal CT for those where AST or ALT is greater than 80 IU/L, or for those with abdominal bruising, tenderness, or distension, or with a report of abusive abdominal trauma. The yield of CT using this protocol is greater than 20% and vastly outweighs the risk from radiation. AST and ALT normalize over hours or days, regardless of whether an injury is present, so rechecking these tests is not a reasonable alternative. Ultrasound has limited sensitivity for solid organ injuries and should not supplant CT.

12. What are the most common myths about physical abuse?

- Spiral fractures are especially concerning for abuse.
  - Spiral fractures imply a twisting or torqueing force about the axis of the bone but are not themselves more concerning than another fracture morphology. In fact, one of the most common accidental fractures (the toddler's fracture) is often a spiral fracture of the proximal tibia.
- A bruise's age can be determined by its color.
  - Although this is biochemically intuitive, it is just not true. In fact, when studied, providers often do not even agree on what colors are present in a bruise. Differences in a bruise's location and depth and the patient's complexion may account for this. Providers are notoriously bad at estimating a bruise's age. Often, the best way to estimate the age of a bruise is by reports of independent witnesses who would have noticed bruising if it was present.
- Mixed-density subdural hematomas (SDHs) imply multiple injuries.
  - Although this has been commonly believed, data suggest it is also not true. Studies of children who have SDHs as a result of MVCs show that mixed-density (bright and dark blood) SDHs can occur after a single episode of trauma. This might be because the cerebrospinal fluid (CSF) mixes with the blood, or because there is an area of hyperacute bleeding. Whatever the reason, do not speculate about the age of an SDH based on its appearance on CT.

13. Which conditions can mimic injuries seen in child abuse?

- Accidental injuries: Obtain detailed history and mechanism.
- Coagulation disorders: Determine prothrombin time (PT)/partial thromboplastin time (PTT) and platelet levels if bruises, retinal hemorrhages, or intracranial bleeding is present.
- Bone fragility syndromes (e.g., osteogenesis imperfecta, rickets, osteopenia of prematurity): Start with calcium, phosphorus, 25 hydroxy (25[OH]) vitamin D if the concern for abuse stems only from fractures.
- Collagen disorders (e.g., Ehlers-Danlos syndrome): A good physical examination and family history help rule these out.
- Other dermatologic conditions: Consider purpurae/petechiae from infection or mongolian spots.
- Phytophotodermatitis: A burnlike rash occurs at sites of citrus/plant juices on skin exposed to sun.
- Traditional remedies
  - Coining: Vigorous rubbing with a coin results in linear ecchymoses.
  - Cupping: Circular suction ecchymoses.

14. What about an infant who is dead on arrival without external signs of injury?

Any child who arrives at the ED in this way should be described as having had 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, because further information may not uncover any other cause for the death, or the cause may be a previously unidentified medical illness.

15. What should I do about siblings and other children in the home?

Violence is a disease that affects the entire household. Routinely ask about other children who live in the home of a child suspected of being abused or who dies under concerning circumstances. CPS can assist with arranging examinations of other children. For children who raise a high level of concern for abuse and at least one serious injury (e.g., fracture, brain injury), perform skeletal surveys for other children younger than 2 years who are living in the home.

SEXUAL ABUSE

16. If a child has not reported sexual abuse, what should make me think about it?

Most ED evaluations for sexual abuse are the result of concern by a caregiver or a report of abuse by the child. However, the possibility of abuse should also be considered for children who show the following signs:

- Genital injury or bleeding (excluding hemorrhagic cystitis in a child with a urinary tract infection)
- Genital discharge
- New behavior changes (enuresis, nightmares, behavioral regression, mood swings)
- Siblings or other children who share a household with a child who has been sexually abused
- Sexualized behaviors

Whereas several surprising behaviors (touching genitals or masturbating, even in public) are actually often normal in children, asking to engage in sexual acts, inserting objects into the genitalia, imitating intercourse, touching animal genitals, and exhibiting behavior that is persistent, resistant to distraction, or a cause of emotional or physical distress are abnormal and should prompt concern for abuse.

17. When there is a report of abuse, should I take any history?

To be useful for medical and legal purposes, forensic interviews require training, experience, and ongoing peer review beyond the resources of most emergency physicians. However, it is appropriate to obtain history from the child's caregiver separately from the child to determine the reason for the concern and how (and if) the concept of abuse was raised to the child. Most emergency physicians should limit questioning of the child to the information necessary to determine whether there is a need for acute intervention (e.g., Is the child in pain? Are there other symptoms or signs of infection or injury? Is there recent contact and possible body fluid exposure prompting need for evidence collection or infection/pregnancy prophylaxis?). Providers should familiarize themselves with local

processes and resources so that a full forensic interview may be obtained later (e.g., child abuse team at a referral center, CPS, and children's advocacy center). Spontaneous outcries from a child should be documented as much as possible in the child's own words.

18. How should I question a child to obtain history and direct my evaluation?

Questioning should be based upon the developmental abilities of the child. Interviews are rarely possible with children younger than 3 years. Children younger than 6 years commonly have the most difficulty with "when" questions but do better with "what" or "who" questions. For example, a 5-year-old child will be unlikely to tell you that an assault happened on Saturday, but they would be more likely to tell you that it happened at their friend's birthday party or that the football game was on television at that time. Questioners should take care to use concepts and vocabulary familiar to the child and should emphasize open-ended, nonleading questions. Providers may find it helpful to ascertain the child's vocabulary for genitalia and other body parts. Useful queries include:

- Tell me more about that.
- Then what happened?
- How did/does that make your body feel?

19. Which children need a genital examination immediately?

Children with acute physical complaints in the setting of possible sexual abuse (bleeding, pain, dysuria, hematuria, discharge) or those who report sexual abuse within the past 72 hours should have an examination at the time of presentation. Other children are unlikely to have acute findings and could reasonably be referred for examination in a clinic setting by a child abuse pediatrician, child advocacy center, or a primary care physician. Never force a genital examination on a resistant child. For children with concern of major, acute injury (substantial bleeding, ill appearing), arrange to conduct an examination while the child is anesthetized.

20. How should I do the genital examination?

Again, remember to never force a genital examination on an uncooperative child. Integrating the examination into the rest of the physical examination and using a matter-of-fact tone can reassure the child. Helpful phrases include:

- I am going to check all of your body from the tip of your head to the tip of your toes and all your parts in between.
- This is OK because I'm a doctor, and because Mom is here, and because Mom says it is OK.

Most examinations can be completed in the supine "frog-leg" position, with heels together and knees on the table. Many small children are more comfortable in their parent's lap. For girls, grasp the labia majora between the thumb and the distal interphalangeal joint of the index finger. Pull the labia toward yourself (as opposed to pulling laterally) with the same amount of force one would use to retract someone's lip or cheek to examine their teeth. By convention, findings are described using a clock face, where the navel is at 12 o'clock and the anus is at 6 o'clock.

Prepubertal girls will have a hymen that is smooth and thin. This tissue is exquisitely sensitive, and the examiner should take care not to contact it with swabs, probes, or other foreign bodies. The speculum is rarely useful in the forensic medical examination and should never be used for the prepubertal child without anesthesia. By contrast, pubertal girls will have a thickened, redundant hymen that is less sensitive to contact.

21. What examination findings are most significant for sexual activity/abuse?

In most cases, the history given by the child, a witness to the sexual abuse, or evidence found at the scene will be the determinative factor for the confirmation of sexual abuse. Even in cases where abuse has occurred, the vast majority of examinations, especially outside of the acute phase, will be normal. In this situation, the documentation can reflect that a normal examination does not rule out sexual abuse. In the emergency setting, it is most important to note acute bleeding, contusion, or abrasion of the genitalia or anus. In this situation, if a regional child abuse specialist or team is available, they should be contacted for further documentation and assistance. Other nonacute findings, such as hymen transections, should be referred to a regional child abuse specialist to be examined nonacutely. Finally, in an adolescent who is able to consent to sexual activity, no finding can conclusively differentiate between consensual and abusive sexual activity. For documentation, the finding should just be described and not interpreted.

22. Which children need an evidence collection kit?

Child victims need a full physical examination (looking for trauma) and possibly evidence collection, depending on the incident. Children with history of sexual contact in the last 72 hours, which may have included secretion of bodily fluids that may contain DNA (saliva, semen, blood), should be

offered evidence collection. Eligible children should have evidence collected as soon as possible, because the utility of the kit decreases within the 72-hour window. Young children rarely need a full rape kit examination performed, and collection should be specific to the allegation and examination. Vaginal speculum examinations should not be done on a child or prepubertal teen. Rarely, children with severe genital trauma require sedation for examination and repair in the operating room. The highest yield component of the kit is the child's undergarments (even if they are not the same as the garments worn at the time of the assault).

23. Which children need testing for sexually transmitted diseases (STDs)?

Because the presence of an STD can dramatically affect the recognition of sexual abuse, testing for gonorrhea or *Chlamydia* seems reasonable for all prepubertal children with discharge and all pubertal children in whom a genital examination is undertaken. Additional testing for hepatitis B and C viruses (HBV, HCV), HIV, and syphilis may be considered as well. Testing is especially useful with:

- A history of multiple assailants
- Known disease or risk factors in the assailant
- Acute physical findings
- Known disease in another child suspected to have been abused by the same perpetrator
- Presence of another STD (even those that are less specific, such as human papillomavirus [HPV] or herpes simplex virus [HSV])
- Patient or parental concern.

Cultures remain the gold standard for prepubertal children who have been sexually abused, but if swabs are used to collect samples, use extreme caution to avoid contact with the prepubertal hymen, which is exquisitely sensitive to contact. Nucleic acid amplification testing (NAAT; e.g., amplicon or Gen-Probe) using "dirty catch" urine has been shown to have increased sensitivity relative to culture and is much easier to obtain in toilet-trained children.

24. Should children suspected of sexual abuse receive empiric treatment for STDs?

For several reasons, puberty is the bright-line distinction here. Although empiric/prophylactic treatment for gonorrhea and *Chlamydia* is reasonable for many adolescents and young adults, do not empirically treat prepubescent children for gonorrhea or *Chlamydia.* Whereas early treatment in older children can prevent ascending pelvic infections (e.g., pelvic inflammatory disease [PID], tuboovarian abscess), physiologic differences make these ascending infections vanishingly rare in prepubescent children. The presence of an STD in a prepubertal child will usually have a profound impact on the recognition of sexual abuse. Because high-stakes decisions (criminal prosecutions, child custody arrangements) can often hinge on the presence or absence of an STD, results of some diagnostic tests are often questioned in legal settings, requiring repeat testing.

Postexposure prophylaxis (PEP) for HIV is more complicated. The risk of transmission of HIV by sexual abuse is currently unknown and depends on the specific factors related to the child, the assailant, and details of the assault. It is reasonable to consult with a child abuse specialist/team and/or infectious disease specialist before initiating PEP in the setting of a possible sexual assault. In the United States, the Centers for Disease Control and Prevention (CDC) has established a 24-hour hotline for PEP (PEPline; 1-888-448-4911) that can give expert advice about the need for PEP for HIV, HBV, and HCV.

25. Which children need pregnancy testing and/ or prophylaxis?

A pregnancy test should be obtained in pubertal or near pubertal victims of sexual assault. Pubertal children who have experienced sexual assault within the previous 120 hours (5 days) should be offered pregnancy prophylaxis using levonorgestrel (Plan B) 1.5 mg as a single dose. A repeat dose may be used if vomiting occurs within 2 hours. Because the mechanism of action is suppression of ovulation, patients may be reassured that levonorgestrel does not terminate established pregnancies. In the United States, levonorgestrel can be obtained over the counter by women 17 years of age and older.

26. Which findings can mimic sexual abuse?

A broad range of infectious and inflammatory diseases and normal variants can cause genital findings in children, raising the concern for sexual abuse. Among the most common are:

- In boys who stand to urinate, a falling toilet seat can cause bruising to the tip of the penis that may mimic a bite mark.
- In girls, excessive or colored (e.g., yellow or green) vaginal discharge should prompt concern for sexual abuse, but bacterial infections (e.g., *Streptococcus, Escherichia coli, Enterococcus, Salmonella,* among many others), yeast, foreign bodies, or poor hygiene can also cause discharge.

- Mild erythema in the genital area or urinary tract infections are nonspecific findings in children and may be related to common hygiene issues, such as poor bathing or improper wiping.
- Vaginal ulcers or vesicles can prompt a concern for HSV, but also can result from Epstein-Barr virus (EBV), cytomegalovirus (CMV), inflammatory bowel diseases, or vaginal aphthosis.
- Straddle injuries can cause bruising and abrasion to the external genitalia or to the area between the labia majora and minora but rarely cause injury to the hymen, fossa navicularis, or internal genitalia.
- Urethral prolapse can cause dysuria and vaginal bleeding. The provider performing the genital examination should always take care to visualize the urethral orifice to determine whether the mucosa appears beefy red and inflamed.
- Venous pooling about the perianal plexus is commonly interpreted as bruising. This finding should rapidly resolve over a few minutes, especially if the patient is able to defecate.

27. What information should I give to parents?

Concern for sexual abuse can result in information overload for a caregiver. Parents may be unable or reluctant to ask some basic questions. Anticipatory guidance or discharge instructions should emphasize the following points:

- Tell parents not to perform their own forensic interview, because this can interfere with other investigations. However, parents should create a safe and open atmosphere if the child approaches them about the event. Outcomes have been shown to be improved for children with at least one caregiver who believes and supports them.
- Let caregivers know that, in the vast majority of cases, the genital examination will be normal, will have normal variants, or will show findings that will heal completely. In these cases, it is helpful to inform the patient and caregiver that they are completely normal and healthy and that, even when the examination does not determine whether abuse has occurred, it certainly does not exclude the possibility of abuse either. It is also useful to point out that no one in the future—spouses, doctors, sports teammates who share a shower—would ever know what had happened merely by looking at the child.
- Reassure caregivers who are worried about whether their child is still a virgin, even if they are reluctant to ask the question. In addition to the advice above, we find it useful to share our belief that a person stops being a virgin when they *choose* to have sex.
- Provide contact information to local CPS and/or child protection teams. If counseling referrals are not available through the ED, parents may be counseled to discuss this with their primary care physician or the child protection team.

REPORTING CONCERNS FOR CHILD ABUSE

28. 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. The health care provider's job is to notify authorities for suspicion of abuse, not to prove a child is being abused. As a mandated reporter, it is the legal obligation to report disclosures to authorities for further investigation. Remember that the medical team's observations are a single snapshot of a child, not an all-encompassing encounter, so you should be careful making assumptions about the appropriateness of a caregiver's behavior or the likelihood of a family member to abuse a child.

29. When should I report my concerns to CPS?

It is seldom possible, and almost never necessary, to conclusively determine whether or not abuse has occurred during an ED visit. In the United States, physicians have a legal mandate to report any reasonable concern for child maltreatment to public CPS agencies. The definition of *reasonable concern* for child maltreatment is not black and white, and doctors who consider and reject the diagnosis of abuse are not mandated to report. Regulations differ by jurisdiction in terms of who must report and when, but one common factor is that a physician may not choose to avoid reporting because they prefer to address the problem themselves, or because they are worried about the effects of involving CPS. In some cases, a physician may be faced with a high level of parental concern for abuse, despite a lack of other signs of abuse on their own history, examination, or testing. In these situations, it is important to remember that CPS reports may be made by anyone, not just physicians. Doctors who do not feel that they have identified a reasonable concern for abuse might provide CPS contact information to concerned parents.

30. How do I go about reporting?

Each jurisdiction has a different procedure for reporting concerns for child maltreatment, but in most cases, a report involves a phone call to a public hotline, followed by a written summary of the key information that caused concern for abuse. An Internet search for "report child abuse [insert state/territory]" is usually sufficient to find specific reporting instructions. When available, child protection teams or clinical social workers may assist in reporting abuse. Because the hotline will have to determine the jurisdiction of the case, doctors may need the following information at the time of the report:

- Child's identifying information (name, birthdate, address)
- Parent/caregiver's information (names, birthdates, addresses, phone numbers)
- Location of the alleged abuse (if known)
- Any information about the alleged perpetrator (name, address, age)
- Presence of other children in the home or exposed to the alleged perpetrator

31. Can I be sued for reporting when the child is not found to be abused?

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. Privacy laws, specifically Health Insurance Portability and Accountability Act (HIPAA), provide specific exemptions for sharing information specific to the situation while making a good faith report to authorities. Researchers operating under a certificate of confidentiality are usually not exempt from the duty to report reasonable concerns for child maltreatment. For more information on local and national resources for reporting, responding to, and preventing child abuse, visit the Child Welfare Information Gateway website (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.

KEY POINTS

- The highest risk for physical abuse is in children who are not old enough to provide their own history (<3 years old), especially in children who are essentially immobile (<6 months old).
- Injuries with very little clinical significance can have very important forensic significance. Even if a metaphyseal fracture is likely to heal on its own, identifying the fracture can help protect the child from future abuse.
- Obtain a skeletal survey for all children younger than 2 years if there is concern for physical abuse. If your center does not routinely perform skeletal surveys, this may require transfer to another center.
- Do not force a genital examination on a reluctant child. The vast majority of examinations will be normal, even when abuse has occurred.
- Do not give empiric treatment for gonorrhea or *Chlamydia* in prepubertal children. Wait until the diagnosis has been confirmed.
- Whenever there is a reasonable concern for abuse, physicians in the United States and Canada have a legal mandate to report these concerns to CPS.

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QUESTIONS

1. In a physically abused child, which of the following can help determine when the abuse occurred?
   a. The color of bruises
   b. The appearance of fractures on radiograph
   c. The appearance of subdural hematomas on CT
   d. The appearance of retinal hemorrhages
   The correct answer is *b.* Determining the age of an injury often affects the perceptions of whether abuse has occurred and who the perpetrator was. However, scientific evidence has debunked old myths that the color of a bruise or the appearance of subdural blood on CT can reliably determine the age of the injury. Similarly, it is not possible to estimate the age of retinal hemorrhages (though the ones associated with birth almost always resolve within a month). In almost all bones (not the skull), fractures begin to show signs of healing, such as periosteal reaction, after 1 to 2 weeks.
2. In a 4-month-old infant who fell 3 feet from a changing table to a hardwood floor, which of the following fractures would be most concerning for abuse?
   a. Rib: Nondisplaced fracture near the spine
   b. Clavicle: Mildly angulated fracture of the middle third of the bone
   c. Skull: 10-cm linear fracture on the right parietal bone
   d. Femur: Buckle fracture of distal femur, not involving the growth plate/metaphysis
   The correct answer is *a.* Any of the above fractures could be the result of abuse, and none is pathognomonic. It might be reasonable to do other testing and/or report for any of them. However, clavicle, simple skull, and distal femur buckle fractures could quite plausibly result from the fall described. In children, rib fractures are extremely uncommon, except with abuse and MVCs, and should always prompt a careful consideration of abuse.
3. A mother brings her 6-year-old daughter to your ED at midnight with concerns of sexual abuse. The mother reports that the child told her tonight that her camp counselor touched her privates when she was away at sleep-away camp 2 weeks ago. The sleepy child refuses to talk to you and resists a physical examination. What needs to be done in the ED tonight?
   a. Administer empiric treatment for STD (ceftriaxone/azithromycin).
   b. Perform a genital examination, under anesthesia if necessary.
   c. Obtain a forensic evidence collection kit (rape kit).
   d. Call CPS to report concern for sexual abuse.
   The correct answer is *d.* Even though abuse has not yet been conclusively proven, there is a reasonable concern for abuse that triggers your mandate to report. Such a report can allow CPS to initiate an investigation but does not imply that abuse is definite or that a diagnosis has been made. Prepubertal children should not be treated empirically for gonorrhea or *Chlamydia,* and PEP for HIV is not indicated after 72 hours. A genital examination is unlikely to change management after 2 weeks and, in any case, should not be forced on a reluctant child. The examination can reasonably be deferred to the child's pediatrician or child advocacy center and is likely to be easier in the light of day with a child that has been rested, fed, and prepared. Forensic evidence collection is unlikely to be useful after 72 hours.

CHAPTER 67

PROCEDURAL SEDATION AND ANALGESIA OF THE PEDIATRIC PATIENT

Joe Wathen, MD, and Guy L. Upshaw, MD

1. Why is it called *procedural sedation and analgesia (PSA)*?

What used to be called *conscious sedation* is now more accurately referred to as *PSA.* This is described 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 opioids, 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?

Frightening or painful procedures can be better tolerated by children with the use of sedatives and analgesics. 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 in situations 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 PSA TO CHILDREN?

1. Relieve fear and anxiety.
2. Provide needed analgesia.
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?

- *Minimal sedation (anxiolysis)* refers to a drug-induced state in which patients respond normally with very little to no depression of level of consciousness (LOC), and is ideal for nonpainful procedures in the older anxious child.
- Moderate sedation/analgesia, previously considered *conscious sedation,* is a drug-induced, depressed LOC in which patients respond purposefully to verbal or light 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).

Abstract

Revised chapter on procedural sedation and analgesia (PSA) in the pediatric patient presenting to the ED has been uploaded.

Keywords:

procedural sedation and analgesia (PSA), pediatrics, emergency medicine

- Deep sedation/analgesia is 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 is at the end of this continuum, and many sedatives can achieve it if given in sufficient doses. This is not desirable, because of the risk of cardiorespiratory depression, loss of airway reflexes, and potential for 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 with minimal effect on airway 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 IM route may be ideal (e.g., IM ketamine). Likewise, if anxiolysis or mild sedation is needed, oral or nasal midazolam may be sufficient. For mild to moderately painful procedures, intranasal fentanyl may be useful.

7. What is key information to obtain in the medical history before beginning PSA?

Information is focused sedation history.

SAMPLE

- **S**igns/symptoms: Respiratory infections or obstruction (snoring/stridor), heart disease, reflux?
- **A**llergies: Allergies to any sedatives or analgesics, egg, soy, or latex?
- **M**edications: Concurrent use of medications (e.g., narcotics; additive or resistant effects)?
- **P**ast medical and sedation history: Chronic medical issues (seizures, chronic lung disease), prior sedation or anesthesia problems?
- **L**ast meal, liquid intake (aspiration risk): When was the last oral intake of liquids and solids?
- **E**vents leading to sedation: Head injury, ingestion, or falls?

8. Are there guidelines for presedation fasting?

There are official guidelines for elective procedures per the American Society of Anesthesiologists (ASA). However, adherence to these presedation fasting guidelines has not been shown to alter the rate of adverse events. 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 before 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 PSA regardless of fasting status or underlying patient risk factors" (2005).

9. What physical examination findings are important to note before providing PSA?

- Items to note are the presence of airway abnormalities, such as large tonsils or adenoids; congenital abnormalities that may cause 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 an adverse respiratory event.

- A visual inspection of the open mouth will tell you what the upper airway looks like (Mallampati score) and will remind you to look for loose teeth or dental hardware (retainers).
- A careful cardiac and neurologic examination 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
- 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 poorly controlled seizure disorders
- Children with vomiting or gastroesophageal reflux
- Children with severe systemic disease
- Children with anticipated complicated and lengthy procedural needs best performed in the operating room

11. What monitoring 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 LOC, 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
- Properly sized bag-mask ventilation connected to oxygen source
- Properly sized advanced airway equipment (i.e., endotracheal tubes and laryngoscope)

Sedation is a continuum; therefore safety and monitoring guidelines should include being able to rescue a patient form a deeper level of sedation than initially intended.

12. What are the agents used for pediatric PSA?

Knowledge of specific medications and comfort with their use is critical to safe PSA practice (Table 67-1).

13. What agents would I use if I 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, have faster diagnostic ability with the possibility of CT being performed without sedatives. If medications are needed, sedatives alone are usually adequate. Potential agents include pentobarbital (Nembutal), midazolam, or methohexital. 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. 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, and in many institutions are administered by a sedation service or anesthesia department because of long monitoring times.

15. What are the advantages and disadvantages of propofol for PSA?

See Table 67-2.

16. What medications would I use for a 2-year-old child with a facial laceration?

For the majority of patients, local anesthetic such as topical lidocaine, epinephrine, and tetracaine (LET), or local injection with lidocaine is sufficient. The difficulty lies in reducing the child's anxiety.

Table 67-1. Procedural Sedation and Analgesia Agents

AGENT	DOSAGE	ROUTE	COMMENT
Anxiolytics			
Midazolam	0.1 mg/kg	IV, IM	Titrate to effect
	0.2-0.4 mg/kg	IN	10-12 mg max (mucosal atomizer ideal administration)
	0.5 mg/kg	PO, PR	15 mg max
Sedative Analgesics			
Fentanyl	1-3 µg/kg	IV	Avoid rapid or high-dose infusion
	1-2 µg/kg	IN	
Morphine	0.1 mg/kg	IV, IM	
Meperidine	1 mg/kg	IV, IM	
Dissociative Agents			
Ketamine	1-2 mg/kg	IV	Give IV dose over 1-2 min
	2-4 mg/kg	IM	Longer recovery, increase vomiting
Pure Sedatives			
Pentobarbital	4-6 mg/kg	IM	
	2-4 mg/kg	IV	
Etomidate	0.1-0.2 mg/kg	IV	Ultrashort, pain with injection
Propofol	0.5-1 mg/kg*	IV	Rapid onset and offset, pain with injection
Methohexital	1 mg/kg	IV	Ultrashort, limited studies
	20-30 mg/kg	PR	
Combination			
Ketofol	1-2 mg/kg (1:1 mixture)	IV	Titrate to effect, limited pediatric studies
Inhalational Agents			
Nitrous oxide	30%-70% NO_2	Inhalation	Cooperative child, scavenger system
Reversal Agents			
Naloxone	0.01 to 0.1 mg/kg/dose, max of 2 mg	IV, IM, IO	Can repeat every 5 min, 4 mg max (opioid reversal)
	Up to 4 mg/dose	ETT	
Flumazenil	0.01 mg/kg, up to 0.2 mg max single dose, max cumulative dose of 1 mg	IV	Titrate to max of 1 mg (benzodiazepine reversal)

ETT, Endotracheal tube; *IM*, intramuscular; *IN*, intranasal; *IO*, intraosseus; *IV*, intravenous; NO_2, nitrous oxide; *PO*, by mouth; *PR*, per rectum.

*Can be given as a continuous infusion: 25 to 150 µg/kg/min or in additional boluses of 0.5 mg/kg IV every 3 minutes as needed.

Table 67-2. Propofol for Procedural Sedation and Analgesia: Considerations

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 opioid for painful procedures
	Contraindicated with egg or soy allergy

Effective sedation can be provided with midazolam, administered intravenously, intranasally, 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 I consider for a 6-year-old child 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 opioid
- "Ketofol" (combination ketamine and propofol)
- Nitrous oxide with a hematoma block

Ketamine has been shown to have fewer adverse respiratory events when compared with fentanyl and midazolam.

18. What makes ketamine or "kidamine" useful as a PSA agent?

Ketamine, a dissociative agent causing a trancelike cataleptic state, is a 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. Ketamine can increase salivation; however, coadministration with an antisialagogue, such as atropine, is no longer recommended because it does not decrease adverse respiratory events. 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% of patients. Ondansetron has also been shown to reduce recovery emesis associated with ketamine. Ketamine, although protective of airway reflexes, is associated with such adverse events as airway or respiratory complications in 4%, transient apnea in 1%, and transient laryngospasm in 0.3%.

19. What are the contraindications for ketamine?

Absolute contraindications include age younger than 3 months or schizophrenia/psychosis. Relative contraindications include:

- Procedures stimulating the posterior pharynx (e.g., endoscopy; although there is no contraindication for typical ED oropharyngeal procedures)
- Airway instability (e.g., tracheal surgery or stenosis)
- Active pulmonary infection or disease (including upper respiratory infection [URI] and reactive airway disease [RAD])
- Cardiovascular disease
- Hypertension
- Porphyria
- Previous adverse reaction
- Central nervous system (CNS) masses or obstructive hydrocephalus (mild association with increasing intracranial pressure [ICP]; however, head trauma is not a contraindication)
- Glaucoma or increased intraocular pressure (conflicting evidence)

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, or 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.

Table 67-3. 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

OR, Odds ratio.
*Respiratory events include hypoxia, laryngospasm, and apnea.

KEY POINTS: HOW TO AVOID ADVERSE EVENTS WITH PEDIATRIC PSA

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, before 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. Before starting PSA, have advanced airway equipment ready, including suction, oxygen, and a properly sized bag-valve mask.
7. Use of local or regional anesthesia (e.g., lidocaine) after a first PSA dose (e.g., ketamine) can decrease the need for additional PSA doses.

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 dosages, it can cause 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?

With proper monitoring, most agents can be used and adverse events promptly treated; reversal agents are seldom needed. Certain drug types used are associated with different adverse event profiles (Table 67-3).

23. What reversal agents are available for children?

For opioids and benzodiazepines, specific reversing agents are available. Naloxone (0.01 to 0.1 mg/kg/dose max of 2 mg IV, IM, intraosseus [IO], or endotracheal up to 4 mg/dose) reverses opioid effects, and flumazenil (0.01 mg/kg IV up to 0.2 mg max single dose, max cumulative dose of 1 mg) reverses benzodiazepine effects.

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 or maintain head control if they are still in a child seat, and respond to commands given in a normal voice.

WEBSITE

Agency for Healthcare Research and Quality. National guideline clearinghouse: www.guideline.gov; accessed 2-12-15.

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QUESTIONS

1. Which of the following statements is false when considering pulse oximetry?
   a. False readings can occur in patients with vasoconstriction.
   b. It is not required for patient transfer after receiving a sedative medication.
   c. It is required for moderate sedation monitoring.
   d. Pulse oximetry may not detect respiratory depression before clinical signs of respiratory depression are present.

   The correct answer is *b*. Pulse oximetry is a required monitoring tool for moderate and deep sedation, and should be in place when transferring patients who have been sedated between or within a facility. Capnography has been shown to detect respiratory depression before either clinical examination or pulse oximetry, and is therefore more sensitive in picking up important changes in ventilation that may signal the potential for an airway or respiratory adverse event.
2. What PSA agent(s) can cause pain with IV injection?
   a. Propofol
   b. Ketamine
   c. Fentanyl
   d. Midazolam

   The correct answer is *a*. Propofol (and etomidate) can cause pain with IV administration. This pain can be minimized by using the antecubital vein or, if using a hand vein, by pretreating with an injection of lidocaine 0.5 mg/kg IV (with tourniquet or vein occlusion) for 1 minute.
3. When caring for a child who has just been given a reversal agent for oversedation with an opioid or benzodiazepine, it is essential to continue to monitor the child for:
   a. Vomiting
   b. Chest wall rigidity
   c. Returning to a sedated state
   d. Increased ICP

   The correct answer is *c*. A patient who has received a reversal agent for either an opioid or benzodiazepine while in a sedated state should be monitored for return of the sedation effects. Additional doses of reversal agents may be needed. Duration of action of naloxone (Narcan) is shorter than most opioid agonists.

PEDIATRIC AND NEONATAL RESUSCITATION

Kelly Stermer, RN, BSN, and Katherine M. Bakes, MD

CHAPTER 68

1. What is the pediatric assessment triangle?

The pediatric assessment triangle is a quick across-the-room assessment 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. What do I look for using the pediatric assessment triangle?

The most important component of the triangle is general appearance. Without touching the child, you can assess for tone, interactiveness, consolability, look/gaze, and speech/cry. A child's work of breathing is a more accurate assessment of oxygenation and ventilation than respiratory rate or auscultation of breath sounds; look and listen for signs of abnormal positioning, retractions, nasal flaring, and abnormal airway sounds. Assessing the child's skin color for pallor, mottling, or cyanosis can provide information on core perfusion and cardiac output. Essentially, this is a quick way to determine sick versus not sick.

3. How do I prepare myself for a child with cardiac arrest coming to the ED?

- Know your equipment. If you use it, review the Broselow pediatric emergency tape or its equivalent often.
- 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 cardiac arrest.

4. What are some other specific things I can do to prepare?

Have different sizes of masks, bags, and endotracheal tubes (ETTs) to treat the premature infant, as well as the adult-sized adolescent and every age in between.

Schedule mock pediatric resuscitations to identify gaps in equipment, medications, and processes. 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. Team resuscitations with clearly defined roles have been directly linked to better patient outcomes.

5. Is survival rate after cardiopulmonary arrest better in children or adults?

Investigators who have studied the two populations in parallel have shown a slightly better predicated survival for adults after out-of-hospital cardiopulmonary arrest. Because of inconsistencies in terminology, estimating exact survival rates after cardiac arrest in children has been difficult. Overall, only about 4% of children with out-of-hospital cardiopulmonary arrest survive to discharge; however, many do not survive neurologically intact. The odds are somewhat better for children than adults with in-hospital cardiopulmonary arrest, at a survival rate of about 27%. Survival to hospital discharge rates for pediatric arrest range from 2% to 12%, with neurologically intact survival at less than 2%. The higher rates are with the younger children.

6. What are the etiologies of pediatric arrests?

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. As in adults, the chance of survival is greater in pediatric patients with an initial rhythm of ventricular

Abstract

The approach to and management of neonates and children in need of cardiopulmonary resuscitation (CPR) is discussed in great detail.

Keywords:

neonatal resuscitation, pediatric resuscitation, newborn, pediatric airway, Neonatal Resuscitation Program (NRP), pediatric assessment triangle, cricoid, laryngeal mask airway (LMA)

fibrillation (VF) or pulseless ventricular tachycardia (VT) than in those with asystole or pulseless electric activity. Although outcomes remain poor, fewer children overall progress to full cardiopulmonary arrest.

7. What are predictors of outcome in pediatric arrest?

Predictors of mortality include:

- Age younger than 1 year
- Bradyasystolic rhythms
- Need for more than 2 doses of epinephrine administration and greater than 30 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 chance of 2% to 12%.

8. 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 because of 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 ventilation a greater risk for iatrogenic upper airway compression. Placing a rolled towel under the shoulders of an infant or under the occiput of a larger child can help maintain optimal positioning to provide a patent airway. The pediatric airway is shaped like an hourglass, with the cricoid cartilage composing the narrowest portion of the airway. An infant's tracheal diameter is approximately the diameter of a little finger; therefore even small amounts of secretions, blood, edema, or small foreign objects can put them at risk for airway obstruction.

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-like in shape than the adult epiglottis. Because of 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.

9. Does the pediatric patient require cricoid pressure during active ventilation?

Yes, it is a common misconception that because the cricoid is the smallest part of the airway, cricoid pressure is not necessary during bag-valve-mask ventilation. Just like with adults, the reasons for cricoid pressure is to compress the esophagus in patients with a presumed full stomach. In addition, because of a child's anatomically smaller thorax, preventing gastric insufflation is essential to prevent the pediatric stomach from distending and impeding ventilation. 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. Gastric distension can impede the diaphragm and may cause serious issues with ventilating a child. For this reason, an oral gastric or nasogastric tube should be placed early in the resuscitation.

10. What about cricoid pressure during intubation?

Cricoid pressure during intubation is not necessary and can interfere with airway management. If needed to visualize the vocal cords, the intubating provider can use his or her own hand to manipulate either the cricoid or the thyroid cartilage to provide better visualization. At that point, another provider can take over the placement of pressure to facilitate intubation.

11. How does the approach to the B (breathing) of ABCs (airway, breathing, and circulation) 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 he or she is to change cardiac contractility. Thus children 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.

12. What size of ETT should I use?

In children between the ages of 1 and 8 years, the simple formula (age/4) + 4 can be used for uncuffed tube sizes, and (age/4) + 3.5 can be used for cuffed tubes. More and more evidence suggests that past the neonatal period, cuffed tubes are preferred to prevent air leaks that can limit effective ventilation and require risky tube changes (particularly in children requiring high ventilator pressures). Cuff pressures must be checked with a manometer and should not exceed pressures greater than 20 cm H_2O, as even 30 minutes of higher pressures can lead to permanent airway injury.

13. What are alternative airway devices that can be used in the pediatric population?

The laryngeal mask airway (LMA) is considered Class IIa, or "weight of evidence/opinion is in favor of usefulness/efficacy," in the latest 2010 Pediatric Advanced Life Support (PALS) guidelines as based on consensus expert opinion. The LMA has gained more recognition as an alternative device to effectively ventilate infants and children. It is viable alternative for ventilation when either ETT or bag-mask ventilation are not options, or if it is only needed for short-term use. It can be especially helpful in maintaining a patent airway on the child with cranial malformations.

14. What about cricothyrotomies in children?

Because of the slitlike cricothyroid membrane and the easily transectable airway, an open surgical cricothyrotomy is not recommended in children younger than 8 years. Needle cricothyrotomy is a rarely used 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.

15. 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). Although PALS lists the higher concentration of 1:1000 epinephrine via the ET route as an option, there is no evidence of its benefit in pediatric resuscitation. If intravenous (IV) access has not been established within 90 seconds, then the provider should establish central venous or intraosseous (IO) access. If there are enough providers to establish alternative access and prepare ET dosing, then these can occur simultaneously but should not delay establishment of IV/IO access.

16. At what point should chest compressions be initiated in children?

Chest compression should be initiated for pulseless arrest or when the heart rate is less than 60 beats per minute with evidence of poor end-organ perfusion. Chest compressions should be performed at 100 per minute at a depth one half to one third of the anteroposterior diameter of the chest. Like advanced cardiac life support, PALS emphasizes continuous, hard, and fast chest compressions with minimal interruptions.

17. 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 in is 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.

18. 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.

19. What should I expect in a normal newborn at the time of birth?

When the infant is born, there are three questions that should be asked immediately:

1. Term gestation?
2. Breathing or crying?
3. Good tone?

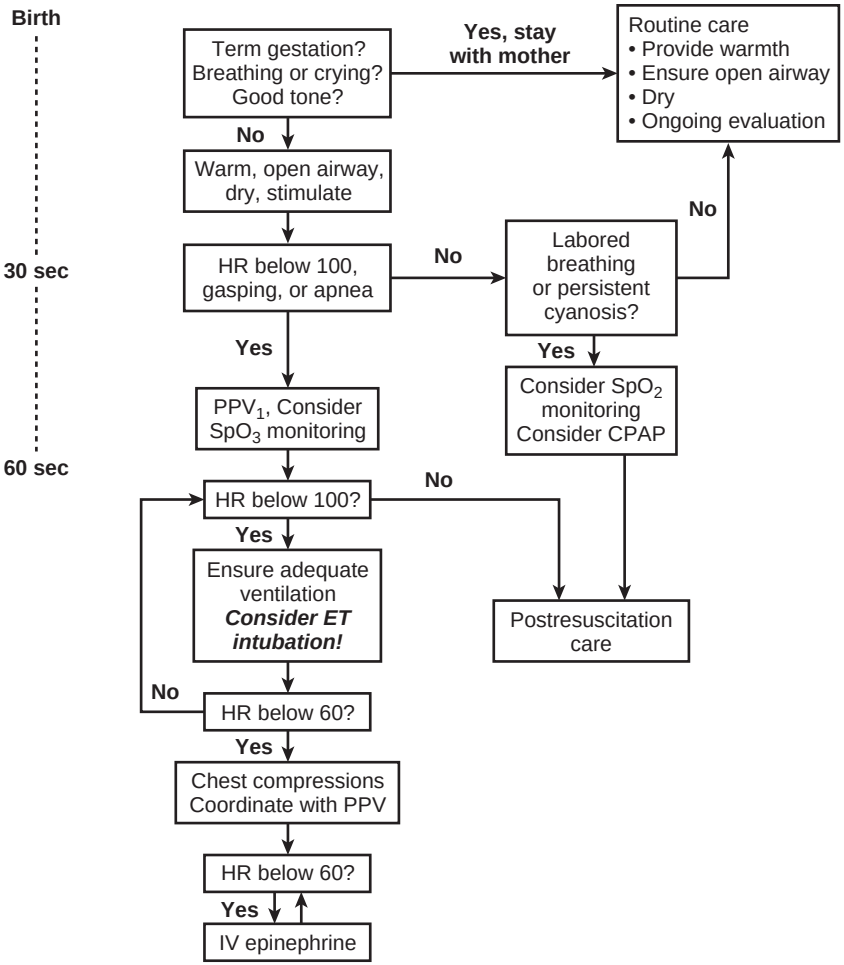

Figure 68-1. Newborn resuscitation algorithm. *CPAP,* continuous positive airway pressure; *ET,* endotracheal; *HR,* heart rate; *IV,* intravenous; *PPV,* positive pressure ventilation; *SpO_2,* blood oxygen saturation. *(From Perlman JM, Wyllie J, Kattwinkel J, et al: Neonatal resuscitation: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations.* Pediatrics *126;e1319, 2010.)*

If the answer to these is *yes,* routine care (warm, dry, and suction only if needed) are recommended. The baby should stay with the mother during ongoing assessments.

20. After delivery in the ED, what is the first priority in the care of the newborn?

See Figure 68-1. The first priority is to prevent body heat loss. The infant must be dried and warmed immediately to prevent evaporative heat loss. Remove wet linens and place the newborn under a radiant warming unit to lessen loss of radiant heat. If no warmer device is available and the infant is at term, crying, and has good tone, place infant skin-to-skin with the mother and cover them with a warm blanket.

Infants can lose a substantial amount of heat from their head, so remember to put on a stocking cap early in resuscitation, especially on premature infants. These initial steps take only

Table 68-1. Targeted Preductal SpO_2 after Birth

1 minute	60%-65%
2 minutes	65%-70%
3 minutes	70%-75%
4 minutes	75%-80%
5 minutes	80%-85%
10 minutes	85%-95%

SpO_2, Blood oxygen saturation.

seconds to accomplish and may prevent serious metabolic derangements. These steps should be taken even before the initiation of CPR. Cold babies respond poorly to even the best resuscitation efforts.

21. When should central cyanosis resolve in a healthy newborn after delivery?

It is not unusual for newborns to appear slightly cyanotic for the first few minutes 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. However, studies have shown clinical assessment of skin color is not very reliable. Therefore any persistent central cyanosis or cyanosis of the oral mucous membranes should be confirmed with attachment of the pulse oximetry.

22. So what is a normal newborn pulse oximetry level?

Studies indicate that healthy newborns undergoing normal transition may take several minutes to increase their blood oxygen saturation (SpO_2) from approximately 60%, which is the normal intrauterine state to more than 90%, which is the desired state of healthy newborns. This transition can take up to 10 minutes. Preductal saturations (oximetry measured on the right wrist or hand) should be monitored and guide the amount of oxygen provided. Term babies should be resuscitated initially with room air (Table 68-1).

23. 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 (preferably with a meconium aspirator) before bulb suctioning in newborns who are nonvigorous, described as weak respiratory efforts, poor muscle tone, and a heart rate less than 100 beats per minute.

24. After the infant is dried, suctioned, and placed under the warmer, how do I decide whether further active intervention is needed?

Only approximately 10% of newborns require additional intervention, and only about 1% will require advanced life support efforts. If the infant is active, crying, and has a heart rate greater than 100 beats per minute, further intervention is seldom needed. If the infant demonstrates apnea, bradycardia, or central cyanosis, then use of bag-mask ventilation is needed, because ongoing efforts to stimulate an apneic baby beyond the initial 30 seconds after birth will waste valuable time. Most of the time, if the infant is near term gestation, the heart rate will respond quickly to positive pressure ventilation with a few effective assisted breaths.

25. For the newborn, define bradycardia and indications for intervention.

A heart rate of less than 100 beats per minute at 30 seconds after 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 less than 100 beats per minute after 30 seconds of drying and stimulating. If the heart rate has not improved after providing 30 seconds of effective respiratory support, consider whether your ventilation technique is correct, and remember the mnemonic *MR SOPA*.

Table 68-2. Central Venous Catheter Size Based on Patient Age, Length/Height, and Weight

AGE	WEIGHT (KG)	LENGTH/HEIGHT (CM)	FRENCH SIZE
Premature	≤2.5	<50	2-2.5
Birth-30 days	3-4	50-55	3
>1 mo-1 yr	4.5-10	5-75	3-4
>1-7 yr	11-25	7-120	4-5
>7-15 yr	26-60	125-175	5-8

From Nadel FM: Vascular access. In Baren JM, Rothrock SG, Brennan J, et al, editors: *Pediatric emergency medicine*, Philadelphia, 2008, Saunders.

Mask adjustment
Reposition, open airway, jaw thrust
Suction mouth
Open mouth
Pressure increase (up to 40 cm H_2O pressure)
Airway alternative

If the heart rate remains at less than 100 beats per minute, positive pressure ventilations should be continued.

26. At what point do I need to initiate chest compressions?

If the heart rate drops below 60 beats per minute despite effective ventilations the provider should initiate chest compressions at a rate of 100 per minute using a two-finger chest encircling technique. Place thumbs or fingers on the sternum, above the xiphoid process, and in line with the nipples. Avoid pressure over the liver, because a liver laceration can occur.

27. How do I know when to stop chest compressions?

Heart rate should be evaluated about every 45 to 60 seconds. Compressions should be performed in between breaths. When a sustained heart rate of greater than 60 beats per minute is achieved, compressions can be discontinued.

28. How many infants will require intubation to provide adequate ventilation?

Almost all newborn infants can receive bag-mask ventilation. Bag-mask ventilation should be done only with equipment designed for newborn and premature infants. Ventilation of the newborn can be performed effectively with a flow-inflating bag, a self-inflating bag, or a pressure limited T-piece resuscitator. Ventilation is the most important intervention that can be done for a newborn. Current guidelines suggest starting with air and blending it with oxygen as needed to maintain target preductal saturations. An increasing heart rate and oxygen saturation are the best indicators of effective ventilation. To avoid barotrauma, inflation pressures should start around 15 to 20 cm H_2O and not exceed 40 cm H_2O in the term infant. For the preterm newborn, pressures should begin around 20 to 25 cm H_2O. Ventilation of the premature infant can be difficult because of immature lungs, and may require higher pressures. All neonatal bags should be equipped with pressure manometers.

29. When should I attempt vascular access, and 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 infants 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 68-2). Peripheral IV/IO is also an option.

30. What drugs should be available for use in newborn resuscitation, and when should they be given?

Fewer than 1% of babies require medications during resuscitation, especially if bag-mask ventilation is started early. Usually, no more than two agents are needed.

Table 68-3. 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.

- 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 the heart rate remains less than 60 beats per minute after 30 seconds of effective bag-mask ventilation and chest compressions. The standard dosage is 0.1 to 0.3 mL/kg (0.01 to 0.03 mg/kg), either via the umbilical vein or peripheral IV/IO. If the ETT route is readily available, Neonatal Resuscitation Program (NRP) guidelines recommend considering a higher dosage (0.5 to 1 mL/kg) of epinephrine (also the 1:10,000 dilution) via the ETT while an IV route is being established. To avoid medication administration errors, it is best to use two different sizes of syringe: a larger, 3-mL syringe for ETT use, and a smaller, 1-mL syringe for the umbilical venous or peripheral line. Because absorption via the ETT is unpredictable and slower, the IV route is still preferred. The drug can be given as frequently as every 3 to 5 minutes if bradycardia persists.
- Dextrose 2 mL/kg of 10% dextrose solution can be given to newborns suspected of having hypoglycemia based on clinical findings and blood glucose levels below 40 mg/dL, although normal nadirs can be as low as 30 mg/dL. Risk factors for newborn hypoglycemia include having a diabetic mother, being either small or large for gestational age, having respiratory distress, and being premature. Clinical signs include respiratory distress, apnea, lethargy, hypotonia, seizures, jitteriness, myoclonus, temperature instability, and weak or high-pitched cry.
- 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 dosage of 10 mL/kg.
- Naloxone is not recommended for the resuscitation of newborns, even if they are born to opioid-dependent mothers. Sudden opioid withdrawal can be deleterious for the newborn, causing seizures and unnecessary distress. Focus should be on support of the airway and breathing.

31. 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 68-3). The score is calculated at 1 minute and at 5 minutes. However, resuscitation is not guided by Apgar scores and should be initiated before the 1-minute Apgar score. If the 5-minute score is less than 7, Apgar scores should continue at 5-minute intervals 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. Because of underlying causes, pediatric patients in cardiac arrest have poor prognoses.
3. The highest impact intervention for a newborn or pediatric resuscitation is appropriate respiratory support.

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QUESTIONS

1. What is the best indicator that a neonate is receiving adequate ventilation?
   a. The baby's color improves.
   b. The heart rate increases above 60 beats per minute.
   c. Blood pressure and oxygen level improve.
   d. Heart rate and oxygen level increase.
   The correct answer is *d*.
2. What percentage of oxygen is recommended to start a resuscitation of a full-term baby?
   a. 100%
   b. Room air
   c. 50%
   d. Blow-by
   The correct answer is *b*.
3. Which of the following is not a predictor of a poor outcome in pediatric cardiac arrests?
   a. Primary respiratory arrest
   b. Bradyasystolic rhythm
   c. More than two doses of epinephrine given
   d. More than 30 minutes of active CPR
   The correct answer is *a*.

GENERAL APPROACH TO POISONINGS

Katherine M. Hurlbut, MD

CHAPTER 69

1. List the 15 most common causes of death from acute poisoning reported to poison centers.

- Sedative/hypnotics/antipsychotics, 14.1%
- Cardiovascular drugs, 12.2%
- Opioids, 8.9%
- Acetaminophen in combination, 6.4%
- Stimulants and street drugs, 6.1%
- Acetaminophen alone, 5.5%
- Alcohols, 5.1%
- Antidepressants, 4.4%
- Selective serotonin reuptake inhibitors, 3.1%
- Antihistamines, 2.4%
- Tricyclic antidepressants, 2.4%
- Fumes/gases/vapors, 2.3%
- Aspirin alone, 2.3%
- Muscle relaxants, 2%
- Anticonvulsants, 2%

Note: Despite a high incidence of involvement, these substances are not the most toxic, but rather may be the most readily accessible.

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 role of activated charcoal?

Activated charcoal is the most widely used method of gastrointestinal (GI) decontamination. It has been shown in studies to decrease drug absorption but has not been shown to affect clinical outcome after overdose. It is most effective if administered within an hour of ingestion but may be useful beyond 1 hour after ingestion of modified release formulations, very large ingestions in which absorption may be prolonged, and after ingestion of highly toxic substances. 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. Adverse effects include aspiration (particularly patients with depressed mental status and those with vomiting) and concretion formation. The decision to administer activated charcoal should take into consideration the potential benefit (amount, time, and toxicity of substances ingested) and risks; it should not be routinely administered to all patients. Patients with trivial ingestions (generally children) do not require activated charcoal therapy.

3. What is the role of gastric lavage in treating acute poisonings?

Gastric lavage has a very limited role, primarily in the treatment of patients with large, recent ingestions of substances with the potential to cause life-threatening toxicity, for which reliable treatment options are limited. Gastric lavage has not been shown to alter clinical outcome in large series of patients with overdose. Although serious sequelae of gastric lavage are rare, it carries the

Abstract

Intentional or inadvertent overdose or poisoning is a common ED presentation. Although specific antidotes are available for a limited number of toxins, the vast majority of patients recover with attentive supportive care. The recognition of specific patterns of clinical and laboratory findings associated with classes of toxins (toxidromes) can be useful in guiding patient evaluation and treatment.

Keywords:

overdose, poisoning, drug intoxication, gastrointestinal (GI) decontamination, toxidrome, toxicology screen, antidote

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 come to the ED within 1 to 2 hours after ingestion. It was used in 0.15% of poison center cases in 2012.

4. 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 substances with limited toxicity, 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 recent deliberate drug overdose involving a substance with the potential to cause moderate to severe toxicity is done easily (although it is often messy) and may reduce toxicity. If a reliable history indicates ingestion of substances with minimal toxicity, or the time since ingestion is long, activated charcoal is not necessary.

5. Is there a role for cathartics in treating acute poisoning?

The theory behind cathartics is that they speed up GI transit time, potentially decreasing drug absorption and possibly preventing 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.

6. What is the 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.

7. 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 GI 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, phenytoin, carbamazepine, and quinine.

8. 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 dextrose 5% in water (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. 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.

9. 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 metabolic acidosis and 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 minimally available, it commonly causes hypocalcemia and thrombocytopenia, and it can result in canister clotting. Drugs for which charcoal hemoperfusion is often employed include theophylline, phenobarbital, and a few other less common agents, such as paraquat and amatoxin.

10. How can the diagnosis of a drug overdose be made when the patient is unconscious and the 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 whether 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 opioids, cocaine, and amphetamine. The physical examination is useful in narrowing the diagnosis to a class of drug or chemicals. Reactions to specific classes of drugs are commonly called *toxic syndromes* (Table 69-1) or *toxidromes*.

11. How can a toxicology screen and other ancillary laboratory tests make the diagnosis of acute poisoning?

The urine toxicology screen has a limited role in the evaluation of overdose patients. Toxicology screens are expensive, often are inexact, and commonly 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 excluded from the screen. Multiple studies have demonstrated that urine toxicology screening rarely impacts clinical decision making; routine use is not warranted. Screening may be useful in evaluating patients with persistent altered mental status or significant vital sign abnormalities.

Urine immunoassays are commonly used and relatively easy to perform; however, they screen for limited classes of drugs (primarily drugs of abuse), and both false-positive and false-negative

Table 69-1. 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)*, some Alzheimer medications
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.

results are common. In addition, these tests are qualitative and often screen for metabolites rather than the parent drug, thus the presence of a substance does not necessarily indicate intoxication. Many of the newer drugs of abuse are not detected on the commonly used urine drugs of abuse assays.

Alternatives to a full toxicology screen include testing discrete serum concentrations of suspected toxins based on the patient's clinical presentation and/or the substances known to be available to the patient. Serum concentrations are generally useful clinically only for those substances for which therapeutic and toxic concentrations have been established and for which serum or blood concentrations can be rapidly obtained from clinical laboratories.

Comprehensive urine toxicology screens can detect a wider variety of toxins; however, they are time consuming to perform, and the accuracy of results is very dependent on the skill and experience of the technician performing the assay. Many more drugs and chemicals are not found on typical toxicology screens than are found on the screens, although many 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 whether other tests are likely to be of benefit.

12. What other studies are useful in the evaluation of a poisoned patient?

- An acetaminophen concentration level should be obtained in patients with deliberate overdose, because this substance is widely available, commonly involved in overdoses, and causes little in the way of initial symptoms. Treatment with *N*-acetylcysteine is most effective if begun within 8 hours of ingestion.
- Nontoxicologic laboratory tests that are often 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, hydrocarbons, or opioids; and, rarely, a kidney, ureter, and 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 enzyme levels are not necessary to determine after most overdoses but may help 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 and/or hematuria, 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. Keep in mind that acid-base changes generally develop only after a toxin has been absorbed and metabolized. A specimen obtained shortly after ingestion may show a normal acid-base status despite ingestion of significant amounts of a toxin that can cause metabolic acidosis.
- In the workup of persistent acidosis, a serum osmolality test 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 significant, although a normal osmolal gap does not rule out toxic alcohol ingestion.
- A pregnancy test should be considered, because unintended pregnancy may be the precipitant for an overdose. Pregnant patients may need counseling regarding the potential effects of the drugs ingested on the fetus.

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.

13. Discuss some other useful antidotes for common poisonings.

- Naloxone and dextrose are the most common antidotes. Any patient with altered mental status in whom a bedside blood glucose measurement cannot be rapidly performed should receive intravenous dextrose. Administration of naloxone that results in awakening of the patient is diagnostic of acute opioid overdose. Small, incremental doses of 0.2 mg should be used if it is suspected that the patient may be opioid dependent, because large doses of naloxone will precipitate withdrawal. Naloxone can also be administered intramuscularly, intranasally, or via nebulization if intravenous access is difficult to obtain. Continuous infusion of naloxone is likely to be necessary if a long-acting or modified-release opioid has been ingested.
- 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, although their occurrence is extremely rare. 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, digoxin immune Fab 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 proconvulsants have been coingested with benzodiazepine or if the patient has a history of seizures. The usual adult dose is 0.2 mg over 30 seconds; if there is an inadequate response after 30 seconds, administer 0.3 mg IV over 30 seconds. Additional doses of 0.5 mg IV over 30 seconds may be given at 1-minute intervals if needed, up to a maximum 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 and 1.5 mL/kg/h in a chronic alcoholic. Blood ethanol concentration should be measured immediately after the loading dose and repeated every hour initially, and the dosage should be 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_5W over 4 hours, followed by 100 mg/kg in 1 L D_5W infused over 16 hours).

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QUESTIONS

1. Which of the following statements are true about activated charcoal therapy after overdose?
   a. It has been shown to improve clinical outcome.
   b. It has been shown to reduce absorption of toxins.
   c. It should be administered to virtually all patients with deliberate overdose.
   d. Its administration is commonly associated with significant adverse effects.
   The correct answer is *b*.
2. Which of the following statements is true regarding urine toxicology screens?
   a. They have been shown to impact clinical decision making in the care of overdose patients.
   b. A positive urine toxicology screen for a given substance correlates well with clinical intoxication.
   c. They are not indicated in most patients with suspected overdose.
   d. Urine toxicology screens have good sensitivity for most drugs of abuse but limited specificity.
   The correct answer is *c*.
3. Which of the following toxin/antidote pairs is not correct?
   a. Opioid/naloxone
   b. Tricyclic antidepressant/flumazenil
   c. Anticholinergic/physostigmine
   d. Acetaminophen/*N*-acetylcysteine
   The correct answer is *b*.

THE ALCOHOLS: ETHYLENE GLYCOL, METHANOL, ISOPROPYL ALCOHOL, AND ALCOHOL-RELATED COMPLICATIONS

CHAPTER 70

Louis J. Ling, MD

ETHYLENE GLYCOL AND METHANOL

1. **Why is it important to understand the metabolism of ethylene glycol?**
   It is not ethylene glycol, but its metabolites, that is toxic. Their formation depends on alcohol dehydrogenase (ADH) for their conversion from the nontoxic parent. Ethanol saturates and fomepizole (4-methyl-1H-pyrazole [4-MP]) blocks ADH, and they impede the metabolism of ethylene glycol and methanol to toxic metabolites. Pyridoxine (vitamin B_6) and thiamine are cofactors in the final steps to form nonharmful end products and can be given to ensure maximal metabolism. Oxalate crystals may not appear until late in the course of the poisoning (Fig. 70-1).

2. **What is the toxicity of ethylene glycol?**
   Initially, there is central nervous system (CNS) intoxication and gastrointestinal irritation, followed by metabolic acidosis. Renal failure occurs often and typically is delayed in presentation. Cranial nerve deficits are a rare complication.

3. **Why is ethylene glycol so dangerous to animals?**
   Ethylene glycol is a common 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.

4. **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. (Note that this is a very nonspecific test as many foods can cause the urine to fluoresce.) A positive test should encourage further investigation, but a negative test misses two thirds of ingestions.

5. **Why is it important to understand the metabolism of methanol?**
   As with ethylene glycol, ethanol and 4-MP saturate and block ADH, respectively, inhibiting conversion of methanol into its harmful metabolites. 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.

6. **List the signs and symptoms of methanol poisoning.**
   Gastrointestinal toxicity
   - Nausea and vomiting
   - Abdominal pain

Abstract

This chapter contains a concise review of ethylene glycol, methanol, isopropanol, and alcohol-related disorders.

Keywords:

ethylene glycol, methanol, ethanol, isopropyl alcohol, anion gap, metabolic acidosis, thiamine, hypoglycemia, alcohol withdrawal, Wernicke-Korsakoff syndrome, fomepizole (4-methyl-1H-pyrazole [4-MP]), pyridoxine, folate

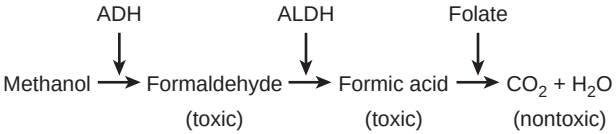

Figure 70-1. Metabolism of methanol. *ADH,* Alcohol dehydrogenase; *ALDH,* aldehyde dehydrogenase; *CO_2,* carbon dioxide; *H_2O,* water.

CNS toxicity
- Headache
- Decreased level of consciousness
- Confusion

Ocular toxicity
- Retinal edema
- Hyperemia of the disc
- Decreased visual acuity

Metabolic acidosis

7. 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.

8. 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.

9. 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:

$$\text{Anion gap} = (Na^+) - (HCO_3^- + Cl^-)$$

10. 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.*

Alcohol
Methanol
Uremia
Diabetic ketoacidosis
Paraldehyde
Iron, **I**soniazid (INH)
Lactate
Ethylene glycol
Salicylate

11. 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.

12. How is an osmolal gap calculated?

One formula is:

$$[2 \times Na^{+}\ (mEq/L)] + [glucose\ (mg/dL)/18] + [BUN\ (mg/dL)/2.8] + [ethanol\ (mg/dL)/4.6],$$

where BUN stands for *blood, urea, nitrogen.* 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 is:

$$2 \times Na\ (mEq/L) + glucose\ (mmol/L) + BUN\ (mmol/L) + 1.25 \times ethanol\ (mmol/L)$$

A toxic ethylene glycol level of 25 mg/dL can be predicted to increase the osmolal gap by 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.

13. 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.

14. How much methanol or ethylene glycol is dangerous?

Death has been reported after 15 to 30 mL (1 to 2 tablespoons) of methanol, but others have survived larger ingestions. A minimal lethal dose for ethylene glycol is approximately 1 to 2 mL/kg. Any intentional ingestion should be taken seriously.

15. 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 or hemodialysis. Ethanol and 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. Pyridoxine (50 mg every 6 hours) and thiamine (100 mg every 6 hours) should be given for ethylene glycol, and folate (50 mg every 4 hours) should be given for methanol, because they are cofactors for the final step to the nontoxic end products.

16. How do I choose between 4-MP and ethanol?

Ethanol is difficult to give consistently; ethanol blood levels are required to adjust the dosage, and infusion can cause pain, resulting in the use of a central catheter. Ethanol may cause hypoglycemia (theoretically) and respiratory depression, especially in young children. These patients usually require the close monitoring of an intensive care unit (ICU). 4-MP is rapidly 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. If ethanol is your only option, ADH has a much higher affinity for ethanol than for ethylene glycol, and one option is to maintain a patient's serum ethanol concentration to 100 mg/dL. This can be accomplished via oral ethanol or intravenously via a 5% to 10% solution administered through a central line.

17. How do I use 4-MP?

The intravenous (IV) dosage is 15 mg/kg loading dose, followed in 12 hours by 10 mg/kg dose every 12 hours for a total of four doses initially. If therapy is needed beyond 48 hours, the dosage is then increased to 15 mg/kg every 12 hours for as long as necessary. If hemodialysis is used as an additional therapy, then it is recommended that 4-MP be given every 4 hours.

18. What are the indications for hemodialysis?

Hemodialysis is the most definitive therapy for ethylene glycol poisoning, because it clears both ethylene glycol and methanol and their toxic metabolites from the blood, and corrects any resulting

metabolic acidosis. Indications for hemodialysis include metabolic acidosis (serum pH < 7.2), signs of end-organ toxicity (e.g., seizures and coma), and renal failure.

19. 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 between 30 and 52 hours, which requires a much longer course.

KEY POINTS: ETHYLENE GLYCOL AND METHANOL

1. Symptoms and acidosis are often delayed for both.
2. Urinary oxalate crystals, early osmolal gap, and metabolic acidosis all suggest ethylene glycol poisoning.
3. Maintain a high level of suspicion for intentional overdoses.
4. 4-MP, ethanol, and dialysis can all be used to treat both.

ISOPROPYL (ISOPROPANOL) ALCOHOL

20. 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. Isopropyl alcohol causes an osmolar gap, but not a metabolic acidosis, because it is a secondary alcohol and not metabolized to an acid.

21. What are the symptoms of isopropanol 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 CNS 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.

22. Why is isopropanol so commonly 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.

23. What is the treatment 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. A test for the isopropyl level usually does not add greatly to clinical observation. In the rare instance of coma or respiratory depression 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.

ALCOHOL-RELATED COMPLICATIONS

24. What complications arise from ethanol?

Alcohol greatly increases the chances that a person will become an ED patient. Whereas ethanol increases the risk for many diseases, its use has even more direct effects. The most lethal effect is respiratory depression and asphyxia, but trauma as a result of incoordination or poor judgment is also dangerous and sometimes deadly.

25. When should an acutely intoxicated patient have their airways intubated?

Hypopnea and hypoventilation are sometimes the issue, but the inability of the patient to protect the airway is much more common. 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.

26. 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. Although the IV treatment is faster and more easily titrated, oral medication is less expensive. Haloperidol is an appropriate adjunct for agitated behavior or hallucinosis. Theoretic 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 the patient is under observation by medical personnel.

27. What is an appropriate workup for repeat alcohol withdrawal seizures (AWDS)?

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 workup, including computed tomography (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.

28. How should AWDS be managed?

- Acute: As with all seizures, ensure a patent airway and administer 50% dextrose in water ($D_{50}W$) and benzodiazepines intravenously (as needed). An observation 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.

29. Can AWDS be prevented?

Benzodiazepines in the acute withdrawal period, particularly in patients with a history of AWDS, can decrease seizures.

30. Who is at risk for alcohol-induced hypoglycemia (AIH)?

Because AIH, alcohol-induced impairment of gluconeogenesis, results from insufficient glycogen stores, the three groups vulnerable to AIH are people with chronic alcoholism, binge drinkers, and young children.

31. What is the clinical presentation?

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 common presentation in children. Localized CNS signs, including a strokelike picture (alcohol-induced hypoglycemic hemiplegia), often occur in adults.

32. How does alcoholic ketoacidosis (AKA) develop?

This commonly occurs early after heavy binge drinking with 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. 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, but these values vary widely because of the commonly 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 blood gases 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, an obvious difference from diabetic ketoacidosis.

33. How should I manage AKA?

Treatment consists of rehydration with dextrose-containing crystalloid, antiemetics, benzodiazepines for withdrawal, and potassium and phosphate as indicated. Bicarbonate is rarely required, and metabolic abnormalities usually resolve within 12 to 16 hours.

34. What is the relationship between alcohols and metabolic acidosis?

- Ethanol: Acute ethanol ingestion results in a mild increase in the lactate-to-pyruvate ratio but does not produce a clinically significant metabolic acidosis.
- 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.
- 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.

35. How is coagulation affected in a patient with chronic alcoholism?

Bone marrow depression from ethanol, folate deficiency, and hypersplenism secondary to portal hypertension all cause thrombocytopenia, but platelet counts less than 30,000/mL are unlikely. Qualitative platelet defects also occur. Liver disease from chronic alcohol abuse depletes all coagulation factors except VIII, particularly II, VII, IX, and X.

36. When is vitamin K useful?

Patients with alcoholism often have inadequate vitamin K, a requisite cofactor for the production of factors II, VII, IX, and X. When faced with gastrointestinal hemorrhage in chronic alcoholism, IV vitamin K supplementation is worthwhile. 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.

37. Must thiamine be administered before glucose in the patient with alcoholism?

Wernicke-Korsakoff syndrome develops over hours to days. Although the initiation of Wernicke-Korsakoff syndrome by dextrose has never been substantiated, the consequences of neuroglycopenia begin within 30 minutes and are easily prevented. In alcoholic patients with known or suspected hypoglycemia, prompt glucose is given with thiamine as soon afterward as possible. Because magnesium is a cofactor of thiamine and because alcoholic patients are commonly hypomagnesemic, 2 g of IV magnesium should also be given with suspicion of Wernicke-Korsakoff syndrome.

38. Is it dangerous to administer thiamine intravenously?

Orally administered thiamine is often absorbed poorly in the alcoholic patient. The intramuscular (IM) route is painful and can result in hematomas or abscesses, particularly in patients with impaired coagulation. The experience with IV thiamine is extensive. Thiamine may be given as part of fluid hydration or by bolus infusion.

39. 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 antiinflammatories, and hydration. The only sure-fire measure is the avoidance of over-exuberant drinking in the first place.

KEY POINTS: ALCOHOL-RELATED DISORDERS

1. Whether an acutely intoxicated patient requires intubation for airway protection is based on the clinicians physical assessment.
2. Phenytoin should only be given to patients with clear indication of an epileptogenic focus; its use for prevention of AWDS is not indicated.
3. Large doses of benzodiazepines may be needed for treating withdrawal.
4. Time and IV hydration improves AKA.

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QUESTIONS

1. How can you differentiate isopropanol ingestion from methanol?
   a. Osmolar gap without an acidosis
   b. Acidosis without an osmolar gap
   c. Acidosis and osmolar gap
   d. None of the above
   The correct answer is *a*.
2. Which of the following can cause an osmolar gap?
   a. Methanol
   b. Ethylene glycol
   c. Ethanol
   d. All of the above
   The correct answer is *d*.
3. AWDS should be treated with:
   a. Phenytoin (Dilantin)
   b. Levetiracetam (Keppra)
   c. Benzodiazepines
   d. None of the above
   The correct answer is *c*.

CHAPTER 71

ANTIPYRETIC POISONING

James C. Mitchiner, MD, MPH

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 262 mg/tablespoon of salicylate, is occasionally the culprit. In adults, concurrent ingestion of aspirin and aspirin-containing prescription or nonprescription 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 = 9 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 is experiencing an acute salicylate overdose?

Early diagnosis is essential. Patients may have nausea, vomiting, tinnitus, vertigo, fever, diaphoresis, and confusion. Hyperventilation may be ascribed mistakenly to anxiety. Patients also may have headache or chronic pain, which prompted the excess ingestion of salicylate.

3. List some common clinical features of acute salicylate intoxication.

See Table 71-1.

4. Describe the acid-base disturbances associated with salicylate toxicity.

Acute respiratory alkalosis, without hypoxia, is caused by 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 as a result of accumulation of lactic acid and ketoacids. A mixed respiratory alkalosis and metabolic acidosis typically is seen in adults. In patients with respiratory acidosis, concomitant ingestion of a central nervous system (CNS) 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 hyperpyretic state. Insensible losses are increased in patients with hyperventilation. Hypokalemia is caused by 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.

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 at least 2 hours apart, while the patient is still under observation in the ED, so that the severity of poisoning can be

Abstract

This chapter contains a concise review of antipyretic poisoning.

Keywords:

acetaminophen, salicylates, ibuprofen, N*-acetylcysteine (NAC)*

Table 71-1. Common Features of Acute Salicylate Toxicity

General	Hyperthermia, dehydration
Respiratory	Hyperventilation (may be mistaken for anxiety), noncardiogenic pulmonary edema
Central nervous system	Tinnitus, confusion, delirium, seizures, coma
Gastrointestinal	Nausea, vomiting, diarrhea, gastrointestinal hemorrhage
Dermatologic	Eyelid petechiae
Laboratory	Acid-base disturbances, azotemia, hyperkalemia or hypokalemia, hypoglycemia (children), elevated CK levels (rhabdomyolysis), coagulopathy

CK, Creatine kinase.

trended. The aspirin (Done) nomogram is of historic interest only and is no longer recommended. Most patients show signs of intoxication at salicylate levels of 40 mg/dL or higher.

9. Which laboratory tests are indicated?

Serial serum salicylate levels should be obtained initially and every 2 hours in the ED, until a decreasing trend in the level is established, the most recent level is below 20 mg/dL, and the patient is asymptomatic with a normal respiratory rate. Additional tests include a complete blood cell count; serum electrolyte, blood urea nitrogen (BUN), creatinine, and glucose levels; and a urinalysis. Prothrombin time (PT), international normalized ratio (INR), and arterial blood gases should be considered. A quantitative acetaminophen level is also recommended to exclude acetaminophen cotoxicity.

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 (IV) normal saline should be given initially, with conversion to alkaline diuresis if the patient is toxic. A slurry of activated charcoal mixed with a cathartic (sorbitol or magnesium sulfate) should be given orally if tolerated or by gastric lavage tube, if there is intubation, at a dosage of 1 g of charcoal per kilogram. Lavage may be useful even if the patient comes to the ED 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 dosage of 20 to 40 mEq/L. Patients with hyperthermia should be cooled with a cooling blanket. Hypoglycemia should be treated with IV dextrose 50% in water ($D_{50}W$). Patients with aspirin-induced noncardiogenic pulmonary edema should be treated with oxygen, noninvasive ventilation (continuous positive airway pressure [CPAP] or bilevel 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 CNS 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 a 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 ions, limiting the amount of salicylate that crosses the blood-brain barrier. Similarly, an alkalotic urine retains salicylate ions, preventing their reabsorption by the renal tubules. Isotonic alkaline diuresis is achieved by adding 3 ampules of sodium bicarbonate ($NaHCO_3$) to 1 L of dextrose 5% in water (D_5W), with infusion at a rate of 2 to 3 mL/kg/h. 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 unionized and more penetrates the blood-brain barrier, resulting in CNS 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), CNS deterioration (e.g., seizures, coma, cerebral edema), and a serum salicylate level greater than 100 mg/dL at 6 hours postingestion. Because ingestion of more than 300 mg/kg predicts severe toxicity, a nephrologist should be consulted 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 commonly 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 is much more to worry about now that there are extended-release preparations and reports of hepatotoxicity resulting from unintentional supratherapeutic ingestions. The IV form of the antidote *N*-acetylcysteine (NAC) is being used more often than the oral formulation.

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 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 71-2.

20. What are the initial CNS manifestations of acetaminophen poisoning?

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. Occasionally there may be mild nausea or vomiting with massive ingestions initially. Hepatic encephalopathy can occur in phase III.

21. Describe the pathophysiology of acetaminophen toxicity.

Acetaminophen is metabolized primarily by the liver. About 90% 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 by-products 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.

Table 71-2. 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.
*Note that these are rarely used in clinical practice but can be of diagnostic benefit.

22. How is hepatotoxicity predicted?

An acute ingestion of 7.5 g or 150 mg/kg (whichever is less) 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. Of note is concomitant diphenhydramine ingestion, which may alter absorption of acetaminophen. If the initial level is close to the treatment line on the nomogram, consider checking another level in 2 hours.

23. Are serial serum acetaminophen levels helpful?

If an accurate estimate of the time of ingestion cannot be obtained, the nomogram cannot be used. Treat patients as having an unknown ingestion, and check a single acetaminophen level and liver function panel; if the acetaminophen level is greater than 20 μg/mL or liver function panel is elevated, treat with NAC for 12 hours and then repeat the test. If acetaminophen is nondetectable and liver function has improved, NAC can be halted; otherwise, continue and contact the local poison center.

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 or the fact that it is in a solution form. 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 a cathartic (e.g., sorbitol or magnesium sulfate) should be administered orally or by gastric lavage tube (if the airway is protected and NAC is delayed). 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, preferably within 10 hours, after an acute overdose with a serum acetaminophen level above the treatment line on the nomogram. Alternatively, NAC is recommended for an acute ingestion in excess of 150 mg/kg or 7.5 g total dose (whichever is less). NAC should also be given intravenously to patients with hepatic failure

where acetaminophen poisoning is suspected, regardless of time after ingestion or acetaminophen level.

KEY POINTS: ED APPROACH TO ANALGESIC TOXICITY

1. Serial salicylate levels should be used to exclude toxicity before 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 NAC. It is most effective when administered within 10 hours, regardless of whether it is given orally or intravenously.

27. How is NAC administered?

Traditionally, NAC (Mucomyst) has been given as the oral formulation by mouth or nasogastric tube, after first diluting it 1:5 with water or juice. The loading dose is 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. Clinicians are now using the IV formulation (Acetadote) more often because of ease of use, less risk of vomiting, and faster infusion time (21 hours versus 72 hours for the oral route), The dosage is 150 mg/kg in D_5W over 15 to 60 minutes, followed by 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours. 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.

28. 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 IV route is recommended in these cases.

29. If the patient has hepatic encephalopathy, is it too late for NAC therapy?

No, NAC should be given by the IV route at the usual dosage, followed by a steady infusion of 6.25 mg/kg/h until the patient is clinically better and has an INR less than 2. Failure to improve suggests the need for liver transplant. The local poison control center should be consulted (800-222-1222).

30. Should I be concerned about potential adverse reactions to IV 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 transient 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, albuterol and epinephrine, and discontinuation of the IV NAC. Oral NAC can be substituted.

31. What is the acetaminophen-alcohol syndrome?

Acute alcohol coingestion is said to be protective, because alcohol competes with acetaminophen as a substrate for cytochrome P-450, thus limiting the production of the toxic metabolite. In contrast, chronic alcohol abuse affects acetaminophen detoxification in two ways:

1. It lowers hepatic glutathione stores, resulting in a reduced capacity to detoxify the toxic metabolite.
2. It induces the cytochrome P-450 system, increasing the proportion of ingested acetaminophen that is converted to the toxic metabolite.

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?

NAC is recommended for patients with detectable acetaminophen levels greater than 20 μg/mL and evidence of liver injury. Treat for 12 hours, and then repeat acetaminophen level and liver function tests. If acetaminophen is not detectable and liver function has decreased or normalized, treatment can be stopped; otherwise, continue and contact your local poison control center for guidance (1-800-222-1222).

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.
- CNS 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, primarily with IV fluids and antiemetics. If hematemesis is present, there should be further investigation. A limited toxicology screen to search for other readily treatable toxins (i.e., salicylates, acetaminophen, opioids, barbiturates, cyclic antidepressants, and ethanol) is recommended if the patient is centrally depressed. Seizures should be treated with IV diazepam. Renal and hepatic function tests should be ordered for massive ingestions. 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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QUESTIONS

1. How can an acute ingestion of acetaminophen be risk stratified?
   a. Done nomogram
   b. Rumack-Matthew nomogram
   c. None of the above
   The correct answer is *b*.
2. Why do acetaminophen levels need to be checked on all patients?
   a. A significant ingestion can be silent.
   b. For risk stratification
   c. Both of the above
   The correct answer is *c*.
3. What are the characteristics of a massive ibuprofen ingestion?
   a. Nausea and vomiting
   b. Renal injury
   c. Both of the above
   The correct answer is *c*.

CHAPTER 72

BITES AND STINGS

Shawn M. Varney, MD, FACEP, FACMT

ARACHNIDA (CHIGGERS, SCABIES, SCORPIONS, AND SPIDERS)

1. What is the difference between *poisonous* and *venomous*?

Poisonous refers to a substance that may cause toxicity or death if absorbed, ingested, or inhaled (e.g., touching a poison dart frog, licking a *Bufo* toad). *Venomous* implies a mechanism of delivering the poison to the victim (e.g., snakes bite, scorpions sting). The key distinction is the delivery mechanism.

2. 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. Tarantula venom contains a family of voltage-gated sodium channel modulators that paralyze its insect prey but tend to cause little toxicity in humans, namely, localized pain, numbness, and lymphangitis. The bites usually do not cause necrosis or serious sequelae. More commonly, 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.

3. 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 2012 the American Association of Poison Control Centers (AAPCC) reported 2246 bites from *Latrodectus* and 1365 from *Loxosceles*. There were no deaths and only 21 major reactions attributed to *Latrodectus* bites (0.94%). Similarly, no deaths and 11 major reactions (0.81%) were attributed to *Loxosceles*. The envenomation syndromes and treatment of these two spiders are distinct (Table 72-1).

4. What is "mustov disease"?

It is a play on words. Although there were 9343 bites attributed to spiders reported to the AAPCC in 2012, this number underestimates the true incidence because these are only the cases reported to poison centers. Another confounder is the effect of "mustov disease" (as in, Doc, I woke up with this; I *must've* been bitten by a spider in my sleep). Numerous nonbite skin lesions (especially community-acquired methicillin-resistant *Staphylococcus aureus* abscesses) are unfairly blamed on spiders. Mustov disease is not specific to spider bites.

5. A 5-year-old boy has 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 caused this, and what is the treatment? (Clue: He had been wearing shorts.)

Chiggers are tiny mite larvae (0.3 mm) that cause intense pruritus. The diagnosis is based on identifying the characteristic skin lesions in a person with an outdoor exposure in a chigger-prone area. Itching begins within a few hours of exposure, and the papules can enlarge to form nodules in 1 to 2 days. Treat with topical (calamine) or oral antihistamines (hydroxyzine, diphenhydramine) and steroids for the symptoms. Wash the clothes in hot water and/or treat with permethrin. Mites do not burrow into the skin, and the pruritic reaction does not develop until the mites have already detached.

6. What are the distinguishing features of scabies?

Scabies bites are typically in the web spaces between the fingers and toes (also the penis, face, and scalp in children). In contrast to chiggers, scabies create burrows of pruritic, white, threadlike patterns with small gray spots at the closed end, where the parasite rests. Treat with a thorough

Abstract

This is a concise and complete review of bites and stings. This chapter covers chiggers, scorpions, spiders, ants, bees, wasps, lizards, mosquitoes, mammals, marine fauna, and snakes.

Keywords:

Arachnida, chiggers, scorpions, spiders, Formicidae, ants, Hymenoptera, bees, wasps, Heloderma*, lizards, mosquitoes, mammals, marine fauna, and snakes*

Table 72-1. Comparison of Black Widow and Brown Recluse Spiders

LATRODECTUS: BLACK WIDOW	*LOXOSCELES:* BROWN RECLUSE
Markings	
Red hourglass shape on ventral abdomen (female)	Dark, violin-shaped spot anterodorsally
Presentation	
Pain at the bite within 1 hour Target-shaped erythema, swelling, localized diaphoresis • Diffuse large muscle cramping, including the back, chest, and abdomen (may mimic peritonitis) • Latrodectisim: 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	
• Wound care • Opioid analgesics, benzodiazepines for spasm • Tetanus prophylaxis • 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 over 2 cm • Transfusion or dialysis • Consider hyperbaric oxygen therapy, corticosteroids, or dapsone but no evidence of efficacy in humans • No available antivenom

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Ig, Immune globulin; *IV*, intravenous.

application of permethrin 5% cream from the neck down and wash off after 8 to 14 hours (repeat 1 week later), or take oral ivermectin 200 μg/kg once and repeat 2 weeks later.

7. How dangerous are scorpion stings?

North American scorpions are generally not dangerous or 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 telson). 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 United States: Arizona, Utah, New Mexico, Nevada, and California) are capable of producing systemic toxicity, usually in small children. They can hitch a ride in unsuspecting travelers' luggage. In 2012 the AAPCC reported 19,262 patients with scorpion stings. There was moderate morbidity in 3.6% of patients, major morbidity in 0.03%, and no deaths.

8. What are the signs of scorpion envenomation?

The sting is acutely painful. Systemic manifestations are rare and mainly occur in small children, 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 in an awake patient.

9. What is the treatment for a scorpion sting?

Supportive care with local wound care, opioid analgesia, and benzodiazepines for the neuromuscular symptoms is the mainstay of treatment. A new U. S. Food and Drug Administration (FDA)-approved scorpion-specific fragment antigen–binding F(ab′)2 antivenom (Anascorp, Rare Disease Therapeutics, Franklin, TN) is available in the United States and costs more than $11,000 for a standard three-vial treatment dose. 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.

FORMICIDAE (ANTS)

10. I have a patient who received multiple stings from fire ants. What do I do?

Do not panic. Fire ants belong to the order Hymenoptera, which includes wasps and bees. 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. Cool compresses, oral antihistamines, and topical steroids for local stings suffice. For multiple stings, use a similar approach along with oral corticosteroids if systemic allergic manifestations are present.

HYMENOPTERA (BEES AND WASPS)

11. What types of reactions occur from Hymenoptera stings?

There are four types of reactions:

1. 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.
2. 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.
3. 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.
4. Unusual reactions reported after Hymenoptera stings include encephalitis, neuritis, vasculitis, and nephritis.

12. 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. In addition, Hymenoptera can release defense pheromones that attract other Hymenoptera and induce them to sting. 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. Stinger removal should be done as soon as possible, because the venom sac continues to pulse venom over the first minute after it has detached from the bee.

13. What about killer bees?

Africanized honeybees *(Apis mellifera scutellata)* were introduced into Brazil in 1956 as a potential honey producer in the tropical environment. They have migrated to the United States. Africanized bees and European bees are similar in appearance, venom toxicity, amount of venom they carry, and number of times they can sting—once. The difference lies in their aggressive defensive behavior. They swarm in larger numbers, pursue victims over greater distances (up to 1 km), and have a lower threshold for stinging. Hence victims typically receive multiple stings during an attack and therefore a greater venom burden. For this reason, the Africanized honeybees have been called *killer bees*.

14. 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, tell the patient to avoid bees and wasps. Second, have him or her carry medical identification describing the bee sting allergy, such as a medical alert bracelet. Third, the patient should carry and learn to use an epinephrine self-injector (Ana-Kit or EpiPen).

HELODERMA (LIZARDS)

15. Are there any venomous lizards in the world?

Yes, there are two species: the Mexican beaded lizard *(Heloderma horridum)* and the Gila monster *(Heloderma suspectum).* Both lizards 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 tissue 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 are difficult to visualize on radiographs. Envenomation is present in about 70% of bites.

16. How do I open the jaws of a Gila monster?

A few ways to disengage the powerful jaws are to submerge the lizard underwater, wrap a towel around its head to frighten it, pour alcohol on it, or pry open its mouth with a stick.

CULICIDAE (MOSQUITOES)

17. What is the major clinical significance of mosquito bites?

There are more than 3000 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 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 encephalitis, malaria, yellow fever, dengue fever, filariasis, West Nile virus, Ross River virus, chikungunya fever, and Rift Valley fever. They transmit disease to more than 700 million people annually, with at least 2 million resultant deaths in Africa, South and Central America, Mexico, and Asia.

MAMMALS (BATS, DOGS, CATS, FOXES, HORSES, HUMANS, RACCOONS, SKUNKS, AND WOODCHUCKS)

18. How many dog and cat bites are there annually in the United States, and what is the risk of infection?

The majority of mammalian bites that require medical attention are from dogs. It is estimated that 4.5 million dog bites occur annually, causing up to 885,000 victims to seek medical attention. The annual incidence of cat bites is about 400,000. 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 may have a risk of infection as high as 30%. Cat bites carry up to an 80% rate of infection, which may be the result of the deep inoculation of bacteria in the wound.

19. 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 1994 metaanalysis, Cummings showed a number needed to treat of 14 to prevent one wound infection after a dog bite. Antibiotics appeared to decrease the infection rate by one half. High-risk wounds (immunocompromised patients, puncture wounds, crushed tissue, hand or foot involvement, treated after 12 hours, and showing signs of infection) may do better when treated with antibiotics than low-risk wounds. Puncture wounds and wounds greater than 3 cm carry a threefold increased risk of infection. Finally, wounds infected with *Pasturella multocida* generally manifest symptoms and signs of infection within 12 to 24 hours of the bite. When choosing antibiotics, consider the polymicrobial nature of these infections (*Staphylococcus, Streptococcus, P. multocida*, anaerobes) and the cost of the antibiotic. Amoxicillin-clavulanate (Augmentin, or "Dog-mentin") is the drug of choice in patients not allergic to penicillin.

20. Can dog bites be closed primarily (sutured)?

Yes, dog bites can be sutured with a few caveats. Favorable conditions include wounds less than 8 hours old, high-pressure irrigation, and cleansing with povidone iodine. Wound infection rate is

similar between sutured and nonsutured wounds, and comparable among all age groups. Sutured wounds have better cosmesis scores, and wounds less than 3 cm in length and on the head or face also result in improved cosmesis.

21. What is *Capnocytophaga canimorsus*?

Capnocytophaga canimorsus (dysgonic fermenter [DF2]) is a gram-negative rod that requires special growth media and 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). It is a rare infection that carries a 25% to 36% mortality rate. Always ask if a patient with a dog bite has his or her spleen.

22. 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 throughout the world except for a few rabies-free areas: 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 raccoons, skunks, bats, foxes, and woodchucks should be considered a risk. Exposures from livestock, rodents, and lagomorphs rarely require postexposure prophylaxis, because the host dies before the rabies virus can replicate sufficiently. Consult your state health department for local recommendations.

23. What is postexposure prophylaxis for rabies?

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 [IM] in the gluteal muscle). Inject 1 mL of human rabies vaccine into the deltoid muscle (or the anterolateral thigh in young children) on days 0, 3, 7, and 14. Do not administer the rabies vaccine and the immunoglobulin in the same site. Also ask about tetanus immunization status.

24. 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 and 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, with the fingers in flexed position, and are notorious for becoming infected. The most commonly cultured organism from human saliva in fight bites is *Streptococcus*, followed by *S. aureus* (usually penicillin resistant); 31% of these wound infections are caused by gram-negative organisms, and 43% are the result of mixed gram-negative and gram-positive organisms. Up to 29% of these infections may be caused by 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 require meticulous care with thorough exploration and irrigation. Consider the polymicrobial nature of these infections when choosing antibiotics. If a patient has an infected wound, give broad-spectrum intravenous (IV) antibiotics and obtain a surgical consult for possible debridement.

MARINE FAUNA (SEA JELLIES, SHARKS, AND VENOMOUS FISH)

25. How do I treat jellyfish or other coelenterate stings?

Jellyfish are invertebrate marine animals with a gelatinous, umbrella-shaped body that does not possess cartilage or scales, and are not fish. A more accurate term is *jelly* or *sea jelly.* Jellies possess thousands of nematocysts on their tentacles and envenomate by injecting small harpoon-shaped spines into their prey. The discharge is triggered by either physical contact or chemical stimulation (e.g., fresh water). Frequently, undischarged nematocysts in the tentacles remain in contact with the victim's skin and may inadvertently be stimulated and inject additional venom. Pour acetic acid (vinegar) over the affected area for 30 seconds to inhibit nematocyst discharge in most sea jellies. One exception to using vinegar is with *Physalia* species (Portuguese man-of-war, or bluebottle jelly), which responds better to hot water (about 45°C [113°F]) for 60 minutes. Although popular in folklore, urine does not inhibit nematocyst discharge and may even stimulate discharge.

Avoid rinsing with fresh water. The nematocysts that remain in the skin can be removed by applying shaving cream, talc, baking soda, or flour, and then shaving the area, scraping the skin with a credit card, or applying sticky tape. The same treatment can be used for the stings from sea anemones or fire coral. There is an Australian box jellyfish *(Chironex fleckeri)* antivenom available. The recommended dosage for the antivenom is 1 to 3 ampules.

26. Name some venomous fish, and state what their venoms have in common. How can that feature of their venom be used in treatment?

Venomous fish have spines that inject a heat-labile poison (heat destroys the toxin). Examples of venomous fish include stingray, lionfish, scorpionfish, stonefish, catfish (i.e., freshwater catfish, sea catfish, coral catfish), and weever fish among others. 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 into hot water (45°C) for 60 minutes. There is an antivenom for stonefish envenomation. Give 1 to 3 vials IM.

27. 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.

28. How many people are killed by sharks worldwide annually?

Between 2006 and 2010 an average of 4.2 people died worldwide annually from unprovoked shark attacks.

CROTALINAE (RATTLESNAKES, COPPERHEADS, WATER MOCCASINS) AND ELAPIDAE (CORAL SNAKES)

29. What are distinguishing physical features of crotaline (pit vipers) and elapid snakes?

Pit vipers include rattlesnakes, copperheads, and water moccasins (or cottonmouths) and are distinguished by a heat-sensing pit located between the nostril and the elliptical pupil on their triangular head. The heat-sensing pit enables the snakes to sense the direction and size of their prey. Elapids include coral snakes (the main indigenous elapid in North America) and cobras, and have round heads and round pupils. Not all pit vipers have rattles, and not all rattlesnakes use their rattles.

30. 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. pit viper bites do not result in envenomation. Coral snakes, lacking long fangs, envenomate by chewing the skin. Up to 50% of their bites are dry.

31. How does a pit viper bite differ from an elapid bite?

Pit viper venom contains toxins that cause local tissue destruction, whereas elapids possess a neurotoxin that may cause weakness and respiratory paralysis. Clinical signs of pit viper envenomation include the presence of 1 to 2 fang marks that ooze nonclotting blood, surrounding ecchymosis, local edema, and severe burning pain. 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 of neurotoxic venom effect from coral snake envenomation include nausea, vomiting, headache, abdominal pain, diaphoresis, and pallor. Coagulopathy and tissue destruction are not features of coral snake envenomation, but respiratory paralysis is the feared outcome.

32. True or false: Snakebites are uncommon but highly lethal in the United States.

This statement is both true and false. In the United States, indigenous snakebites are uncommon, and mortality is rare. Snakebite is a problem of morbidity, not mortality. The 2012 AAPCC report documented 3663 pit viper snakebites and 84 coral snakebites but only two deaths (one from a rattlesnake and one from a cottonmouth). Rattlesnakes accounted for more than half of the major medical outcomes (i.e., produced life-threatening signs or symptoms, or resulted in significant residual disability or disfigurement), and copperheads for about one fourth. An additional 2854

snakebites occurred from nonpoisonous and unknown types of snakes and caused 1.3% major outcomes and no deaths.

33. List some of the epidemiologic characteristics of snakebites in the United States.

- 75% occur from April to October.
- 45% occur between 2:00 PM and 6:00 PM.
- Male-to-female victim ratio is 7:1.
- 55% of victims are age 17 to 27 years.
- 85% of bites are on the fingers or hand; 15% involve the foot or ankle.
- 30% to 60% of victims were intoxicated with ethanol (especially if pet snakes were involved).
- 15% had previous snakebites.

34. List the three main clinical effects of crotaline (pit viper) envenomation.

Crotaline snakebite manifests with three main clinical effects:

1. Local (e.g., pain, edema, ecchymosis, bullae, oozing blood)
2. Systemic (e.g., nausea, weakness, hypotension, fasciculations, or multiorgan dysfunction syndrome)
3. Coagulopathy (e.g., low platelets, elevated international normalized ratio [INR], low fibrinogen)

35. What FDA-approved antivenom is available in the United States for crotaline (pit viper) envenomation, and when should it be administered?

CroFab (Crotalidae polyvalent immune Fab, ovine antivenom) is antivenom produced from the pooled serum of sheep immunized with one of four crotaline snake venoms (western diamondback, eastern diamondback, Mojave, and cottonmouth), 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 (described 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 within 6 hours of the bite. After initial control, additional 2-vial maintenance doses are infused at 6, 12, and 18 hours. Each vial of CroFab costs the hospital approximately $2000.

Antivenom is indicated for any patient with progressive local tissue effects, hematologic effects (significantly abnormal prothrombin time [PT], platelet count, and fibrinogen level) or systemic signs (nausea, vomiting, hypotension, localized fasciculations) caused by the venom. Antivenom is not indicated for localized pain and edema that do not progress over 4 to 6 hours.

36. Is the antivenom maintenance dosing always required?

This is controversial. Some affirm giving maintenance doses, because they were included in the study data that the FDA approved for CroFab. Others state that antivenom dosing should be tailored to the severity of the envenomation and that achieving initial control may be adequate. Some physicians report that copperhead bites are less severe in general and may not require maintenance dosing or possibly any antivenom. A randomized, double-blinded trial is currently underway comparing CroFab versus placebo for mild to moderate venom effects from copperhead envenomation.

37. What aspect of CroFab antivenom theoretically contributes to the need for additional and/or maintenance doses of antivenom?

Because of 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. The FDA approved CroFab with maintenance dosing. The Mexican pharmaceutical company Instituto Bioclon (Mexico City, Mexico) has developed an equine-derived polyvalent crotaline immune F(ab′)2 antivenom (Anavip) for use in Mexico that is effective in neutralizing 15 venoms from North American snakes and improves coagulopathic profiles. The F(ab′)2 fragment is larger than the Fab and is not removed from the patients' circulation as quickly as the Fab fragment antivenom. The F(ab′)2 antivenom is undergoing FDA review but is not approved.

38. Can a crotaline bite cause compartment syndrome?

Compartment syndrome rarely results from crotaline envenomation, 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, but it is still rare. Compartment syndrome cannot be diagnosed reliably without directly measuring intracompartmental pressures, because the signs and symptoms (e.g., paresthesias, pain on motion, and decreased pulses) are similar to signs and symptoms of envenomation. Antivenom is the treatment for compartment syndrome. In addition, elevate the extremity.

39. What is the importance of the coloring of coral snakes, and what are the active components of its venom?

This small (usually up to about 18 inches), thin, brightly colored snake is venomous; however, the king snake, which is nonvenomous, has similar coloration but a different pattern. "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.

40. How is coral snake envenomation treated?

Coral snake antivenom is no longer manufactured in the United States. Any remaining supply has exceeded its initial shelf life, but the FDA continues to extend the expiration date. Supportive care with good wound care and attention to impending muscle paralysis are the treatment. Coralmyn, a coral snake antivenom produced by the Mexican pharmaceutical company Instituto 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.

41. Which prehospital treatments for crotaline bites 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% to 2% in one study). Nonsteroidal antiinflammatory drugs may compound a crotaline venom–induced thrombocytopenic bleeding diathesis and should be avoided.

42. Which prehospital non-antivenom treatments are reasonable?

Remain calm, avoid activity, remove jewelry or constricting items, immobilize the extremity, follow good basic life support principles, and transport the patient rapidly to the ED. A lymphatic constriction band (broad and flat band as opposed to a ropelike tourniquet) is controversial but 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 delays the systemic absorption of venom and may have use in cases with prolonged transport time. Despite the American Heart Association's adoption of this practice in its 2010 guidelines, many toxicologists recommend against using constriction bands. Local tissue damage may occur, and people have difficulty applying the correct amount of pressure.

43. What about exotic snakes (at least exotic by North American standards)?

In 2012 the AAPCC reported 110 exotic snake exposures (i.e., poisonous, nonpoisonous, and unknown if poisonous). There were four major medical outcomes (3.6%) and one death. 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.

44. What are other general guidelines for crotaline (pit viper) snakebite patient care?

Antibiotics for snakebite are not indicated. Elevation of the affected extremity, along with antivenom, reduces pain. Avoid nonsteroidal analgesics due to antiplatelet effect from snake venom. Remove constricting bands/jewelry from patient's extremities. Administer the same dose of antivenom to adults and children, because dosages are based on the amount of venom injected, not the size of the patient. Finally, avoid fasciotomy.

KEY POINTS: BITES & STINGS SECRETS

1. *Centruroides* scorpion stings are most dangerous for small children. Severe envenomation manifests as salivation, tachycardia, roving eye movements, involuntary muscle jerking, opisthotonos, and tongue fasciculations in an awake patient.
2. Dog and cat bites are common, but rabies is uncommon. Nevertheless, administer rabies immune globulin and the vaccine series (on days 0, 3, 7, and 14) to patients experiencing bites from suspicious animals.
3. Treatment for marine envenomations: If stinging attack, pour vinegar over wound for 30 seconds. If spine attack, immerse affected area in hot water (45°C [113°F]) for 60 minutes.
4. Crotaline (pit viper) venom contains toxins that cause local tissue destruction, as well as systemic findings and coagulopathy. In contrast, elapids possess a neurotoxin that may cause weakness and respiratory paralysis but no tissue destruction.
5. CroFab (Crotalidae polyvalent immune Fab, ovine) is antivenom for crotaline (pit viper) bites. Four to six vials are administered initially and can be repeated until initial control is achieved. Maintenance doses are controversial.

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QUESTIONS

1. Which of the following statements is true regarding Hymenoptera stings?
   a. Africanized honeybees may sting multiple times, resulting in toxic venom levels and have caused human deaths.
   b. People who survive anaphylaxis from a bee sting should avoid bees/wasps, wear an allergy bracelet, and should carry an epinephrine self-injector.
   c. The best way to remove the Hymenoptera stinger is with a pair of forceps/tweezers.
   d. Bee/wasp stings often cause a local cellulitis that should be treated with antibiotics.
   The correct answer is *b*.
2. Rattlesnake, cottonmouth, and water moccasin snakebites may result in all of the following clinical findings except:
   a. Systemic (e.g., nausea, weakness, hypotension, fasciculations, or multiorgan dysfunction syndrome)
   b. Coagulopathy (e.g., low platelets, elevated INR, low fibrinogen)
   c. Neuromuscular (e.g., muscle weakness, respiratory paralysis)
   d. Local (e.g., pain, edema, ecchymosis, bullae, oozing blood)
   The correct answer is *c*.
3. Which of the following statements is true regarding *Latrodectus* (black widow) and *Loxosceles* (brown recluse) spider bites?
   a. *Latrodectus* spiders are identified with a dark, violin-shaped spot anterodorsally, whereas *Loxosceles* have a red hourglass shape on the ventral abdomen of the female.
   b. *Latrodectus* and *Loxosceles* are aggressive spiders.
   c. *Loxosceles* bites cause a syndrome of facial muscle spasm, lacrimation, photophobia, and periorbital edema classically known as *loxoscelism.*
   d. *Latrodectus* bites are painful, cause local erythema, swelling, and diaphoresis, and may cause diffuse large muscle cramping of the back, chest, and abdomen (which may mimic peritonitis).
   The correct answer is *d*.

CHAPTER 73

SMOKE INHALATION

Richard E. Wolfe, MD

1. What is the most common way to die in a fire?

Although there are many ways to die in a fire, smoke inhalation is by far the most common cause. It accounts for as much as 80% of fire-related deaths. Up to 20% of fire victims have an inhalation injury. The prevalence of smoke-related lung injury increases with the burn surface area, but significant pulmonary complications may still occur even without cutaneous burns.

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. Injuries from superheated air are thus generally limited to the upper airway. Lung injury from smoke inhalation is usually caused by a wide variety of toxic substances that cause direct chemical injuries and inflammatory responses from the trachea to the alveoli. Steam, however, has 4000 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%-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.

1. Acute respiratory distress occurs 1 to 12 hours postinjury and is caused by bronchospasm, laryngeal edema, and bronchorrhea.
2. Noncardiogenic pulmonary edema (adult respiratory distress syndrome) occurs 6 to 72 hours postinjury secondary to increased capillary permeability.
3. Strangulation occurs 60 to 120 hours postinjury from cervical eschar formation in patients with circumferential neck burns.
4. Onset of pneumonia occurs 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 given 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 the patient's airway has been intubated, it 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

Abstract

This chapter contains a concise review of smoke inhalation and management.

Keywords:

Smoke inhalation, fire-related injuries, acute lung injury, cyanide poisoning, hyperbaric oxygen (HBO), carbon monoxide (CO) poisoning, inhalation injury

- 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 after 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 for smoke inhalation?

- HBO therapy provides increased oxygen to poorly functioning mitochondrial enzymes inhibited by CO and cyanide.
- HBO therapy at 3 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 on the endothelial membrane, which prevents neutrophils from infiltrating the central nervous system and causing a damaging inflammatory reaction and permanent neurologic sequelae.

Despite these theoretic benefits, there has not yet been sufficient evidence to obtain consensus on the use of HBO therapy for smoke inhalation.

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 (COHb), 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 arterial partial pressure of oxygen (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 partial pressure of carbon dioxide [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?

Chest radiographs are normal immediately after smoke inhalation injury, and abnormalities usually appear on a delayed basis. A chest radiograph is usually not indicated in asymptomatic patients, and in most instances, it is useful only as a baseline in symptomatic patients. A decision to obtain a radiograph should be made on a case-by-case approach, pending clinical presentation.

14. Can I use the standard burn formula for intravenous (IV) 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

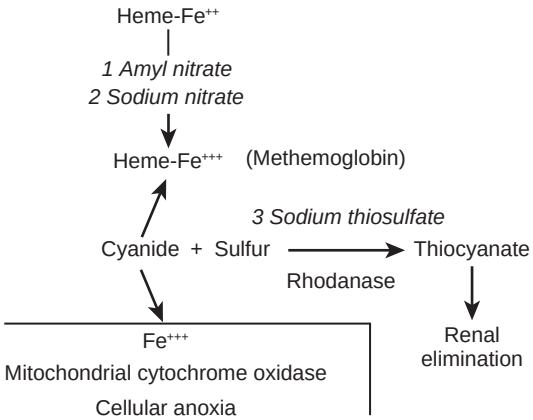

Figure 73-1. Mechanism of the Lilly cyanide antidote kit. *Fe,* Iron.

develop membrane permeable pulmonary edema. IV 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 (CYANOKIT) 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 IV. Unlike the cyanide antidote kit, it does not cause methemoglobinemia and may be redosed as needed.

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:

1. Nitrites generate methemoglobin, creating heme-ferric ions to compete with cyanide with mitochondrial ferric ions.
2. 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. 73-1).

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 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).

19. How do I administer the cyanide antidote kit?

Administer a dose of 12.5 g of IV sodium thiosulfate. Amyl nitrite inhalers in patients in extremis without IV 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 IV line is established and indications for nitrites are present, the full amount of a 10-mL ampule, or 300 mg 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 from all patients in whom the diagnosis is considered.

CONTROVERSY

22. Is 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 caused by 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 given 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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QUESTIONS

1. All of the following patients should be treated with high-flow oxygen via a nonrebreather mask after exposure to smoke except:
   a. An asymptomatic pregnant patient found inside a burning house
   b. An unconscious child from a burning home
   c. An elderly woman with chest pain
   d. A man who was close proximity to a pile of rubbish burning outdoors
   The correct answer is *d*.
2. When should hydroxocobalamin be used for the treatment of cyanide exposure?
   a. When there is a concern for concomitant CO and cyanide exposure
   b. In adults
   c. In children
   d. In the elderly
   The correct answer is *a*.
3. When should you obtain a CO level?
   a. Any patient with significant fire exposure in an enclosed space
   b. A patient found down in a car in a garage
   c. Patients found down in an apartment fire
   d. All of the above
   The correct answer is *d*.

CHAPTER 74

COMMON DRUGS OF ABUSE

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1. Are 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 in opioid-related deaths since 1995, with a steep increase since 2003. Analgesics (opioids and nonopioids) are the most common killer in toxic ingestions of pharmaceuticals every year since 1995. Opioids represent approximately half of those deaths each year. Since 2008, the Centers for Disease Control and Prevention (CDC) has reported that unintentional overdose, mostly from opioids, is the most common cause of unintentional death, higher than traumatic deaths from motor vehicle accidents.

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 is 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 patients with opioid intoxication cases have miosis?

No, mydriasis or normal pupils can occur in the following cases:

- Intoxication of specific synthetic opioids (i.e., meperidine [Demerol], propoxyphene, or pentazocine)
- Diphenoxylate-atropine (Lomotil) use
- 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 74-1).

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's airway if the patient does not awaken after naloxone, but make sure to give him or her enough time to respond to naloxone. Obtain a serum glucose level and administer oxygen. Consider activated charcoal if the opioid ingestion was recent and the airway is protected.

6. What is the appropriate naloxone dose?

For children younger than 5 years or less than 20 kg, administer 0.01 mg/kg intravenously (IV) for respiratory depression initially. If there is no response, then attempt 0.1 mg/kg IV. In an adult patient who has coma and respiratory depression (severe bradypnea or hypoxia), infuse an initial dose of 0.2 to 0.4 mg IV. If there is no response to this dose, repeated doses up to 2 mg can be given. Administer 1 to 2 mg IV initially for the apneic or cyanotic adult or child. For patients who abuse

Abstract

This chapter is a comprehensive summary of the emergency clinical presentation and treatment of common drugs of abuse, including cocaine, amphetamines, opioids, synthetic agents, cannabis, and common hallucinogens.

Keywords:

cocaine, amphetamine, opioids, heroin, bath salts, synthetic cannabinoids, cannabis

Table 74-1. Common Causes of Non-opioid-Related Miosis	
Sympatholytic agents	Clonidine, antipsychotics, oxymetazoline, and tetrahydrozoline
Cholinergic agents	Organophosphates, carbamates, nicotine, pilocarpine, phencyclidine, and similar congeners
Miscellaneous	Pontine infarct and Horner syndrome

opioids or who use opioids for chronic pain, infuse 0.05 to 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 IV?

Yes, if venous access cannot be accomplished, administer the naloxone intramuscularly or subcutaneously. A dose of 0.8 mg intramuscularly has an equal time to effect as 0.4 mg IV. Naloxone can also be administered via an endotracheal tube, intranasally via a nebulizer, intraosseously, or via sublingual injection. 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 IV 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 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 1 L 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 has 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 I observe in the ED, and for how long?

It depends. Observe patients who have injected opioids for at least 2 hours after a dose of naloxone, because 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 in an asymptomatic patient to be adequate. 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. Observe for 8 hours or admit the patient who ingested short-acting oral opioids, such as oxycodone or hydrocodone. Admit patients who have ingested and injected long-acting opioids, such as methadone or long-acting preparations of oxycodone, for 12 to 24 hours or longer. Patients should be normoxic off oxygen, awake, and ambulatory before discharge. The patients should 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, altered mentation, dysrhythmias, and other life-threatening complications are not consistent with opioid withdrawal.

14. How is opioid withdrawal best treated?

Treatment is symptomatic. Treat with IV fluids, sedation, antiemetics, and antidiarrheal agents. 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 administered, the most severe withdrawal symptoms commonly 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 and packers be managed?

Urine drug screening is not helpful for determining which drug, if any, was ingested. In addition, the patient's history for timing, content, and amount of ingested substance 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 whether 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 result 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 has 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 taking 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 (MAOIs) is also contraindicated, because MOAIs 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?

Diphenoxylate 2.5 mg plus atropine 0.025 mg (Lomotil) can be toxic. Most toxic cases occur in children. Classically the overdose is a two-phase toxicity: phase 1, with anticholinergic symptoms (flushing, dry mouth); and phase 2, with 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 (Imodium) 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 2000 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.

1-Methyl-4-phenyl-1,2,5,6 tetrahydropyridine (MPTP) is a compound that was produced accidentally during the synthesis of desmethylprodine or 1-methyl-4-phenyl-4-propionoxypiperidine (MPPP), a meperidine analog. MPTP is cytotoxic for dopaminergic neurons in the substantia nigra. It produces a parkinsonian-like syndrome that is permanent and occurs after a single ingestion of MPTP. The symptoms do not respond to typical antiparkinsonism 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. Although 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 a common 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 drug?

Sedatives-hypnotics drugs 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 four groups: benzodiazepines, barbiturates, the "Z-drugs" (zolpidem, zopiclone, zaleplon, and eszopiclone), and the miscellaneous group. Some examples of miscellaneous sedative-hypnotics are chloral hydrate, ethanol, and γ-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 are the Z-drugs?

These drugs are nonbenzodiazepines that are prescribed for insomnia. Many of these drugs are have names that start with the letter *Z*. Their mechanism is similar to benzodiazepines, by binding at the α-1 subunit of the γ-aminobutyric acid (GABA)-A receptor, and are useful as a sleep aids. Because of its specific affinity for the α-1 subunit, zolpidem has minimal muscle relaxant, anxiolytic, and anticonvulsant properties. Therefore intoxication presents with CNS depression, and respiratory depression is uncommon. Their structure is distinct from benzodiazepines. The Z-drugs can be reversed with flumazenil. Hallucinations and psychosis are rare but unusual adverse effects of some of these drugs. The Z-drugs can impair driving and alertness for up to 8 hours after taking a dose. Women may be more affected by the sedation than men, and thus a lower dosage is advised in women.

31. 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

Table 74-2. 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 74-3. 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

specific to individual drugs. Some examples are choral hydrate (pear odor), ethchlorvynol (pulmonary edema, vinyl odor), and glutethimide (anticholinergic effects) (Table 74-2).

32. Many overdoses seem to present this way, so how are sedative hypnotics different?

Many overdoses present with CNS depression. However, some intoxications also present with a pattern of symptoms known as a *toxidrome* (Table 74-3). 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 when evaluating these patients for such illnesses as meningoencephalitis, intracranial hemorrhage, hypoglycemia, shock, and sepsis.

33. How do sedative-hypnotics cause CNS depression?

Most sedative-hypnotics, particularly benzodiazepines and barbiturates, cause CNS depression by enhancing the effects of GABA, an inhibitory neurotransmitter in the brain. Benzodiazepines increase the rate 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.

34. 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 his or her 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 or timely.

35. Is there a role for drug screens or specific drug levels?

Routine drug screens are often not useful in acute management of these patients. 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.

36. 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 neurologic 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.

37. How do patients die of sedative-hypnotic overdose?

Respiratory depression and resultant hypoxia is the cause of most deaths.

38. What is the appropriate way to decontaminate the gastrointestinal tract?

Administer 1 g/kg orally of activated charcoal to all patients with life-threatening ingestions who arrive within approximately 1 hour of ingestion. Pulmonary aspiration of activated charcoal causes a significant pneumonitis and occasionally permanent sequelae. Intubate airways of patients with decreased mental status and airway reflexes before administering activated charcoal. Do not perform orogastric lavage for sedative-hypnotic intoxication. Do not insert a nasogastric tube in the sedated or vomiting patient, because the patient may aspirate the charcoal and cause chronic pulmonary disease.

39. Are there specific antidotes for sedative-hypnotic intoxication?

Flumazenil can be used for overdose of benzodiazepine and related medications such as zolpidem.

40. 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 IV in increasing doses to a generally accepted maximum dose of 5 mg. Most patients respond to 0.6 to 1 mg; infuse at a rate of 0.2 mg/min. If there is no response with 5 mg, consider another intoxication, coingestant, or other source for the patient's responsiveness.

41. 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-naive 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 the duration of effect is 1 to 4 hours. Sedation will resume after its effects have worn off. Most patients with a benzodiazepine overdose only require supportive care and do not require flumazenil.

The ideal patient for flumazenil use would be one with iatrogenic oversedation who does not have the following:

- Prior seizure history
- Electrocardiogram (ECG) evidence of cyclic antidepressants
- Chronic benzodiazepine use
- Abnormal vital signs, including hypoxia
- Coingestants that provoke seizures or dysrhythmias

42. What is GHB?

GHB 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 U.S. Food and Drug Administration (FDA) 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 γ-butyrolactone and 1,4-butanediol, are metabolized to GHB and have the same effects and are common.

43. How does a GHB overdose present?

Most ingestions of GHB are mild and produce minimal sedation and euphoria. Rarely, patients overdose on GHB and come 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.

44. 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.

45. What is a Mickey Finn, and what are date rape drugs?

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. Date rape drugs (encompassing a vast array of agents that cause CNS depression) are often used in a similar fashion to induce CNS depression, causing the victim to lose consciousness and leading to assault (see Chapter 78).

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 74-4).

Table 74-4. 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.

47. Which mushroom's toxins cause the most concern?

The most concerning are 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.

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 or phencyclidine)

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 commonly occurs as a result of the disinhibition and aggressiveness caused by hallucinogen abuse.

53. Why would someone "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)
- 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 freebase 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 because of 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, IV, and smoked cocaine, even in young patients with normal coronary arteries. Benzodiazepine is the initial treatment of choice for cocaine-induced chest pain.

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 CHCl, this pure base form of methamphetamine HCl evaporates easily at room temperature and is absorbed rapidly from the lungs. Similar to IV methamphetamine, it causes an immediate euphoric effect but without the risks of IV 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 MDMA. *Eve* is a street name for 3,4-methylenedioxyethylamphetamine (MDEA) 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 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, by adding cooling measures (i.e., evaporation, cooling blanket, and cool IV fluids), or by sedation with paralysis. 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 patient with cocaine toxicity, because they allow unbridled α-agonism, which can result in elevated blood pressure and coronary artery vasoconstriction.

65. What are "bath salts"?

Bath salts are designer drugs that are synthetic β-ketone cathinones, which are similar to amphetamines in structure. They are a white powder that is similar in appearance to Epsom salt and other actual bath salts. They are often sold as potpourri or plant food "not for human consumption" to avoid the laws prohibiting these products. Effects are similar to amphetamines (tachycardia, hypertension, psychomotor agitation), and psychosis, hallucinations, and aggression may be more common with these drugs. Common examples are mephedrone, methedrone, methylenedioxypyrovalerone (MDPV), Methylone, and pyrovalerone.

66. What are synthetic cannabinoids?

Synthetic cannabinoids are research chemicals that are analogs to tetrahydrocannabinol [THC], cannabidiol [CBD], and cannabinol [CBN]. The chemicals are infused into herbals and then consumed (most commonly smoked) by the user. Common names are *spice, space, K2,* and *chill out.* They are sold in head shops as drugs that are "not consumable for humans," similar to bath salts. They are sold as potpourri or as plant food, albeit most of the consumers buy it to consume it by smoking or ingesting. Effects include symptoms typical of marijuana use, including eye injection, euphoria, hunger, tachycardia, and paranoia. In addition, these drugs can cause psychosis, seizures, hallucinations, and acute kidney injury. They are not detected on the drug screen for THC. Treatment is supportive.

67. Can consumable (edible or drinkable) marijuana cause a patient to come to the ED?

Yes, consumable (edible or drinkable) THC products are concentrated and contain 5 to 50 times more THC than THC designed for smoking. Children, in particular, can develop sedation, hallucinations, and altered mentation with accidental ingestions. Adults can develop similar symptoms with large ingestions or with intentional "overdose" of concentrated edible THC products.

68. I had a patient with ear and nose ischemia from cocaine use. Why did that happen?

Cocaine use alone should not cause this type of focal ischemia. However, most cocaine is adulterated or contaminated, and one of the most common adulterants is levamisole. Levamisole is an anthelminthic and immunomodulator. It can cause vasculitis and neutropenia.

ANTICHOLINERGIC AGENTS

69. What are anticholinergic agents, and how do they present?

Common anticholinergic agents, including antihistamines, classically diphenhydramine, along with other over-the-counter cough and cold products. Another profoundly anticholinergic agent is jimson

weed, a weed that can be found ubiquitously across the United States and is often steeped for tea to be ingested; it is profoundly anticholinergic. The anticholinergic toxidrome consists of altered mentation but not aggression, dry flushed skin, mydriasis, hypertension, hyperthermia, tachycardia, urinary retention, and seizure. Physostigmine may be used diagnostically to identify patients who might have ingested jimson weed; however, care must be taken if other coingestants like tricyclic antidepressants or diphenhydramine have been used.

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 because of 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 occult triad of death (OTD): pyrexia, acidosis, and rhabdomyolysis.

70. 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 level. Also, obtain a creatine kinase level and repeat it if the patient continues to be physically agitated. Finally, check the patient's core temperature early and again before disposition.

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QUESTIONS

1. What is the classic triad of opioid toxicity?
   a. Agitation, tachycardia, and seizures
   b. Coma, respiratory depression, and miosis
   c. Mydriasis, coma, and respiratory distress
   d. Hypotension, tachycardia, and sedation
   The correct answer is *b*.
2. What is the most common adulterant of cocaine?
   a. Bath salts
   b. Strychnine
   c. Table salt
   d. Levamisole
   The correct answer is *d*.
3. *Amanita* mushrooms cause:
   a. Liver toxicity
   b. Seizure
   c. Methemoglobinemia
   d. Fever
   The correct answer is *a*.

CARDIOVASCULAR TOXICOLOGY

Ryan Chuang, MD, and Jennie A. Buchanan, MD

CHAPTER 75

1. How do different poisons affect heart rate, blood pressure, and QRS duration?
See Table 75-1.

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 (TCA), propranolol, cocaine, diphenhydramine, carbamazepine, lidocaine chloroquine, cyclobenzaprine, and norpropoxyphene (a metabolite of propoxyphene). Patients poisoned with these agents have 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 sodium bicarbonate, patients with cardiovascular toxicity also often require fluids and vasopressors for hypotension, benzodiazepines for seizures, and endotracheal intubation for altered mental status.

4. What symptoms do patients with a calcium channel blocker (CCB) overdose experience?
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 have 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 (such as gastric lavage, activated charcoal, and whole bowel irrigation) 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 dosages, are used next. Calcium is an adjunctive treatment in toxicity. The dosage is intravenous (IV) 1 to 2 g of calcium chloride or calcium gluconate, and doses may be repeated every 10 minutes three to four times. Calcium chloride requires a central line or large IV line because of risk for vein necrosis, but delivers three times the amount of elemental calcium compared with calcium gluconate. An infusion of calcium may be used to keep the serum calcium at the upper limits of normal.. Glucagon (5 to 10 mg IV push) may be given; if improvement is noted, a drip at 5 to 10 mg/h should be initiated. Glucagon may cause vomiting, so patients must be able to maintain their own airways or have airways intubated in order for the drug to be administered. Hyperinsulinemia euglycemia (HIE) therapy or high-dose insulin (1 unit/kg bolus, 25 g dextrose bolus, then 0.5 units/kg/h with supplemental dextrose 0.5 g/kg/h and potassium) is a newer therapy that has shown some efficacy. Heroic

Abstract

Accidental or purposeful overdose with cardiovascular drugs is a common cause of life-threatening ingestions encountered in all EDs. This chapter discusses in detail the pharmacokinetics and management of these overdoses.

Keywords:

calcium channel blocker (CCB), β-blocker (BB), sodium channel blocker, digoxin toxicity, antibody fragments (Fab)

Table 75-1. Cardiovascular Effects of Different Poisons

Bradycardia with Hypertension

- Centrally acting presynaptic α_2-agonists (clonidine, guanfacine, 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, guanfacine, oxymetazoline, and tetrahydrozoline): Inhibit sympathetic outflow in the central nervous system, resulting in hypotension, bradycardia, pinpoint pupils, and somnolence.
- BBs without sodium channel effects
- 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 caused by decreased cardiac conduction, which may degenerate into ventricular tachycardias
- Quinidine, procainamide, and disopyramide (class 1a antiarrhythmics that cause sodium channel blockade, prolonged QRS and QT intervals): Patients may present with bradycardia caused by decreased cardiac conduction, which 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): Because of decrease in vagal tone and agitation from delirium

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: Result of 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

BB, β-Blocker; *CCB*, calcium channel blocker; *CNS*, central nervous system.

measures, such as using IV lipid emulsion (1 to 2 mL/kg of a 20% lipid emulsion, followed by an infusion of 0.25 mL/kg/min for 30 to 60 minutes), extracorporeal membrane oxygenation, intraaortic balloon pump, and cardiopulmonary bypass, may be used in severe refractory cases. Other experimental therapies to mention include methylene blue (1 to 2 mg/kg), the calcium sensitizer inotrope levosimendan (6 to 12 μg/kg bolus over 10 minutes and then a continuous infusion of 0.05 to 0.2 μg/kg/min), and L-carnitine (6 g IV bolus and then 1 g IV every 4 hours).

6. What are the symptoms in patients with β-blocker (BB)?

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 experience symptoms similar to those of 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 sometimes occur.

7. Describe the treatment for BB toxicity.

Treatment is similar to that for CCB overdose. Glucagon is used 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. There are case reports of using dialysis for atenolol overdoses, because it has relative low protein binding and volume of distribution.

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 rate of greater than 90% if untreated.
- Chronic digoxin toxicity occurs when there is a change in the dosage 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 seek treatment 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 administered?

Digoxin Fab may be administered 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: Milligrams of digoxin ÷ 0.5 mg of digoxin bound per vial = Number of vials needed to treat.
- If the steady-state serum level is known, the following formula should be used: Serum digoxin level (ng/mL) × Ideal patient weight (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, give sodium bicarbonate boluses 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.

Acknowledgment

We would like to thank Christopher DeWitt, Jody J. Rodgers, and Kennon Heard, who authored this chapter in previous editions and contributed substantially to this edition.

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QUESTIONS

1. In a digoxin overdose, if the patient is critically ill, what is the correct response?
   a. 1 to 2 vials of digoxin Fab should be given empirically.
   b. Get a level.
   c. 10 to 20 vials of digoxin Fab should be given empirically.
   d. No digoxin Fab is indicated.
   The correct answer is *c*.
2. A patient has hypotension, bradycardia and hyperglycemia. What did this patient ingest?
   a. CCB
   b. TCA
   c. Digoxin
   d. BB
   The correct answer is *a*.
3. Which of the following agents is a BB with sodium channel blocking effects?
   a. TCAs
   b. Diphenhydramine
   c. Cocaine
   d. Propranolol
   The correct answer is *d*.

PEDIATRIC INGESTIONS

George Sam Wang, MD, FAAP

CHAPTER 76

1. **How common are pediatric ingestions?**
   Approximately two thirds of ingestions reported to U.S. poison centers are pediatric, with 80% of those occurring in children younger than 6 years. 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 and have not yet developed the cognitive capacity to completely understand the potential danger of the ingestion. The increase in adolescence is largely the result of self-administration for self-harm or abuse purposes. The vast majority of pediatric ingestions result in minimal or no clinical effects, with the overall mortality rate less than 1%. Approximately 2% of pediatric exposures reported to U.S. poison centers result in moderate or major outcomes or death, with approximately 50% of those being in the adolescent age group.

2. **What is different about children from adults with ingestions and exposures?**
   Fortunately, most ingestions are lip, sip, or taste in nature and not large volume, which leads to low morbidity and mortality rates. However, small amounts of highly concentrated products or a therapeutic adult dosage of some medications can be very dangerous. Children can be more vulnerable to dense gas and vapor exposures, because they are shorter in stature and lower to the ground, have less capability to remove themselves from a dangerous environment, and have higher minute ventilation. They also have a large body surface area-to-weight ratio, making them more vulnerable to dermal exposures and hypothermia. Adolescent ingestions are similar to adult ingestions, because they are typically the result of drug abuse or suicidal gestures.

3. **What are some household agents that can be dangerous?**
   Most household products are benign in accidental ingestions or exposures. However, the following substances can be dangerous in low doses: caustics, hydrocarbons, products that contain ethanol or toxic alcohols, button or disc batteries, magnets, and camphor.

4. **What products contain caustics?**
   Many cleaning detergents, such as bathroom and kitchen cleaners, bleach, and rust and automotive cleaners, can contain either alkali or acidic caustic products.

5. **What are concerning signs after caustic ingestion?**
   Most caustics have a low enough concentration that small exposures do not cause significant injury. However, large-volume exposures, or highly concentrated products, can cause significant esophageal burns. Dangerous symptoms include stridor, persistent vomiting, and drooling. Some products, such as hydrofluoric acid, can cause systemic symptoms, such as hypocalcemia.

6. **What products contain hydrocarbons, and what are the symptoms of exposure?**
   Hydrocarbons include essential oils, kerosene, and petroleum distillates. Some can cause sedation and central nervous system (CNS) depression, but the most concerning exposure is aspiration, leading to pneumonitis.

7. **How should hydrocarbon exposures be managed?**
   Most children who are asymptomatic with normal chest radiography findings after 6 hours will not go on to develop serious toxicity; others may require hospital admission for hypoxia or respiratory distress. Antibiotics and steroids have not been shown to have significant benefit after acute exposure. Complications include respiratory failure, CNS depression, superinfection pneumonia, and pneumatocele formation. Significant toxicity requiring intubation as a result of respiratory distress, acute respiratory distress syndrome (ARDS), or poor oxygenation has been successfully treated with high-frequency ventilation, extracorporeal membrane oxygenation, and surfactant.

Abstract

Most unintentional pediatric ingestions and exposures are low risk and do not cause significant toxicity. However, some common medications and household products in small doses or volumes can easily reach toxic levels in pediatric patients.

Keywords:

pediatrics, ingestions, poisonings

8. What products contain ethanol and the toxic alcohols?

Many products contain high concentrations of ethanol, including hand sanitizing solution, perfumes, hair sprays, and food extracts. Methanol is typically found in windshield wiper fluid, and ethylene glycol is in antifreeze. Pediatric patients can develop more profound CNS depression and possibly hypoglycemia after small amounts of ethanol and at lower serum ethanol concentrations compared with adults.

9. When are button battery and magnet ingestions dangerous?

Button or disc batteries that are retained in the esophagus can lead to significant burns and erode through the entire esophageal wall, leading to sentinel bleeds and death. Magnets can also be dangerous, because ingestion of more than one can lead to bowel wall ischemia.

10. Are there any over-the-counter (OTC) products that can be dangerous?

Analgesics, such as acetaminophen and salicylates, are very common and can lead to liver failure and acidosis, respectively. Oil of wintergreen contains very high amounts of methyl salicylate (see Question 36). Lomotil (see Question 19) can also lead to dangerous toxicity. Iron is found in many OTC multivitamins (typically omitted in candy vitamins). Many cough and cold medications contain acetaminophen, dextromethorphan, and an antihistamine (diphenhydramine, doxylamine, chlorpheniramine, brompheniramine). Imidazolines found in eye drops and nasal decongestants can cause toxicity similar to clonidine (see Question 32).

11. How much iron is needed to cause significant toxicity, and what are the symptoms?

Approximately 20 mg/kg of elemental iron leads to symptoms. Common formulations include iron fumarate (33% elemental iron), iron gluconate (12%), and ferrous sulfate (20%). Symptoms classically progress through five stages:

1. Vomiting/gastrointestinal (GI) symptoms
2. Latent
3. Metabolic acidosis/shock
4. Hepatic failure
5. Gastric outlet obstruction

12. What are symptoms of OTC cough and cold medication overdose?

Dextromethorphan can lead to psychosis, agitation, hallucinations, sedation, and, rarely, seizures and serotonergic toxicity. Diphenhydramine can lead to anticholinergic toxicity, and in large overdoses, seizures and cardiac dysrhythmias, similar to tricyclic antidepressant toxicity. Acetaminophen is found in many of these preparations.

13. How do ingestions of camphor present?

Ingestions initially cause GI 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 after the exposure. There is no specific antidote, and treatment is primarily symptomatic and supportive. A 2009 case series suggested that camphor should be considered as a cause of undifferentiated seizures in children from communities with widespread use of the substance. Camphor is found in products such as Campho-Phenique, Vick's Vaposteam, Vick's VapoRub, Tiger Balm, Anbesol Cold Sore Therapy Ointment, BenGay Ultra Strength, and many other OTC topical creams. The U.S. 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. Five hundred milligrams can cause serious toxicity in a child. In an 11% solution, this would equal approximately 4.6 mL.

14. Why are children more predisposed to methemoglobinemia?

Pediatric patients younger than 4 months are at higher risk for developing methemoglobinemia because they do not have the ability to reduce ferric iron to ferrous iron, as do older children and adults. Common causes of methemoglobinemia include nitrites/nitrates (well water and foods), local anesthetics (benzocaine, lidocaine), dapsone, sulfonamides, naphthalene, and silver nitrate.

15. How is methemoglobinemia treated?

Indications are to treat include symptomatic patients with methemoglobin concentrations greater than 20% to 25%. Methylene blue is given at a dosage of 1 mg/kg intravenously (IV).

16. Are there any plants that can cause serious illness?

Most plants in small amounts will not cause significant toxicity. However, there are some plants that can be dangerous, such as foxglove, lily of the valley, oleander (digoxin-like toxicity), jimson weed/moon flower (anticholinergic toxicity), poison hemlock (respiratory paralysis), and water hemlock (seizures).

17. 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 dosage. 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.

18. What drugs may be found on the one pill can kill list?

Although not a consensus list, these drugs are often mentioned:

- Diphenoxylate and atropine (Lomotil)
- Tricyclic antidepressants
- Calcium channel blockers
- β-Blockers
- Sulfonylureas
- Clonidine
- Camphor
- Salicylates
- Phenothiazines
- Opioids
- Benzonatate

19. What are the components and clinical presentation of Lomotil, and what are their mechanisms of action?

Lomotil is an antidiarrheal agent composed of diphenoxylate (an opioid) and atropine (an anticholinergic).

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 ingestion should be thought of as ingestion of a long-acting opiate that may include features of atropine toxicity. Lomotil ingestions should be observed for 24 hours.

20. What is the potential lethal dosage of a tricyclic antidepressant (TCA)?

Ingestions of 10 to 20 mg/kg can lead to significant toxicity, with fatalities occurring from as little as 250 mg of amitriptyline in pediatric patients.

21. What electrocardiogram (ECG) finding in TCA ingestions is helpful in children?

The terminal 40-msec QRS axis has been shown in adults to be a useful marker in identifying TCA overdose. In a retrospective study of 35 children with TCA ingestions, the terminal 40-msec QRS axis was not helpful in predicting TCA ingestions. 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.

22. 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) because of direct cardiotoxicity, there is a report of a death in a 14-month-old child from a single 10-mg ingestion of nifedipine.

23. What is the pediatric dosage 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. In children, 0.1 to 0.2 mL/kg of 10% calcium chloride or 0.3 to 0.5 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.

24. What other therapy is used in treatment of calcium channel blockers and β-blockers?

Hyperinsulinemia/euglycemia has shown to be effective in animal models of calcium channel blocker toxicity. There are no human clinical trials, but experience published in human case reports and case series support improved hemodynamics with insulin/dextrose administration in both children and adults. The typical suggested starting dose is 1 U/kg insulin bolus, followed by a continuous infusion of 0.5 to 1 U/kg/h titrated to effect, with reports as high as 10 U/kg/h of insulin. Dextrose should be administered to maintain euglycemia.

25. What is a potential side effect of β-blocker and calcium channel 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-blocker pills is very unlikely to result in any toxicity. Calcium channel blocker toxicity may show hyperglycemia, because the release of insulin is a calcium-dependent exocytosis.

26. For how long should a sulfonylurea ingestion in a child be observed?

A child should be observed for 12 to 24 hours, depending on the type and preparation. 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.

27. How often should blood sugars be monitored?

Initially, blood sugars should be monitored hourly.

28. 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 his or her blood sugar up.

29. What is the rule of 50?

The *rule of 50* is a mnemonic for calculating a dextrose dosage 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.

30. What is considered the antidote of sulfonylurea ingestions?

Octreotide is the antidote. 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.

31. How is octreotide administered in pediatric sulfonylurea ingestions?

The appropriate dosage, 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 dosage is 1 μg/kg subcutaneously with an initial dosing interval of every 6 hours.

32. 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 CNS depression, respiratory depression, hypothermia, and miosis. No specific antidote exists; treatment is generally focused on general respiratory and hemodynamic support. Most unintentional ingestions do well.

33. 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.

34. What are some common OTC 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.

35. At what dosage 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.

36. How does the potency of methyl salicylate compare to salicylate?

One milligram of methyl salicylate is roughly as potent as 1.4 mg of salicylate. Methyl salicylate is found in oil of wintergreen, many topical OTC creams, and many Asian herbal remedies.

37. Approximately how much aspirin (or acetylsalicylate) is equal to 5 mL of 100% methyl salicylate?

Five milliliters (or 1 teaspoon) of 100% methyl salicylate is equal to approximately 7000 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.

38. 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).

39. What is the pathophysiology of chloroquine and hydroxychloroquine ingestions?

These drugs are believed to exhibit quinidine-like effects, 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 antiinflammatory agent. Although hydroxychloroquine is considered safer than chloroquine, both have the potential to cause serious toxicity, including cardiotoxicity, respiratory depression, CNS depression, and seizures.

40. What other drug besides standard therapy has been used to treat chloroquine poisoning?

Other than sodium bicarbonate for QRS widening, diazepam (2 mg/kg IV 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.

41. What newer opioid can result in significant toxicity with ingestion 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 given 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 administered to children at 0.01 mg/kg IV for respiratory depression and 0.1 mg/kg IV (up to 2 mg) for apnea.

42. What symptoms develop after benzonatate (Tessalon) perle ingestion?

Young children can become symptomatic after only ingestion of a few benzonatate perles. Toxicity is similar to local anesthetic toxicity and can lead to seizures, CNS depression, and cardiac dysrhythmias.

KEY POINTS: PEDIATRIC INGESTIONS

1. Most pediatric ingestions and exposures are low risk and do not cause significant toxicity.
2. However, pediatric patients can easily reach toxic dosages of common medications from small volumes of exposure.
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.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Dr. Shan Yin for his previous contributions to this chapter.

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QUESTIONS

1. What common OTC medications have a similar toxicity as clonidine?
   a. Nonsteroidal antiinflammatory drugs (NSAIDs)
   b. Nasal decongestants and ophthalmologic solutions
   c. Antihistamines
   d. Cough and cold preparations
   The correct answer is *b*.
2. What is considered the antidote to sulfonyurea toxicity?
   a. Glucagon
   b. High-dose insulin euglycemic therapy
   c. Octreotide
   d. Phosphodiesterase inhibitor
   The correct answer is *c*.
3. Which essential oil contains methyl salicylate?
   a. Tea tree oil
   b. Lavender oil
   c. Pennyroyal
   d. Oil of wintergreen
   The correct answer is *d*.

CHAPTER 77

PELVIC INFLAMMATORY DISEASE

David B. Richards, MD, and Bartholomew B. Paull, MD

1. What is pelvic inflammatory disease (PID)?

PID is a spectrum of acute infectious disorders involving the upper genital tract structures of women. PID can include any of the following: endocervicitis, endometritis, salpingitis, oophoritis, tuboovarian abscess, or peritonitis. The sexually transmitted organisms *Neisseria gonorrhea* and *Chlamydia trachomatis* are often causative agents, although vaginal flora has also been implicated.

2. What are the risk factors for PID?

Young women with multiple sexual partners have the greatest risk for PID. Other risk factors include earlier age at first intercourse, intrauterine device (IUD) insertion (not the presence of the device itself), and sexual activity during menses or immediately after menses. Older sex partners, prior involvement with a child protection agency, prior suicide attempts, alcohol use before intercourse, and concurrent *C. trachomatis* infection have also been shown to increase the risk of PID.

3. What are the signs and symptoms of PID?

There are no specific signs or symptoms that are diagnostic for PID. Lower abdominal pain is a common presenting symptom, although it may be subtle. Dyspareunia, abnormal vaginal discharge, abnormal uterine bleeding, or dysuria can be the only presenting symptoms. On examination, patients may have lower abdominal tenderness, cervical motion tenderness, and/or bilateral adnexal tenderness.

4. What are the microbiologic causes?

PID is a community-acquired infection typically initiated by a sexually transmitted agent, most commonly *N. gonorrhea* or *C. trachomatis*, although in many cases the etiology of PID is unknown. It is thought that a number of community-acquired agents are capable of disturbing the normal endocervical mucous barrier, allowing vaginal flora access to the upper genital tracts of women. Clinically, PID should be viewed as a mixed (facultative and anaerobic) polymicrobial infection after an initiating event. Microbes found in these infections include pelvic anaerobes, endogenous pelvic flora, gram-negative rods, group B streptococci, *Mycoplasma hominis, Staphylococcus aureus*, *Gardnerella vaginalis*, and *Haemophilus influenzae*.

5. What are the diagnostic criteria for PID?

There is no gold standard for the diagnosis of PID, and laboratory testing adds little to the diagnosis. A low threshold for the diagnosis should be maintained as delay in treatment leads to substantial morbidity. The Centers for Disease Control and Prevention (CDC) recommends empiric treatment of PID when cervical motion tenderness, uterine tenderness, or adnexal tenderness is associated with lower abdominal or pelvic pain in sexually active young women or other women at risk for sexually transmitted diseases (STDs). The diagnosis should be considered in any female patient with fever, vaginal discharge, abnormal bleeding, dyspareunia, or dysmenorrhea.

Additional criteria used to support a diagnosis of PID include the following:

- Oral temperature greater than 38.3°C (100.9°F)
- 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*

Abstract

Pelvic inflammatory disease (PID) is a constellation of primarily sexually transmitted diseases (STDs) affecting females that is common in all EDs. The diagnosis and treatment of these disorders is discussed in this chapter.

Keywords:

pelvic inflammatory disease (PID), gynecology

The most specific criteria for diagnosing PID include the following:

- Endometrial biopsy demonstrating endometritis
- Imaging demonstrating thickened, fluid-filled tubes with or without pelvic free fluid or 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.
- A catheter-obtained urinalysis may reveal a urinary tract infection.
- Nucleic acid amplification for *C. trachomatis* and *N. gonorrhoeae* should be obtained.
- Ultrasound for all pregnant patients to exclude ectopic pregnancy, patients being considered for admission because of systemic symptoms, or for those with a possible tuboovarian abscess.

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 includes:

- Cervicitis
- Endometriosis
- Ovarian cyst
- Ovarian torsion
- Spontaneous abortion
- Septic abortion
- Ectopic pregnancy
- Cholecystitis
- Appendicitis
- Diverticulitis
- Gastroenteritis
- Cystitis
- Pyelonephritis
- Renal colic

In some 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 short-term and long-term complications. Acutely, PID can result in tuboovarian abscess, perihepatitis (Fitz-Hugh-Curtis syndrome), or peritonitis. Long-term sequelae include chronic pelvic pain, tubal factor infertility, and increased risk of ectopic pregnancy. Chronic pelvic pain may occur in up to 33% of patients with PID. The incidence of infertility and ectopic pregnancy substantially increase with each episode of PID. This is thought to be primarily the result of tubal occlusion from scarring and adhesions within tubal lumens. The rate of a potentially fatal ectopic is 12% to 15% higher in women who have had PID.

9. Who should be hospitalized?

In women with PID of mild to moderate clinical severity, outpatient therapy is reasonable. Some criteria for hospitalization suggested by the CDC include the following:

- A surgical emergency (e.g., appendicitis) cannot be excluded
- Pregnant patients
- Patients who do not respond clinically to 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 tuboovarian abscess

10. Summarize the recommended antibiotic regimens for PID treatment.

See Table 77-1. Note that there maybe regional variation in treatment recommendations.

11. Are there alternative outpatient treatment regimens for PID?

Evidence suggests that azithromycin is more effective than doxycycline when given with ceftriaxone for cervicitis; that is, in patients with evidence of lower genital tract infection/inflammation and no pelvic organ tenderness. In a trial of women with cervicitis treated with ceftriaxone 250 mg intramuscularly and randomized to azithromycin 1 g orally once weekly for 2 weeks, versus

Table 77-1. Treatment of Pelvic Inflammatory Disease

Recommended Outpatient 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

Recommended Inpatient Regimen A

Cefotetan, 2 g IV every 12 hours, 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 the 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 tuboovarian 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.

Recommended Inpatient 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 after 24 hours. Doxycycline 100 mg PO bid or clindamycin 450 mg PO qid, after a patient improves clinically, should be used to complete a 14-day course of therapy. When tuboovarian abscess is present, clindamycin may be used for continued therapy rather than doxycycline, because clindamycin provides more effective anaerobic coverage.

Modified from Centers for Disease Control and Prevention: Sexually transmitted diseases treatment guidelines 2015. *MMWR Recomm Rep* 64:1–37, 2015.
bid, Twice a day; *IM*, intramuscularly; *IV*, intravenously; *PID*, pelvic inflammatory disease; *PO*, per os, orally; *qid*, four times a day.

doxycycline 200 mg/day for 14 days, results demonstrated a 90.3% versus 72.4% cure rate for the azithromycin versus the doxycycline group. The CDC does not recommend fluoroquinolones for treatment of gonococcal infections and associated conditions such as PID.

12. Does the presence of an intrauterine pregnancy effectively rule out PID?

PID can occur in pregnant women, although it is extremely rare. Suppurative salpingitis during the first trimester is reported in case reports, where the infection is most commonly thought to occur concurrently with fertilization.

13. Does a history of tubal ligation preclude the diagnosis of PID?

No, tuboovarian abscesses have been reported up to 20 years after tubal ligation.

14. What is the appropriate follow-up care for patients with PID?

Patients treated as outpatients should be assessed within 3 days. For reliable patients, a follow-up phone call may suffice. For all patients, a test of cure by repeat examination and cervical nucleic

acid amplification tests for *C. trachomatis* and *N. gonorrhoeae* is recommended 3 to 6 months after the initial intervention.

15. Summarize the principles of management of acute PID.

- Rule out pregnancy and surgical emergencies.
- Maintain a low level of suspicion for PID, because the consequences of untreated PID include infertility and chronic pelvic pain.
- Treat early with antibiotics if PID is suspected.
- Recommend all patients with PID be tested for other STDs, 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: PID

1. Maintain a high suspicion for PID in any sexually active patient with pelvic pain.
2. There are no historical, physical, or laboratory findings that can conclusively diagnose PID.
3. PID requires 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.
6. Rule out surgical emergencies and complications of pregnancy before empirically treating for PID.

Acknowledgment

The editors and authors of this chapter would like to acknowledge and thank Leslie L. Armstrong, MD, and Susan Brion, MD, for their previous contributions to this chapter.

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QUESTIONS

1. Complications of PID include:
   a. Infertility
   b. Chronic pelvic pain
   c. Increased risk of ectopic pregnancy
   d. All of the above
   The correct answer is *d*.
2. Which of the following statements is true?
   a. PID in a woman with an IUD mandates IUD removal.
   b. Tubal ligation precludes a diagnosis of PID.
   c. One must rule out complications of pregnancy and surgical emergencies before empirically treating PID.
   d. Laboratory analysis is critical in the diagnosis of PID.
   The correct answer is *c*.
3. Which of the following is an appropriate treatment regimen for PID?
   a. Ceftriaxone and doxycycline
   b. Azithromycin monotherapy
   c. Fluoroquinolones
   d. Penicillin
   The correct answer is *a*.
4. Indications for hospital admission in PID include all of the following except:
   a. A patient who is HIV positive with a normal CD4 count
   b. Pregnancy
   c. Tuboovarian abscess
   d. Patients who are unable to tolerate an outpatient oral regimen
   The correct answer is *a*.

CHAPTER 78

SEXUAL ASSAULT

Michelle Metz, RN, BSN, SANE-A, CEN, and Jennie A. Buchanan, MD

1. What is the definition of sexual assault?

The legal definitions of sexual assault vary from state to state. *Sexual assault* generally refers to any deliberate sexual contact to areas of the genitalia, anus, or mouth; manual penetration of the victim's body by way of force; threatened physical abuse; coercion; abuse of authority; or lack of victim consent. Individuals who have an impaired mental function because of intoxicants or central nervous system depression are unable to give consent. The more traditional term *rape* is defined by the Federal Bureau of Investigation (FBI) as penetration, no matter how slight, of the vagina or anus with any body part or object, or oral penetration by a sex organ of another person, without the consent of the victim. This definition of *sexual assault* is inclusive of gender, age, and sexual preference. Sexual assault is a crime of violence and control.

2. How common is sexual assault?

Sexual assault is one of the most underreported crimes; only 40% of sexual assaults are reported to law enforcement. Every 2 minutes someone in the United States is sexually assaulted. One in 6 women and 1 in 33 men will report a completed or attempted rape during their lifetime. Women ages 16 to 19 years are four times more likely to be sexually assaulted. Of women who report being raped, 44% were younger than 18 years. 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 women can perpetrate sexual assaults against other women or men. Approximately two thirds of sexual assaults are perpetrated by someone the victim knows, with only 3% sentenced to jail time.

3. What role does a medical provider have in cases of sexual assault?

The ED is the most common place for a victim of sexual assault to come for acute medical care and forensic evidence gathering. Thirty-two percent 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. The physician's primary responsibility is to provide for the patient's physical and psychological well-being. Then, if the patient consents, he or she should 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/forensic examiners (SAN/FEs) who have been specially trained to care for these victims of violence. These SAN-FEs are educated and trained to complete a comprehensive medical-legal examination. If a SAN/FE is not available, each ED should have a comprehensive ED sexual assault protocol that addresses medical care and evidentiary collection.

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 use, 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 that occurred before 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 without the presence of law enforcement personnel.

Abstract

This chapter discusses care of victims of sexual assault.

Keywords:

sexual assault, rape, postexposure prophylaxis, evidence collection, rape kit, medical forensic examination

Table 78-1. Forensic Evidence Kit Contents: Based on Jurisdiction

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

Modified 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, Stapczynski JS, editors: *Emergency medicine: a comprehensive study guide*, ed 6, New York, 2004, McGraw-Hill, pp 1851–1854.

5. What should be included in the physical examination?

The purpose of the physical examination is to detect injuries requiring treatment and to record and gather forensic evidence. A complete head-to-toe medical examination should be performed, regardless of whether law enforcement has requested a forensic examination or whether the patient has consented to a forensic examination. General body trauma occurs more commonly than genital trauma. Injuries may include, but are not limited to, 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, tears, bleeding, or tenderness. Semen or saliva may fluoresce under an alternative light source. 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 identify anogenital 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 may be necessary 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 78-1).

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 likely the result of the assault. The routine collection of gonorrheal 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 obtained in 2 weeks.

8. What about blood alcohol levels and tests for drug use?

In general, routine drug screens and routine tests for alcohol levels are not recommended. Proof of intoxication or drug use may or may not be used against the victim in court. Alternatively, collecting blood or urine may help prove the victim was too intoxicated to consent. If tests are medically indicated and will influence treatment (such as a patient with an unexplained altered mental status, or unexplained tachycardia), then laboratory testing may be indicated.

9. What historical features might indicate a drug-facilitated rape?

A history of amnesia or suddenly feeling very intoxicated at a social event 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 the 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 toxicologic screening. The samples should be refrigerated after receipt by law enforcement to preserve the detection of drugs of abuse. Conviction of DFSA increases legal penalties.

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, trichomoniasis, 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 geographic 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, hepatitis C, and HIV can be transmitted via sexual contact.

11. Is empiric 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 g orally, or doxycycline 100 mg orally twice a day for 7 to 10 days for chlamydial prophylaxis, or ceftriaxone 250 mg intramuscularly in a single dose. If ceftriaxone is unavailable, cefixime 400 mg orally can be used as an alternative 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 the same regimen for pregnant patients. Quinolones and tetracyclines should be avoided in pregnancy. Contracting bacterial vaginosis during pregnancy carries a risk of premature rupture of membranes, preterm labor, and chorioamnionitis; pregnant women should be encouraged to seek follow-up care with a gynecologist 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, with approximately 5% of all sexual assault victims becoming 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. The two most common oral emergency contraceptives are Plan B, containing levonorgestrel, and Ella, with ulipristal acetate. Products may be taken up to 5 days after sexual contact, but ideally within 72 hours. Common side effects include nausea, vomiting, and vaginal spotting. The failure rate with Plan B is less than 2%. A copper intrauterine device (IUD) can also be placed as emergency contraception up to 5 days after the event. If a dedicated emergency contraceptive product is not available, a levonorgestrel-containing oral contraceptive pill may be utilized.

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 sometimes has a history of repetitive assaults. In addition to documenting signs of acute trauma, the examiner should look for signs of previous trauma, such as healed hymenal tears/transections, posterior fossa or fossa navicularis lacerations, and healed anal injuries. It is important to look for vaginal discharge, concerning for vulvovaginitis or a foreign body. The anogenital examination should take into account the disclosure of events. In children, the prepubertal vaginal introitus and hymen are exquisitely sensitive, and any contact (e.g., speculum or vaginal swabs) should not be attempted in an unsedated child. A speculum examination is only necessary if there is concern for active vaginal vault bleeding, and is best supervised by a gynecologist with a physician specializing in child abuse. The child should be protected from further abuse by immediate referral to the appropriate social service agency.

16. Should pediatric patients be given prophylactic antibiotics?

Prophylactic antibiotics are not always indicated for prepubertal children who have been sexually abused. The baseline infection rate in children is significantly lower than in adults, and the presence of an STD (including human papillomavirus [HPV] and herpes simplex virus [HSV]) in a child is highly suggestive of abuse. In children, chlamydial and gonorrhea nucleic amplification testing on the urine is better tolerated than vaginal specimens. HIV prophylaxis should be considered based on level of risk and in consultation with a pediatric infectious disease physician.

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, 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 after 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?

Provide counseling regarding transmission risks and offer nonoccupational postexposure prophylaxis (nPEP).

20. What is nPEP?

nPEP is 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 men who have sex with men)? Studies in prison populations reveal that HIV infection rates are higher in male sexual assailants than in the general male population (1% versus 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 after 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 U.S. Food and Drug Administration (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.

A 3- to 5-day starter pack is recommended, with a follow-up visit scheduled to review the results of HIV testing, 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 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 STDs.
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 postassault counseling is critical.

WEBSITES

Antiretroviral postexposure prophylaxis: www.cdc.gov/hiv/basics/pep.html; accessed 2-20-15.
Facts about sexual assault: www.rainn.org/statistics; accessed 2-20-15.
National Crime Victimization Survey 2014: http://ovc.ncjrs.gov/ncvrw2014/pdf/StatisticalOverviews.pdf; accessed 2-20-15.
Centers for Disease Control and Prevention: 2015 sexually transmitted disease treatment guidelines. Available at www.cdc.gov/std/tg2015/sexual-assault.htm; accessed 7-31-15.
Sexual violence fact sheet: www.cdc.gov/ncipc/factsheets/svfacts.htm; accessed 2-20-15.

Acknowledgment

The editors gratefully acknowledge the contributions of Kim M. Feldhaus, Katherine M. Bakes, and Bernadine L. Mellinger, authors of this chapter in the previous editions.

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QUESTIONS

1. Should empiric antibiotic treatment be recommended for all adult sexual assault patients?
   a. Yes, because these patients have a poor history of seeking follow-up care.
   b. No, rates of infection are so low it is not necessary.
   c. No, rates are high, but evidence of disease should first occur.
   d. Maybe, it depends on the situation.
   The correct answer is *a*.
2. Are routine drug and alcohol testing recommended for all patients of sexual assault?
   a. Yes, all patients should be tested.
   b. No, do not test unless the patient is a known recreational user.
   c. No, they should never be sent.
   d. If there is concern for DFSA, they should be sent.
   The correct answer is *d*.
3. What does toluidine blue dye do?
   a. Toluidine blue dye does not reveal injury that is not visible to the naked eye but highlights genital injuries, such as tears or abrasions.
   b. Toluidine blue dye reveals injury that is not visible to the naked eye.
   c. Toluidine dye identifies infected tissue.
   d. Toluidine dye reveals semen.
   The correct answer is *a*.

SPONTANEOUS ABORTION, ECTOPIC PREGNANCY, AND VAGINAL BLEEDING

CHAPTER 79

Brandon H. Backlund, MD, FACEP

1. What are the important causes to consider in the ED evaluation of first-trimester vaginal bleeding or pain?

- Spontaneous abortion
- Ectopic pregnancy (EP)
- Gestational trophoblastic disease (molar pregnancy)
- Subchorionic hemorrhage (SH)
- Vaginal or cervical trauma
- Consider nongenital (e.g., urinary or gastrointestinal) sources of bleeding

KEY POINTS: APPROACH TO THE PATIENT WITH A POSITIVE PREGNANCY TEST AND FIRST-TRIMESTER VAGINAL BLEEDING OR PAIN

- Recognize immediately that this clinical scenario may represent EP. These patients are at risk for precipitous hemodynamic deterioration and should be approached with a high index of suspicion.
- Establish appropriate intravenous (IV) access. Two large-bore (18-gauge or larger) 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.
- Establish maternal rhesus (Rh) type to determine the need for Rh immunoglobulin (RhoGAM).
- Quantitative serum β-human chorionic gonadotropin (β-HCG) assay is helpful in assessing ectopic risk and coordinating follow-up care.
- Treat hypotension or tachycardia with IV fluids or transfusion of blood products, as indicated by patient condition.
- Perform a speculum and bimanual 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 active bleeding, remove any visualized tissue using gentle traction with ring forceps; this may help abate ongoing bleeding.
- Determine whether an intrauterine pregnancy (IUP) has been previously documented by ultrasound. If no IUP has been documented, ultrasound should be performed to evaluate for a possible EP. Unstable patients should not be allowed to leave the ED for diagnostic studies.
- Consult a specialist in obstetrics and gynecology (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.

2. What is spontaneous abortion or miscarriage?

Miscarriage is spontaneous termination of IUP before achieving fetal weight or maturity compatible with survival (<20 to 22 weeks' gestation or fetal weight <500 g).

Abstract

Vaginal bleeding or pain are common presenting complaints to the ED during pregnancy. Important causes for the emergency physician to consider during the first trimester include ectopic pregnancy (EP) and spontaneous miscarriage. Important emergency diagnoses during later pregnancy include placenta previa, placental abruption, and uterine rupture.

Keywords:

pregnancy, complications, ectopic pregnancy (EP), spontaneous abortion, miscarriage, ultrasound in pregnancy, vaginal bleeding

3. State the incidence and timing of spontaneous abortion.

Ten percent to 20% of clinically recognized pregnancies less than 20 weeks' gestation miscarry; 80% of these occur in the first 12 weeks' gestation. Approximately 70% of all spontaneous abortions occur before pregnancy is clinically detected.

4. 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 completed 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.

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 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 septic abortion is 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. What is the prognosis for the pregnancy in 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' gestation and the mother has stable vital signs without fever. Successful spontaneous abortion occurs in 91% of incomplete miscarriages and 26% with missed abortions.

10. 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. Radiation therapy, however, does increase the incidence of spontaneous abortion.

11. What factors are associated with spontaneous abortion and/or fetal abnormalities?

During the first 4 to 8 weeks' gestation, the most important factor is chromosomal abnormalities causing abnormal development of the zygote, seen in 50% to 60% of spontaneous abortions. Later in the first trimester, factors such as isolated chromosomal abnormalities, maternal factors (such as insufficient progesterone production, or use of alcohol, cocaine, tobacco, or nonsteroidal antiinflammatory drugs [NSAIDs]), and structural uterine abnormalities, become important. Other factors include:

- 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). This should not be prescribed to pregnant women or women planning to become pregnant.
- Increased maternal parity and advanced maternal and paternal age. The incidence increases from 12% in women younger than 20 to 26 years to 40% in women age 40 years.
- 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 uterine developmental abnormalities, including müllerian duct malformation or fusion, and septate, bicornuate, or unicornuate uterus

12. 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.

13. Describe 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.

14. Name the drug used to prevent Rh immunization.

RhoGAM; any pregnant woman who is experiencing vaginal bleeding must have an Rh type checked. If she is Rh negative and the fetus is less than 12 weeks' gestation, she should receive a minidose of RhoGAM 50 μg intramuscularly. If she is Rh negative and the fetus is greater than 12 weeks' gestation, the full dose of RhoGAM, 300 μg, should be given. Some sources recommend giving the 300 μg dose to all patients regardless of fetal gestational age.

15. 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 β-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, to minimize the risk of introducing infection.

16. 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 the result of spontaneous chromosomal abnormalities and not the patient's own actions.

17. What is an 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%), intraabdominally (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 coming to EDs have an EP diagnosed. Patients with EP may have abdominal pain, syncope, 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.

Table 79-1. Odds Ratios 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)

Modified from data in Ankum WM, Mol BWJ, Van Der Veen F, et al: Risk factors for ectopic pregnancy: a meta-analysis. *Fertil Steril* 65:1093–1099, 1996.
DES, Diethylstilbestrol.

18. 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 79-1.

19. Define heterotopic pregnancy. What is the main risk factor for this condition, and what is its incidence?

A heterotopic pregnancy is 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, with natural conception, 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 4000 or higher. The incidence is much higher among patients undergoing fertility treatments, occurring with an incidence of 1 in 100 or higher in this population.

20. 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 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.

21. 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.

22. 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

23. Why are corpus luteum cysts commonly 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' gestation and may become cystic, growing to 5 cm in diameter or more. Cyst rupture can occur in the first trimester, presenting in a patient in early pregnancy as sudden pain, unilateral peritoneal findings, adnexal tenderness, and perhaps a mass.

24. 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' gestational age. 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 β-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.

25. Describe the role of bedside ultrasonography in the ED evaluation of the patient with first-trimester complaints.

Emergency physician–performed point-of-care ultrasound (POCUS) has become an increasingly common modality in the evaluation of patients with complaints of pain or bleeding in the first trimester. When used appropriately, POCUS can more rapidly confirm the diagnosis of IUP, and has been shown to decrease the ED length of stay, as compared with ultrasound performed in the radiology department. In this setting, the goal of bedside ultrasonography should not be to rule out EP, but rather to rule in IUP. In the absence of risk factors for, or signs and symptoms suggestive of, heterotopic pregnancy, definitive identification of IUP by POCUS makes the diagnosis of EP unlikely. It can also be used to assess the abdomen for the presence of free fluid, suggestive of hemoperitoneum when concern for ruptured EP exists.

26. 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 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, a poorly-defined fluid collection within the endometrial cavity that may be seen with an EP.
- Yolk sac: Seen at about 5 weeks' gestational age 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' gestational age by transvaginal ultrasound. Fetal cardiac activity is generally recognizable by about 6 to 7 weeks.

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 1000 and 2000 mIU/mL, depending on institutional protocols. For transabdominal ultrasound, the discriminatory zone is generally set around 6500 mIU/mL. 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 β-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

Table 79-2. 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	Empty uterus
Ectopic fetal pole	Nonspecific fluid collections
	Echogenic material
	Abnormal gestational sac

Modified from Dart RG: Role of pelvic ultrasonography in evaluation of symptomatic first trimester pregnancy. *Ann Emerg Med* 33:310–320, 1999.
EP, Ectopic pregnancy; *IUP,* intrauterine pregnancy.
*Complex mass most suggestive of EP, but a cyst also can be seen with EP.

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 β-HCG is above the discriminatory zone or rising on serial measurements. A rapidly falling β-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 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 79-2.

31. What patients with EP 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 β-HCG interpretation, or specialist consultation. Stable patients with indeterminate ultrasound results (rule out EP) may be monitored on an outpatient basis. Expectant management or chemotherapy for women with few symptoms and low hormonal levels should be determined in consultation with an obstetrician. The role of the ED physician is to consider the diagnosis, make every effort to exclude or confirm 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.

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 monitored closely. Failure rates are higher when the β-HCG is greater than 5000. Patients commonly have significant pain with or without peritoneal signs several days after treatment with methotrexate. Abnormal vital signs, a decreasing hematocrit level, or diffuse peritoneal signs are indications of EP rupture. The decision to use this medication should be made in by an OB/GYN specialist.

KEY POINTS: CANDIDATES FOR MEDICAL TREATMENT OF EP WITH METHOTREXATE

1. Hemodynamically stable
2. No evidence of ectopic rupture
3. The ability and willingness to comply with posttreatment monitoring
4. Ectopic gestational sac < 3.5 cm*
5. No fetal cardiac activity on ultrasonographic examination*

Modified from American College of Obstetricians and Gynecologists: ACOG Practice Bulletin No. 94: Medical management of ectopic pregnancy. *Obstet Gynecol* 111:1479–1485, 2008.
*Sac size >3.5 cm or presence of cardiac activity are relative, not absolute, contraindications.

33. What are contraindications to methotrexate therapy for EP?

- 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. What is gestational trophoblastic disease?

Gestational trophoblastic disease, also known as *molar pregnancy,* is a neoplasm that develops in placental trophoblastic cells. Signs and symptoms include painless first- or second-trimester vaginal bleeding, hyperemesis, hypertension that develops before the third trimester, and uterine size on bimanual examination that is larger than expected for gestational age. Serum β-HCG measurements are commonly significantly higher than predicted for gestational age based on time since last menstrual period. Ultrasound shows a "snowstorm" appearance, with hypoechoic areas scattered throughout a hyperechoic background within the uterus. Obstetric consultation should be obtained when this condition is diagnosed, because uterine evacuation is required, and the possibility of malignancy exists.

35. What is SH?

SH is bleeding that occurs between the chorionic layer and the uterine wall. It is the most common sonographic abnormality seen with a live embryo. The cause is unclear, because it may develop spontaneously, but may sometimes be seen after blunt trauma. Large SH may increase the risk of miscarriage, but small ones are not thought to significantly affect the course of pregnancy.

36. 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% to 20% present without vaginal bleeding)
- *Uterine rupture: 0.05% of pregnancies
- Marginal sinus rupture
- Bloody show
- Local trauma
- Cervical polyps and lesions

37. What is placenta previa?

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.

38. How is placenta previa diagnosed?

It may be diagnosed incidentally early in pregnancy during routine ultrasound, and if asymptomatic, may be followed with serial ultrasound studies until delivery, spontaneously resolving in up to 90% of cases diagnosed before 20 weeks' gestational age. In the ED, placenta previa should be suspected when a pregnant patient in the second half of pregnancy has 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.

39. How is placenta previa treated?

Early consultation with an OB/GYN specialist should be obtained when the diagnosis is suspected. Administer supplemental oxygen, obtain two large-bore IV lines, and perform maternal cardiopulmonary and fetal monitoring. Obtain a CBC, hemoglobin level, or hematocrit level, 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 placental manipulation, do not perform a pelvic examination if the diagnosis is known. 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.

40. 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.

41. Describe the treatment of placental abruption.

Immediately consult a specialist in OB/GYN. Start two large-bore IV lines, and administer oxygen. Monitor fetal heart tones and the maternal vital signs. Obtain a CBC, hemoglobin level, or hematocrit level; type and screen blood for anticipated transfusion; and perform 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.

42. What is uterine rupture, and why is it dangerous?

Uterine rupture is a grave complication of late pregnancy in which the uterus ruptures, usually during contractions. It can produce massive, life-threatening intraabdominal hemorrhage. The maternal mortality rate is 8%, and fetal mortality rate 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 cesarean section or other uterine surgery.

43. What is the treatment of uterine rupture?

Start two large-bore IV lines, administer oxygen, and support respiration and hemodynamics as necessary. Emergent OB/GYN consultation is required, because the treatment is emergent laparotomy and hysterectomy. Ultrasound may be useful in selected cases to distinguish uterine rupture from placental abruption.

44. 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.

Acknowledgment

The editors gratefully acknowledge the contributions of Dane M. Chapman, MD, author of this chapter in the previous edition.

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QUESTIONS

1. Which of the following is not a risk factor for EP?
   a. Multiple sexual partners
   b. Prior sexually transmitted infection
   c. Prior EP
   d. Genital piercing
   The correct answer is *d*.
2. When should the yolk sac be visualized by transvaginal ultrasound?
   a. 5 weeks' gestation
   b. 4 weeks' gestation
   c. 6 weeks' gestation
   d. 8 weeks' gestation
   The correct answer is *a*.
3. Which of the following would exclude a patient from methotrexate administration in the setting of a spontaneous abortion?
   a. Hemodynamically unstable
   b. No evidence of ectopic rupture
   c. The ability and willingness to comply with posttreatment monitoring and no fetal cardiac activity on ultrasound
   d. Ectopic gestational sac <3.5 cm
   The correct answer is *a*.

CHAPTER 80

THIRD-TRIMESTER COMPLICATIONS AND DELIVERY

Deborah Vinton, MD

1. What are the major hypertensive disorders in pregnancy?

- Chronic hypertension
- Gestational hypertension
- Preeclampsia
- Preeclampsia superimposed on chronic hypertension

2. What is preeclampsia?

Preeclampsia is a condition that occurs after 20 weeks' gestation. The condition is characterized by new-onset high blood pressure (BP) with either proteinuria or end-organ dysfunction because of widespread vascular endothelial malfunction and vasospasm. The disease spectrum can range from mild to severe.

3. What is gestational hypertension, and how does it differ from chronic hypertension?

Gestational hypertension is 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 who does not have any other signs or symptoms of preeclampsia. BP returns to normal less than 12 weeks' postpartum. Chronic hypertension predates a patient's pregnancy and is detected before 20 weeks' gestation

4. Which conditions must be present to diagnose preeclampsia?

- Hypertension: New-onset systolic BP greater than 140 mm Hg or a diastolic BP greater than 90 mm Hg on two occasions at least 4 hours apart, or a systolic BP greater than 160 mm Hg and a diastolic BP greater than 110 mm Hg on a single occasion. In a woman who has preexisting hypertension, the systolic BP has increased by 30 mm Hg or the diastolic BP by 15 mm Hg.

  And one of the following:
- Proteinuria: Excretion of 300 mg or more of protein in the urine in a 24-hour period
- Thrombocytopenia: Less than 100,000 platelets/μL
- New renal insufficiency: Elevated serum creatinine greater than 1.1 mg/dL in a patient without renal disease
- Impaired liver function: Elevated transaminases at least twice normal concentrations
- Pulmonary edema
- Cerebral or visual symptoms

Note: The diagnosis of preeclampsia previously required the presence of proteinuria to meet criteria for the condition. Although proteinuria in the setting of hypertension can still be used to make the diagnosis, it is no longer required if other end-organ dysfunction is present.

5. What are the diagnostic criteria for severe preeclampsia?

The presence of one or more of the following constitutes severe preeclampsia:

- Systolic BP greater than 160 mm Hg or diastolic BP greater than 110 on two occasions 4 hours apart
- Pulmonary edema
- Thrombocytopenia (<100,000 platelets/μL)
- Symptoms of central nervous system (CNS) dysfunction, such as severe headache, vision changes, or altered mental status
- Transaminase concentration greater than twice-normal level, epigastria, or right upper quadrant pain
- Progressive renal insufficiency with creatinine greater than 1.1 or doubling of creatinine

Abstract

This chapter discusses third-trimester complications and delivery in the ED.

Keywords:

preeclampsia, eclampsia, hemolysis with elevated liver enzymes and low platelets (HELLP), labor and delivery, postpartum hemorrhage (PPH), shoulder dystocia

6. How is a diagnosis of preeclampsia superimposed on chronic hypertension made?

This diagnosis is made in a patient who has hypertension recognized in early pregnancy, who then has a sudden onset of proteinuria, increased hypertension, or new onset of signs and symptoms of preeclampsia, such as thrombocytopenia, right upper quadrant pain, or new renal insufficiency after 20 weeks' gestation.

7. What causes preeclampsia?

The exact pathophysiology is unknown. Research suggests there may be incomplete invasion of cytotrophoblasts into the uterine spiral arteries, resulting in placental vasculature abnormalities. This then leads to placental hypoperfusion, hypoxia, and ischemia. The placenta then releases inflammatory substances into the maternal circulation, resulting in widespread endothelial dysfunction and end-organ damage.

8. What are the risk factors for preeclampsia?

Preeclampsia is primarily a complication of first pregnancies. Other risk factors include a personal or family history of preeclampsia, conception before age 20 years, multifetal 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 antiphospholipid antibody syndrome, insulin-dependent diabetes, connective tissue disease, renal disease, and hypertension.

9. How common is preeclampsia?

Worldwide, 5% to 18% of pregnancies are complicated by preeclampsia. In the United States, the prevalence is between 3% to 4%.

10. What is the definitive treatment for preeclampsia?

Delivery of the fetus is definitive treatment for preeclampsia. The decision to initiate delivery is based on gestational age, severity of disease, and the well-being of the mother and fetus.

11. When is immediate delivery indicated?

Immediate delivery is indicated in any patient with greater than 34 weeks' gestation who meets criteria for severe preeclampsia, or when maternal or fetal condition is considered to be unstable regardless of gestational age. For mild disease, expectant management is appropriate up to 37 weeks, at which time delivery is recommended.

12. What is the treatment for preeclampsia in the ED?

Magnesium sulfate should be administered immediately. Magnesium slows neuromuscular conduction and decreases CNS irritability, but does not lower BP. Therefore antihypertensives should be used in consultation with an obstetrics provider for sustained systolic BP greater than 160 mm Hg or sustained diastolic BP greater than 110 mm Hg. For patients between 24 and 34 weeks' gestation who come to the ED, corticosteroids are administered to enhance fetal lung maturity.

13. Which antihypertensive medications can be used?

- Hydralazine 5 to 10 mg intravenously (IV) every 20 minutes until desired effect to a maximum dose of 30 mg
- Labetalol 20 mg IV; 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

14. Which antihypertensive medications should be avoided in pregnancy?

- Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and direct renin inhibitors should be avoided because of the association with fetal renal abnormalities.
- Sodium nitroprusside is also best avoided, because cyanide poisoning of the fetus can occur, It is only to be used for severe refractory hypertensive emergency when alternative medications have been ineffective.

15. What are the findings of magnesium toxicity, and how should the patient be monitored?

Patients with magnesium toxicity may demonstrate loss of patellar reflexes, somnolence, slurred speech, and flushing. These findings are typically present when serum magnesium levels reach 8 to

12 mg/dL. 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. In patients receiving IV magnesium, patellar tendon reflexes and respiratory rate should be checked every hour and magnesium levels should be drawn every 4 to 6 hours. If signs of magnesium toxicity develop, 1 g of calcium gluconate should be administered IV.

16. 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).

17. What complications to the fetus can occur?

Fetal growth restriction and oligohydramnios with preeclampsia.

18. Is there a way to prevent preeclampsia?

No proven definitive method of prevention has been identified. Some studies have described the use of low-dose aspirin to prevent preeclampsia in patients at moderate to high risk of developing preeclampsia. The use of calcium fish oil or vitamins C plus E does not appear to prevent the onset of preeclampsia.

19. What is eclampsia?

Eclampsia is the new onset of generalized tonic-clonic seizure or unexplained coma in a patient with signs and symptoms of preeclampsia and without other neurologic conditions. Symptoms may occur any time after the second trimester or in the postpartum period. Patients may report persistent frontal headache, visual disturbances, right upper quadrant pain, or altered mental status in the hours preceding the onset of seizure activity. Eclampsia occurs in 2% to 3% of patients with severe preeclampsia.

20. What is the treatment for eclampsia?

Immediate stabilization of airway, breathing, and circulation (ABCs) is necessary, with the administration of oxygen, placement of IV lines, and monitoring of mother and fetus. The patient should be rolled onto her left side to prevent compression of vena cava. Administer magnesium sulfate 4 to 6 g IV over 15 to 20 minutes. This bolus should then be followed by a magnesium drip run at 2 g/h. Administration of a benzodiazepine, such as diazepam or lorazepam, may be considered if the patient has a second seizure. After magnesium, BP should be controlled with hydralazine or labetalol. There is no role for mannitol in the treatment of eclampsia. Similar to preeclampsia, the definitive treatment of eclampsia is delivery. Prompt delivery is indicated, because eclampsia is considered to be an absolute contraindication for expectant management of pregnancy.

21. What is the most common cause of death in a patient with preeclampsia-eclampsia?

CNS complications, such as cerebral edema, hemorrhages, and infarctions, as well as pulmonary edema, are the most common causes of maternal death.

22. 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 postpartum.

23. Are there atypical presentations of preeclampsia or eclampsia?

Yes, preeclampsia or eclampsia can occur at less than 20 weeks' gestation, primarily in the setting of a molar or partial molar pregnancy. However, hypertension and proteinuria at less than 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.

24. What is HELLP syndrome?

HELLP is an acronym referring to a syndrome characterized by **h**emolysis, **e**levated **l**iver enzymes, and a **l**ow **p**latelet count. HELLP syndrome may represent a severe form of preeclampsia. Patients with HELLP syndrome most typically have epigastric or right upper abdominal pain, nausea, and vomiting. Some patients may also complain of malaise, headache, or visual disturbances. Others may atypically have nonspecific symptoms, which can be mistaken for a viral syndrome or viral hepatitis.

25. How is HELLP syndrome diagnosed?

- Microangiopathic hemolytic anemia: Evidence of schistocytes on blood smear or other findings indicative of hemolysis, such as elevated indirect bilirubin or low serum haptoglobin level
- Thrombocytopenia of less than 100,000 platelets/μL
- Total bilirubin greater than 1.2 mL/dL
- Serum aspartate transaminase (AST) greater than 70 IU

26. How is HELLP syndrome treated?

In the patient greater than 34 weeks' gestation or a patient with severe maternal or fetal distress, 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. Antihypertensive medications similar to those used in preeclampsia (labetalol, hydralazine, or nifedipine) should be administered to treat severe hypertension. Magnesium should also be administered.

For gestations of less than 27 weeks, conservative management can be considered for 48 to 72 hours of evaluation, along with corticosteroid administration.

27. What are the complications of HELLP syndrome?

The most common complication of HELLP syndrome is disseminated intravascular coagulation (DIC). Additional complications include abruptio placentae, severe postpartum hemorrhage (PPH), rupture of a subcapsular liver hematoma, intracranial hemorrhage or infarction, pulmonary edema, and acute renal failure.

28. What are the maternal and fetal mortality rates associated with HELLP?

- The maternal mortality rate 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% to 20%, with improved survival after 32 weeks' gestation. The most common causes of neonatal death with HELLP syndrome are abruptio placentae, placental insufficiency, and prematurity.

KEY POINTS: PREECLAMPSIA AND ECLAMPSIA

1. Preeclampsia is characterized by new-onset hypertension with signs of end-organ dysfunction that can present any time from 20 weeks' gestation to 4 weeks' postpartum.
2. Delivery of the fetus is the definitive treatment for preeclampsia, eclampsia, and HELLP syndrome.
3. Hydralazine or labetalol in addition to magnesium sulfate are the first-line agents to treat preeclampsia and eclampsia in the ED.

29. What do I need to do to stabilize a pregnant patient brought into the ED?

The unplanned delivery of babies will occur in the ED. Thankfully, however, labor and delivery is a natural process, which usually proceeds to its conclusion with limited intervention and without difficulty. When a pregnant patient arrives in the ED, after vital signs and the chief complaint are obtained, immediately establish IV access. In general, pregnant patients should be transported in the left lateral recumbent position to reduce pressure on the vena cava. Help should be sought from obstetrics and pediatrics specialists upon patient arrival if there is any concern that a delivery may occur in the ED.

30. 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 or prior pregnancies, including gestational diabetes, gestation hypertension, or preeclampsia. Clarify whether she is only carrying one baby presently. In addition to identifying her current medications and allergies, ask about illicit drug use. Find out where she is getting her prenatal care and the name of her doctor or midwife.

31. 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's condition 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. If time permits, a bedside ultrasonography can be helpful in determining fetal position, number of fetuses, presence of cardiac activity, and quantity of amniotic fluid.

32. How do I 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. Do not perform a digital pelvic examination in any patient who has vaginal bleeding in the late second or third trimester, because this may exacerbate placenta previa. Immediate obstetric consultation should be initiated in these patients instead, because a digital examination is contraindicated.

33. 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 the 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 the placenta

34. How can I determine whether a delivery is imminent?

This can be difficult. Signs of imminent delivery include expulsion of the mucus plug (often referred to as *bloody show*), breakage of the amniotic sac, or 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. Some patients, however, may not appear to be in any distress just before a delivery is to occur. If digital examination of the patient reveals the cervix is 6 cm or more dilated, delivery may occur before or during transport to labor and delivery.

35. 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 and perineum to prevent sudden expulsion and to allow gradual stretching of the perineum. As the head emerges, suction the baby's nose and mouth. Apply very gentle guidance of the head downward to deliver the anterior shoulder; then provide very gentle traction upward to deliver the posterior shoulder. Once both shoulders have been delivered, the remainder of the delivery will usually occur very quickly, so keep a good grip because the baby may be slippery. When the feet are out, rotate the baby 180 degrees and again suction the nose and mouth. Double clamp the umbilical cord 7 to 10 cm from the baby and cut the cord between the clamps.

36. 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.

37. What is shoulder dystocia?

Shoulder dystocia occurs when the baby's anterior or posterior shoulder becomes impacted behind the pubic symphysis or sacral promontory. It is a clinical diagnosis that should be suspected when the fetal head retracts into the perineum after expulsion (the turtle sign) and routine, gentle, downward traction of the fetal head fails to deliver the anterior shoulder.

38. What maneuvers are available to resolve shoulder dystocia during delivery?

The McRoberts maneuver should be attempted first, because it is noninvasive and often successful. To perform this maneuver, have two assistants flex each of the mother's thighs back against her abdomen and have the patient continue to push. Other options include applying suprapubic

pressure, delivering the posterior arm, or performing the Woods screw maneuver, in which the fetus is rotated 180 degrees by exerting pressure on the anterior clavicular surface of the posterior shoulder. Excessive neck rotation/traction and fundal pressure should be avoided, because this can lead to increased shoulder impaction, brachial plexus injuries, or uterine rupture.

39. If the umbilical cord is wrapped around the neck during a delivery, what should I do?

If the cord is wrapped around the baby's 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.

40. The placenta has not delivered. 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, because this can cause the uterus to involute. 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).

41. How do I manage a breech presentation?

The major risk of breech delivery is entrapment of the head; it occurs in approximately 4% of deliveries. If the fetus is breech, call for immediate obstetric and pediatric assistance. If the delivery is imminent, fetal rotation will be needed for delivery of the fetal legs and arms. If the head becomes entrapped, apply suprapubic pressure and insert fingers to draw the fetal chin down to the chest.

42. How do I recognize PPH?

PPH can be defined as greater than 500 mL blood loss after a vaginal birth or excessive bleeding that causes the patient to be symptomatic, including experiencing pallor, lightheadedness, palpitations, confusion, syncope, or other symptoms associated with blood loss.

43. What are the most common causes of PPH?

Uterine atony is the most common cause, contributing to 80% of cases of PPH. Atony is most common in the setting of uterine overdistention secondary to multiple gestations or polyhydramnios. Additionally, atony may occur in the setting of infection, prolonged or induced labor, uterine inversion, or retained placenta. Lacerations caused by trauma or underlying coagulation defects, such as von Willebrand disease, can also contribute to PPH.

44. How is PPH managed?

Establish large-bore IV access and place the patient on oxygen. Begin fluid resuscitation with crystalloid. If the patient has persistent hypotension despite 2 to 3 L of crystalloid, administer blood products, beginning with 2 units of packed red blood cells. While fluids are infusing, if uterine atony is suspected, perform firm uterine massage and compression. Inspect the vagina and cervix for obvious lacerations to repair. Evacuate any retained products of conception. If uterine atony continues despite massage, administer uterotonic drugs, beginning with oxytocin 10 to 40 MU/min. Adjust the infusion rate to maintain uterine contraction. Misoprostol (400 μg sublingually) may be given if other uterotonic medications are not available.

KEY POINTS: CHILDBIRTH IN THE ED

1. Spontaneous term labor and delivery is a natural process, which usually proceeds to its conclusion without difficulty. However, pediatric and obstetric specialists should be notified immediately if imminent delivery is suspected.
2. It is essential to provide oxygen and have IV access during the delivery process in the ED setting.
3. During delivery, the health care provider should not attempt to speed up the process by pulling or pushing on the baby; however, if shoulder dystocia occurs during delivery, the McRoberts maneuver should be performed first.

Acknowledgment

The editors gratefully acknowledge the contributions of Dr. Gina Soriya for her contributions to this chapter in the previous edition.

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QUESTIONS

1. A 32-year-old female patient G2P1 at 30 weeks' gestation has had headache and nausea for 2 days with vision that "got funny" when standing up. She has had no prenatal care. Her BP is 180/110, with a pulse of 108. She appears uncomfortable, and there is moderate right upper quadrant tenderness present. Which medication should be used to treat her BP?
   a. Nitroprusside
   b. Nitroglycerin
   c. Hydralazine
   d. Lisinopril
   The correct answer is *c*.
2. A 34-year-old female has just delivered a full-term infant boy in the ED after an uncomplicated pregnancy. After delivering the placenta, she reports feeling lightheaded and short of breath. Shortly thereafter, a large amount of vaginal bleeding is noted. Her uterus is noted to feel boggy on palpation. How should her condition be managed?
   a. Emergent hysterectomy
   b. 1 L of crystalloid IV fluid, 4 mg of IV ondansetron (Zofran), and supplemental oxygen
   c. Firm uterine massage followed by oxytocin infusion
   d. Two units of packed red blood cells and terbutaline infusion
   The correct answer is *c*.
3. A 27-year-old female at 38 weeks' gestation with a history of diabetes comes to the ED in active labor. During the delivery, the baby's head has delivered, but it is noted to retract intermittently into the perineum. What should be done to assist with delivery?
   a. Apply firm fundal pressure.
   b. Continue to provide downward traction on the fetal head until the anterior shoulder delivers.
   c. Push the fetal head up into the vaginal vault to prevent further descent.
   d. Flex the patient's thighs back against her abdomen as she continues to push.
   The correct answer is *d*.

MULTIPLE TRAUMA

Peter Rosen, MD

CHAPTER 81

1. What is multiple trauma?

Multiple trauma is significant injury to more than one major body system or organ.

2. Describe mechanism of injury.

Mechanism of injury refers to the events and conditions that lead to traumatic injuries. Significant mechanism of injury is associated with a higher likelihood of multiple trauma. Less obvious mechanisms are of greater concern, particularly with increasing age or preexisting disease. 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.

3. Are there any factors that should raise concern about the impact of the mechanism of injury?

One of the parameters that increases the apparent severity of a mechanism of injury is the age of the patient. The geriatric patient, for many reasons, has an inability to tolerate major trauma. A 70-year-old patient with ankylosing spondylitis is much less able to tolerate blunt trauma to the spinal column or trauma to the pelvis than is a healthy younger person. Even a trivial mechanism of injury can produce significant damage in patients with prior disease or in patients of advanced age. It is prudent to increase the concern for all levels of injury in anyone older than 60 years.

4. Give some 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 to 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 team 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 the field treatment.
- Obtain vital signs while the patient is being undressed.
- Assess the patient's 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 often necessary until the spine can be cleared, all movement should be avoided. Therefore to obtain complete visualization of the patient 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.

Abstract

Multiple organ trauma management has certainly evolved from performing surgery in almost all patients, to a selective but safe approach of careful observation. Key to this are the techniques of bedside ultrasound imaging studies, improved radiologic imaging, and nonsurgical interventions, such as embolization of pelvic fractures. The management of the airway in the trauma patient continues to evolve again with the assistance of technologies such as video laryngoscopy. What has not changed, however, is the importance of the mechanism of injury in sorting out which patients are most likely have internal injury; the need for surgical airway, especially in the presence of blunt impact to the trachea; and the need for aggressive prophylactic airway management in the presence of any injury that has the potential to distort the anatomy of the airway.

Keywords:

multiple trauma, trauma resuscitation, trauma ultrasound, trauma airway management, surgical airway, tranexamic acid (TXA), mechanism of injury

7. What are the ABCs (and D) of trauma?

- **A**irway
- **B**reathing
- **C**irculation
- **D**isability

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, teeth, emesis, or foreign debris). The trauma team captain must determine whether the patient needs active airway management and verify that supplemental oxygen is being administered continuously to all patients who do not require immediate intubation of the airway.

9. When do I need to manage the airway?

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

10. Is there any role for ketamine in the management of trauma patients?

For many years ketamine was thought to be harmful when used 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. It does raise blood pressure, unlike most sedating agents; it 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.

11. What is the role of surgical airway management in the ED?

Whereas there are fewer indications for surgical airway, even in the trauma patient, there will still be some cases in which this may be necessary. Central facial destruction is such an instance. Even in these cases, however, it may be possible to intubate the patient's airway, with or without video laryngoscopy. Displacing the tongue anteriorly by grasping it with a towel clip, and pulling forward, may reveal the glottis, and enable an oral intubation and the avoidance of having to do a cricothyrotomy.

12. 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. Holding a gloved hand over the nose and mouth enables one to count respirations and estimate the tidal volume. The chest should be palpated gently for subcutaneous air and bony crepitus. Oxygen saturation should be monitored continuously. The trauma team captain determines whether or not tube thoracostomies or ventilatory support is needed immediately.

13. 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 team captain determines whether additional vascular access or volume is needed, and whether blood should be administered. A focused assessment with sonography for trauma (FAST) examination should be performed on any patient with torso trauma to detect free fluid (blood) in the abdomen or perineum (see Chapter 5).

14. What is the role of ultrasound in the ED management of trauma?

Ultrasound imaging obtained at the bedside has become a standard practice in the evaluation of the multiple trauma patient. It provides immediate and accurate information about the lungs, the heart, and the abdomen. It is more accurate than plain chest radiography in demonstrating an anterior and superior pneumothorax. Unfortunately, as with the computed tomography (CT) scan, ultrasound probably reveals too many small pneumothoraces, most of which will not require intervention unless the patient is going to be mechanically ventilated with or without surgery.

It can also show fluid in the abdomen. However, when performed early in the patient's course, the initial ultrasound image may not demonstrate this; therefore serial tests are recommended. Similar to diagnostic peritoneal lavage (DPL), it will show fluid in the abdomen but not the seriousness of the organ injury that produced the fluid. This still must be determined by serial physical examinations and the ongoing monitoring of vital signs.

15. What about DPL? Is there still any role for it?

DPL, which for many decades was the principle diagnostic study for an objective evaluation of a traumatized abdomen, now has a limited role in the management of abdominal trauma (see Chapter 88).

16. 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.

17. What type of intravenous (IV) access should be established in a patient with major trauma?

At least two large-bore (16-gauge) IV 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 IV 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.

18. 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, front and back.
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.

19. What parameters should be monitored in multiple trauma victims?

Monitor vital signs, neurologic status, cardiac rhythm, oxygen saturation, and, if possible, central venous pressure and urinary output. If the airway has been managed, end-tidal carbon dioxide monitoring is also indicated. Hypothermia adversely affects outcome, and core temperature can drop rapidly when the patient is disrobed and receives large quantities of cold IV fluids. 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.

20. When should blood be administered?

O-negative (universal donor) blood should be reserved for patients who are in arrest from hypovolemic shock and in whom reasonable chances for successful resuscitation exist. O-positive blood should be used in pregnant women. If 50 mL/kg of crystalloid (up to 2 L) is infused rapidly and there is no significant improvement in the patient's circulatory status, type-specific non–cross-matched blood should be administered if available (see Chapter 4). As a result of the experience gained in the Gulf War, resuscitation using a combination of packed red blood cells, fresh-frozen plasma, and platelets is preferred over the traditional use of crystalloids for volume restoration in the hospital in the face of uncontrolled hemorrhage.

21. What is permissive hypotension?

This refers to the concept of allowing a trauma patient to remain moderately hypotensive until the source of bleeding has been controlled. Studies have shown that returning a trauma patient's blood pressure back to normal levels before obtaining hemorrhage control increases the amount of blood lost. Therefore fluid resuscitation is targeted to maintain a blood pressure of 80 to 90 mm Hg in trauma patients without brain injury and 90 to 100 mm Hg in trauma victims with associated brain trauma.

22. What can I find out about tranexamic acid (TXA) and its role in trauma care?

TXA is an antifibrinolytic agent that has been getting significant attention as a potential treatment for trauma patients. There have been two studies (CRASH-2 and MATTERS) that have shown a survival benefit when TXA is given within the first 3 hours after sustaining a major injury. However, a number of questions about TXA remain. The complication rate for deep vein thrombosis (DVT) and pulmonary embolism (PE) appears to be significant. In addition, the settings and situations in both studies are quite different from the organized emergency medical services (EMS) and trauma systems of the United States. Thus the specific role of TXA in the management of trauma in the United States has yet to be defined.

23. 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 (when repeated serially) 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 laboratory tests in multiple trauma include the following:

- Complete blood cell count
- Type and cross-match
- Electrolytes
- Urinalysis
- Blood urea nitrogen, creatinine
- Blood alcohol level as indicated
- Glucose
- Toxicology as indicated
- Prothrombin and partial thromboplastin times
- Amylase, lipase

24. What is the secondary survey?

The secondary survey is 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, complete neurologic examination and rectal examination also should be done.

The purpose of the rectal examination is to determine whether there is gross blood in the rectum, whether there is adequate sphincter tone and sensation, and whether the prostate gland is in a normal position.

25. 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. If the patient will be undergoing CT scan of these areas, the plain films can be deferred.
- 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, evaluation of the lumbar spine film should be added to the radiographic studies.

26. 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 abdominal ultrasound or abdominal CT scan 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 patient's 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 taking an anticoagulant medication, even minor head injury mandates a CT scan.

27. How are fluids managed in pediatric trauma?

Start with a bolus of 20 mL/kg of normal saline (NS) or lactated Ringer solution (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 91).

28. 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 the mother 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 signs of abruption occur, emergency cesarian section must be performed (see Chapter 90).

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QUESTIONS

1. What is the major concern with trauma in the geriatric patient?
   a. Overtriage to the trauma center
   b. Overattention to concomitant disease
   c. Heightened pain sensitivity
   d. Underestimation of the patient's response to trauma
   The correct answer is *d*. Geriatric patients are often unable to localize trauma, and because of alterations in their physiology from age, as well as from pharmacology taken for concomitant disease, such as β-blockers, may not show the seriousness of their injuries. Answer *a* is incorrect; because of underestimation of the seriousness of trauma, the geriatric patient is often undertriaged to the trauma center. Option *b* is incorrect, because concomitant diseases are often forgotten and commonly destabilized even by minor trauma; *c* is incorrect, because the geriatric patient often seems to have less pain than would be expected from the injuries that are present.
2. A 20-year-old man is shot in the chest. Select the optimal strategy.
   a. Immediate portable supine chest radiographic study
   b. Immediate surgical thoracotomy
   c. Immediate thoracostomy on the side of injury
   d. Immediate pericardiocentesis
   The correct answer is *c*. There will always be a hemothorax with a gunshot wound of the thorax. Instead of wasting time proving its presence, it is more prudent to treat and stabilize the patient. Option *a* is incorrect because it not only wastes time but is an inaccurate study and may disguise and miss a small hemopneumothorax. Option *b* is incorrect because most gunshot wounds of the chest can be managed by fluid and blood replacement and decompression of the chest with a thoracostomy tube. Only if there is persistent, massive, or recurrent hemorrhage will thoracotomy be necessary. Answer *d* is incorrect because the bullet may not have penetrated the heart. A bedside ultrasound after the placement of the thoracostomy tube may reveal pericardial fluid, but it need not be an initial step unless the patient is failing as evidenced by bradycardia, hypotension, elevated central venous pressure, and a positive ultrasound imaging study.
3. A 27-year-old man has a stab wound of the neck. His oxygen saturation is normal, and he is breathing 14 breaths/min. There is a mass on the side of the stab wound. Choose the best airway strategy.
   a. Close observation with intubation orally should the patient develop desaturation, or dyspnea
   b. Immediate cricothyrotomy with local anesthesia
   c. Immediate oral rapid-sequence intubation using video laryngoscopy
   d. Immediate transfer to the operating room for tracheostomy and neck exploration
   The correct answer is *c*. Any trauma patient who has an injury that may produce anatomic distortion of the airway should have his or her airway intubated before any destabilization occurs. There is no reason not to use rapid sequence with a videoscope, because this will produce the highest success rate. Answer *a* is incorrect, because even with the best observation, if the patient becomes destabilized from the expanding neck hematoma, it may be impossible to successfully intubate him. Choice *b* is incorrect, because it would be preferable not to enter the cervical fascia that is already showing a hematoma. A surgical airway is therefore to be avoided if at all possible. Option *d* is incorrect, because although the patient does need to have a neck exploration, the airway needs to be managed before the patient's condition destabilizes.

CHAPTER 82

MAXILLOFACIAL TRAUMA

Joshua J. Solano, MD, 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 bones, vomer, 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 to manage in these patients. Significant facial trauma may cause distortion of the airway as a result of bleeding, swelling, loose teeth, or fractures. In patients with mandibular fractures, the tongue loses its support and can occlude the airway.

3. **How should the airway be managed in patients with maxillofacial trauma?**
   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 using direct or video laryngoscopy, should be attempted first. However, airway distortion resulting from facial trauma may lead to a difficult airway situation that 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%.

4. **Which procedure is contraindicated in patients with maxillofacial trauma?**
   Nasogastric tube placement should not be performed because of the risk of inadvertent intracranial placement through a fracture in the cribriform plate. The small size and flexibility of the nasogastric tube allow it to be misdirected through such a fracture into the brain. There is also a 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 extremely low.

5. **What is a blow-out fracture, and 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. The sudden increase in 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 floor defect. Diplopia is noted by having the patient visually follow and count fingers using an upward gaze. Other physical findings include infraorbital anesthesia and enophthalmos (posterior displacement of the globe into the orbit). Patients may have tenderness or palpable step-offs at the infraorbital rim or subcutaneous emphysema secondary to a fracture into the maxillary sinus. Ophthalmologic evaluation for associated ocular trauma (globe rupture, hyphema, retinal tear or detachment, blindness), despite an initially normal visual acuity and funduscopic examination, should be considered.

6. **What is a lateral canthotomy, and when is one necessary?**
   This is a procedure that involves incising the lateral canthal ligaments of the orbit. If a patient sustains trauma to the orbit resulting in a retrobulbar hematoma, the buildup of pressure behind the globe can lead to ischemia of the optic nerve and retina and permanent blindness. This complication may occur in as little as 90 to 120 minutes after injury. Performance of a lateral canthotomy allows the pressure to be relieved.

7. **What findings indicate the need for a lateral canthotomy?**
   Patients with blunt trauma to the orbit who have proptosis, impaired extraocular movement, decreased vision, and increased intraocular pressure are candidates for lateral canthotomy.

Abstract

This chapter summarizes the approach to patients with maxillofacial trauma, including choices for diagnostic imaging, safe procedures for the patient with maxillofacial trauma, and treatment of these injuries.

Keywords:

Le Fort fracture, cerebrovascular injury, septal hematoma, frontal sinus fracture, zygoma fracture, tongue blade test, mandible fracture, temporomandibular joint dislocation, Stensen duct, ear trauma

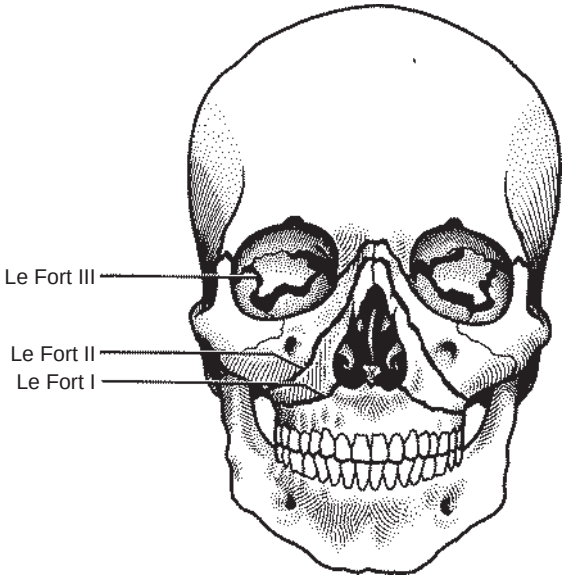

Figure 82-1. Le Fort classification of facial fractures. Le Fort I: palatofacial disjunction. Le Fort II: pyramidal disjunction. Le Fort III: craniofacial disjunction. *(From Cantrill SV: Face. In Marx JA, Hockberger RS, Walls RM, et al, editors:* Rosen's emergency medicine: concepts and clinical practice*, ed 5, St. Louis, 2002, Mosby, p 325.)*

8. What are Le Fort fractures?

The Le Fort classification is used to describe maxillary fractures (Fig. 82-1). Midface fractures can often be 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

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).

9. Is there a role for screening patients with Le Fort 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 these vascular injuries are left untreated, and there is commonly 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 tomography angiography (CTA) in patients with signs or symptoms of BCVI and patients at high risk. High-risk patients that should be screened include those with a Le Fort 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 score lower than 6, or near hanging with anoxic brain injury.

10. 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.

11. What is a septal hematoma, and 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 grapelike swelling over the nasal septum. If left undrained, it may result in septal abscess, necrosis of the nasal cartilage, and permanent saddle nose 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 to otolaryngology.

12. 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. However, reduction is associated with significant pain, and systemic analgesia should be considered. Patients should be referred to an otolaryngologist or a maxillofacial or a plastic surgeon for follow-up care in 4 to 7 days. Immediate consultation is suggested for nasal fractures with associated facial fractures, cerebrospinal fluid rhinorrhea, and sustained epistaxis.

13. 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 whether there is involvement of the anterior or posterior walls of the sinus or intracranial hemorrhage.

14. 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.

15. 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:

1. 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).
2. 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), maxillofacial CT is necessary to better define the extent of the fracture. For these fractures, admission and consultation with a plastic or maxillofacial surgeon are required.
3. 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.

16. What are the typical findings of a mandible fracture?

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.

17. What is the tongue blade test?

The tongue blade test is performed by asking the patient to bite down strongly on a tongue depressor and keep the tongue depressor clenched between the teeth. The tongue blade should be twisted by the examiner. If there is no fracture of the mandible, the examiner should be able to break the blade. In the presence of a mandible fracture, the patient opens his or her mouth because of pain from the fracture, and the tongue blade remains intact.

18. 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 site. If available, the panoramic 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 panoramic radiograph machine is unavailable, maxillofacial CT is indicated to define fracture fragments.

19. 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.

20. What is the mechanism for a temporomandibular joint dislocation, and 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 have 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 or by standing in front of the seated patient. The mandible is then pushed downward and posterior. Another approach relies on the application of rotational force on the mandibular ramus with both index and middle fingers applying clockwise force on the molars and thumbs pushing clockwise on the mental portion of the mandible.

21. 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, traditional plain radiography is used as a screening test, although in many sites maxillofacial CT is the preferred modality because of its increased sensitivity. 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 and avoiding the additional cost of the plain film studies. 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).

22. How do I recognize an injury to the Stensen duct?

The Stensen (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. 82-2). Any laceration along this pathway may involve the parotid gland, parotid duct, or buccal branch of the facial nerve. Laceration of the parotid system is 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. In addition, the buccal branch of the facial nerve travels in close proximity to the Stensen duct; injury to the nerve leads to drooping of the upper lip, which indicates a possible parotid duct injury. To assess for parotid duct patency, the parotid gland should

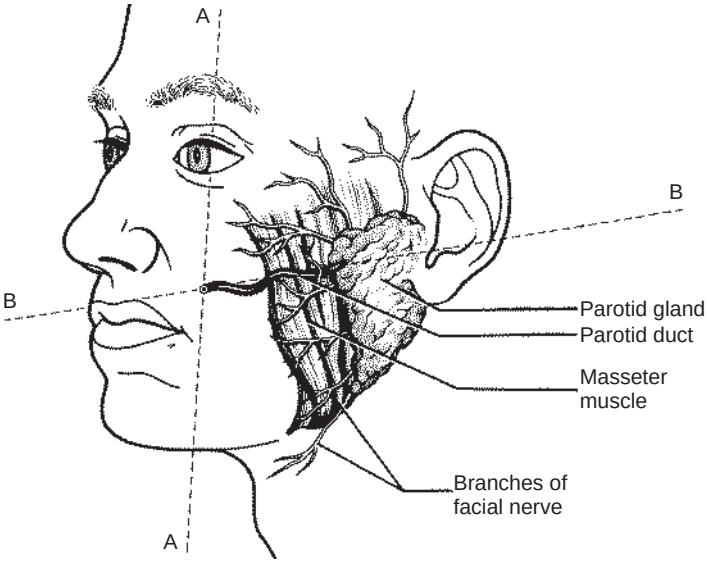

Figure 82-2. Parotid gland and parotid duct with nearby branches of the facial nerve. Line *B* demonstrates approximate course of the parotid duct from the parotid gland, entering the mouth at the junction of lines *A* and *B. (From Cantrill SV: Face. In Marx JA, Hockberger RS, Walls RM, et al, editors:* Rosen's emergency medicine: concepts and clinical practice, *ed 5, St. Louis, 2002, Mosby, p 323.)*

be milked to see if saliva is expressed from the intraoral opening of the parotid duct. Damage to the duct requires consultation with a plastic surgeon and repair over a stent.

23. 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.

24. 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 is 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.*

25. 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 canal require topical anesthesia with 4% lidocaine or local injection.

KEY POINTS: MAXILLOFACIAL TRAUMA

1. Concern for facial fracture is a contraindication to nasogastric tube placement.
2. Always make sure to check extraocular movements in patients with facial trauma.
3. CT scan has largely replaced plain films in diagnostics for maxillofacial fracture.

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

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QUESTIONS

1. A 25-year-old male comes to the ED after an altercation. He sustained multiple blows to the face and has a swollen right eye and blood from both nares. His vital signs are temperature 98.7°F (37.1°C), pulse 90 beats per minute, blood pressure 138/90, and respirations 16 breaths per minute. His physical examination reveals no other injuries. He has a Glasgow Coma Scale score of 15, pupils are equally round and reactive to light, extraocular movements are intact, and he is conversant. His cervical spine is nontender. What is the next best step in management?
   a. Assessment of visual acuity
   b. Maxillofacial CT scan
   c. Plain films of the face
   d. Brain CT
   The correct answer is *a.*
2. A 45-year-old male is intoxicated after an altercation. He sustained multiple blows to the face and has a swollen left eye with a sunken appearance and blood from both nares. His vital signs are temperature 97.4°F (36.3°C), pulse 110 beats per minute, blood pressure 133/75, and respirations 14 breaths per minute. His physical examination reveals no other injuries. He has a Glasgow Coma Scale score of 15; pupils are equally round and reactive to light; and extraocular movements are intact, except that he is unable to perform upward gaze of the left eye. His visual acuity is 20/20 in both eyes. His cervical spine is nontender. What is the next best step in management?
   a. Maxillofacial CT scan
   b. Maxillofacial surgery consultation
   c. Perform a lateral canthotomy
   d. Ophthalmology consultation
   The correct answer is *b.*
3. An 83-year-old male with atrial fibrillation on warfarin comes to the ED after he was the restrained driver involved in a motor vehicle accident at highway speed. The airbag deployed, striking him in the face. He has a swollen right eye with a proptotic appearance. His vital signs are temperature 97.8°F (36.6°C), pulse 80 beats per minute, blood pressure 175/83, and respirations 16 breaths per minute. His physical examination reveals no other injuries. He has a Glasgow Coma Scale score of 15, pupils are equally round and reactive to light, extraocular movements are intact, and visual acuity is 20/20 in the left eye but 20/200 in the right eye. His cervical spine is nontender. A focused assessment with sonography for trauma (FAST) examination is normal. What is the next best step in management?
   a. CT of the torso
   b. Maxillofacial surgery consultation
   c. Lateral canthotomy
   d. Ophthalmology consultation
   The correct answer is *c.*

CHAPTER 83

CERVICAL SPINE AND SPINAL CORD TRAUMA

Gladston R. Hackett, MD, and Robert M. McNamara, MD, FAAEM

1. **What is the annual incidence of spinal cord injury (SCI) in the United States?**
   There are approximately 12,000 new cases per year. Of these cases, the proportion of sports-related injuries have been decreasing, whereas the proportion related to falls has been increasing.

2. **Name the most common causes of SCI.**
   - Vehicular crashes (36.5%)
   - Falls (28.5%)
   - Violence, primarily gunshot wounds (14.3%)
   - Sports (9.2%)
   - Other (11.4%)

3. **What are the most common levels of injury?**
   The most common level of injury in adults is C5, followed by C4, C6, C7, T12, and L1. Overall, about half of all spine injuries are cervical injuries. In children, fractures of C1 and C2 are more common.

4. **Who gets SCIs?**
   SCI primarily affects young, healthy adults—mostly young, adult males (80.7% male predominance)—and thus is a devastating and life-altering injury. Average age of injury is 42.6 years old, with the predominant age range between 16 and 30.

5. **In patients discharged from the hospital with neurologic impairment, what percentage has paraplegia and what percentage has tetraplegia (quadriplegia)?**
   An injury to one of the seven cervical vertebrae can cause tetraplegia. People with paraplegia have injuries to the thoracic, lumbar, or sacral regions. Since the 1970s, the percentage of persons with incomplete tetraplegia has increased, whereas complete paraplegia and complete tetraplegia have decreased.
   - Incomplete tetraplegia (40.6%)
   - Incomplete paraplegia (18.7%)
   - Complete paraplegia (18.0%)
   - Complete tetraplegia (11.6%)

   Less than 1% of patients has complete recovery by discharge.

6. **If most spinal injuries do not cause neurologic injury, why should I worry?**
   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, as well as proper immobilization and handling of the patient. It is equally imperative to have good-quality diagnostic imaging, because inadequate studies and interpretation errors can lead to permanent injury.

7. **What is the financial impact of an SCI?**
   It is huge. The health care and living costs can vary greatly, depending on the severity of injury sustained and the age of the individual at the time of injury. The estimated yearly costs can range from $41,393 to $181,328. This does not include the first-year costs, which range from $340,787 to $1,044,197, depending on severity of injury, length of hospitalization and rehabilitation, education, and employment status at time of injury.

8. **Name the causes of reduced life expectancy in patients with SCI.**
   Pneumonia and septicemia

Abstract

This chapter discusses emergency management of cervical spine injuries.

Keywords:

cervical spine, C-spine, spinal cord injury (SCI), neck trauma, spine trauma, backboard, steroids for spinal cord injury, spine fracture, incomplete cord syndromes, central cord syndrome, Brown-Séquard syndrome

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 patient with Down syndrome. Osteoporosis and metastatic cancer may lead to a vertebral fracture with insignificant trauma.

10. How do I immobilize the patient with a potential spinal injury?

Traditionally, when any spinal injury has been suspected, the entire spine has been immobilized onto a long board, along with placement of a rigid collar to immobilize the cervical spine. Stabilization also included the forehead taped to the board and accessory towels or other bolsters to prevent further neck movement. It is interesting that total spinal immobilization has never been proven to be of benefit, and the use of the long backboard has come under significant question.

11. Why is using a backboard considered a problem?

It has been shown that immobilization onto a backboard causes significant discomfort to the patient in a relatively short amount of time. It has also been demonstrated to potentially compromise ventilatory status in some patients. Last, patients who are allowed to remain on the board for prolonged periods have developed skin breakdown and decubitus ulcers. Thus for all of these reasons, patients who are placed on a backboard initially should be removed from them as soon as practical, given their condition.

12. If the backboard causes all of these problems, should it ever be used?

The backboard should continue to be used for specific purposes. It is an excellent device to help extricate patients or when patients need to be carried over large distances. However, its utility as an immobilization device has yet to be definitively demonstrated.

13. How should I 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:

Airway
Breathing
Circulation
Disability
Exposure

According to another mnemonic, a proper history is *A MUST*:

Altered mental state: Check for drugs or alcohol.
Mechanism*: Does the potential for injury exist?
Underlying conditions: Are high-risk factors for fractures present?
Symptoms: Is pain, paresthesia, or neurologic compromise part of the picture?
Timing: When did the symptoms begin in relation to the event?

14. 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 paraspinous 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 to assess sphincter tone and sensation. Regarding sensory testing, the assessment of light touch examines the integrity of the posterior neurologic columns, and pinprick testing assesses the anterior spinothalamic tracts. In an unconscious patient, the only clues to an SCI may be poor rectal tone, priapism, absence of deep tendon reflexes, or diaphragmatic breathing.

*Fall injuries are common. In the case of a fall, the physician should get information as to the height of the fall and any preceding events, such as syncope, chest pain, or seizure.

15. What is neurogenic shock, and how is it treated?

Neurogenic shock is a syndrome resulting from 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 hypotension, bradycardia, hypothermia, and ileus. The diagnosis of neurogenic shock should only be made after all other forms of shock, particularly hemorrhagic, 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.

16. 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, coordinated extrication, and movement of the patient only when absolutely necessary. A higher level of cervical injury results in a more devastating injury to the patient. Any patient with an SCI above C5 probably should be considered for intubation because the phrenic nerve roots, which supply the diaphragm, emerge from C3 to C5. Rapid-sequence intubation (RSI) orotracheal intubation with manual in-line cervical spine stabilization is considered the safest way to intubate the airways of 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 the spine 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.

17. 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.

18. 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 4000 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.

19. What is the CCR?

The CCR asks three questions:

1. Is there any high-risk factor that mandates radiography? These are:
   - Age older than 65 years
   - Significant mechanism of injury (i.e., fall from >1 m, axial loading injury, high-speed motor vehicle accident [MVA]/rollover/ejection, bike collision)
   - The presence of paresthesias
2. 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)?
3. 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, the clinician needs to review the rule completely before applying it to patients.

20. 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

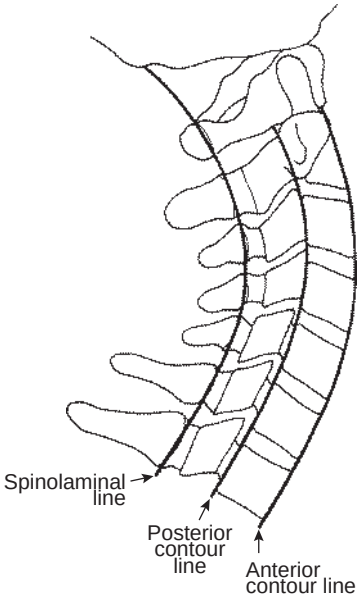

Figure 83-1. Curve of alignment.

21. 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 rules are difficult to verify in toddlers and children because of their inherent immaturity and verbal communication limitations.

22. Which radiographs should be obtained?

The standard three views of the cervical spine are anteroposterior (AP), lateral, and open-mouth (odontoid) views. During the initial evaluation, a cross-table lateral radiograph should be taken, because it does not require any movement on the part of the patient. It is extremely important that cervical spine precautions not be discontinued based solely on the cross-table lateral view. 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. Some series include oblique and pillar views of the vertebrae as well.

23. How do I interpret the lateral cervical spine radiograph?

The first rule is to make sure that the radiograph is technically adequate, that all seven cervical vertebrae are seen, and that the top of T1 is visible on the film. Next, follow the mnemonic *ABCS*:

Alignment: Check for a smooth line at the anterior and posterior aspect of the vertebral bodies and the spinolaminar line from C1 to T1 (Fig. 83-1).

Bones: 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.

Cartilage: Check the intervertebral joint spaces and the facet joints.

Soft-tissue spaces: Look for prevertebral swelling, especially at the C2 to C3 area (>5 mm) and check the predental space (Fig. 83-2), 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.

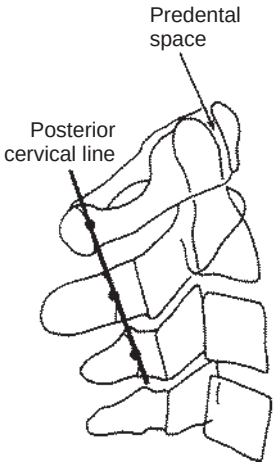

Figure 83-2. Posterior cervical line and predental space.

24. 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) scan or magnetic resonance imaging (MRI) provides more information in the presence of specific clinical concerns for fracture or ligamentous damage not diagnosed on plain films.

25. 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, because both studies can be accomplished at the same time. A CT scan is also needed for the clearance of the cervical spine in a 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.

26. 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 CCR, 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 patients, need a more thorough workup than the normal adult.
5. The three-view cervical spine series is mandatory in evaluations if you are getting plain radiographs, whereas the flexion-extension view is not needed in the acute setting.

27. 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 with anterior subluxation of the body of C2 on C3.
- 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.

28. 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).

29. 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.

30. What can emergency physicians do to prevent spinal injuries?

Get involved in injury prevention and education. Because of the predominance of vehicle crashes causing SCIs, one can work to reduce driving under the influence of alcohol and drugs, as well as the use of cell phones or texting while driving. Furthermore, 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

31. 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 (1992) and NASCIS 3 (1998), which evaluated regimens of high-dosage methylprednisolone. Initial support for the use of steroids was encouraging, but multiple critical 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. Further, a recent level I study showed that patients treated with high-dosage methylprednisolone had a higher incidence of serious complications, such as gastrointestinal bleeding and pneumonia. Unfortunately, the overwhelming desire for any improvement in these devastating injuries made steroid therapy a de facto standard of care at many institutions. Recent position statements by the American Academy of Emergency Medicine and Canadian Association of Emergency Physicians state that the use of steroids may be considered a treatment option but should not be considered a standard of care. The American Academy of Neurological Surgeons and

the Congress of Neurological Surgeons have gone further, and both recommended that steroids not be used at all in the first 24 to 48 hours after SCI. In view of these statements, it seems prudent to consult with the treating neurosurgeon before considering steroid therapy.

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QUESTIONS

1. You arrive on scene at an MVA, in which a driver struck a light pole at a high speed. The restrained passenger shouts to you, "Help! I can't move my arms!" What should you do next for this patient?
   a. Intubate the patient, because cervical spine injuries may compromise ventilation.
   b. If the person is not intoxicated, attempt to clinically clear the spine to avoid delays associated with immobilization.
   c. Carefully extricate the patient, ensure cervical spine immobilization, and place the patient on a backboard for transport.
   d. See if patient can stand and get into the ambulance for immediate transport to the nearest trauma center.

   The correct answer is *c*.
2. You are taking care of a young man who was shot in the back. Which of the following neurologic findings would be most consistent with left-sided Brown-Séquard syndrome?
   a. Impaired pain sensation on the right; impaired motor function and light touch sensation on the left
   b. Impaired pain sensation, motor function, and light touch sensation on the right
   c. Impaired pain sensation and motor function below the level of the injury but intact light touch sensation
   d. Impaired pain sensation, motor function, and light touch sensation on the left

   The correct answer is *a*.
3. Which of the following spinal fractures is not correctly paired with its classic mechanism of injury?
   a. Hangman fracture (disruption of the arch of C2): Cervical hyperextension
   b. Chance fracture: Hyperflexion of the lumbar spine
   c. Clay shoveler fracture: Forceful cervical hyperextension
   d. Jefferson fracture (C1 ring disruption): Cervical hyperflexion

   The correct answer is *d*.

HEAD TRAUMA

Edward Newton, MD

CHAPTER 84

1. What is the scope of head injury in the United States?

There are more than 1.3 million ED visits and approximately 52,000 deaths as a result of traumatic brain injury (TBI) every year in the United States.[1] Although the incidence of severe head injury is increasing, deaths from TBI are decreasing, most likely because of the preventive benefits of helmets, and seat belts and air bags in automobiles. In spite of this, 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 at the same time 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 patients with head injury, 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, some other 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.
- Patients with chronic alcoholism are at risk because of their greater incidence 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. Patients with chronic alcoholism
3. Patients with coagulopathy

3. What is a cerebral concussion?

A cerebral concussion is a sudden, transient loss of central neurologic function secondary to trauma. It is typically characterized by loss of consciousness (LOC; although LOC is not necessary to make the diagnosis), transient amnesia, confusion, disorientation, or transient visual changes, without any gross cerebral abnormalities or neurologic deficits on examination.

4. What is 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 commonly report migraine-type headaches,

Abstract

This is a review of key topics in the diagnosis and management of head trauma. The chapter examines the essential diagnostic findings, general and specific management strategies, and certain complications of head injury.

Keywords:

head injury, trauma, herniation, increased intracranial pressure (ICP), subdural hematoma (SDH), epidural hematoma, cerebral resuscitation, mannitol, hypertonic saline, computed tomography (CT) scan

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.

5. What is second impact syndrome?

A phenomenon known as *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.[2] Consequently, athletes should be held out of contact sports until all postconcussive symptoms have resolved. Repeated concussions can result in permanent impairment in cognition, speech, balance, and movement.

6. What complications are associated with basilar skull fractures?

Basilar skull fractures are often complicated by cranial nerve or cerebrovascular injury. CT angiography is often used to detect vascular injuries associated with basilar skull fracture.[3] 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 leakage of cerebrospinal fluid (CSF). 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. CSF leaks may present days to weeks after the initial injury

7. 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 instead select for antibiotic-resistant bacteria. Patients must be monitored 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 or endoscopic repair.

8. What are signs or symptoms of a patient with epidural hematoma?

Epidural hematoma occurs in 5% to 10% of severe head injuries.[4] In the classic pattern, a patient loses consciousness from the initial trauma, 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 have minor hemorrhages, and they 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.

9. How does an SDH present?

An SDH may be acute, subacute (6 to 14 days), or chronic (>14 days after trauma).

- An 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 have 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. 84-1). At times, the injury causes a minimal amount of bleeding and the patient does not immediately seek medical care. The SDH undergoes lysis over several days and eventually organizes into an encapsulated mass.
- A subacute or chronic SDH is a difficult clinical diagnosis, because the symptoms are vague, nonspecific, 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.

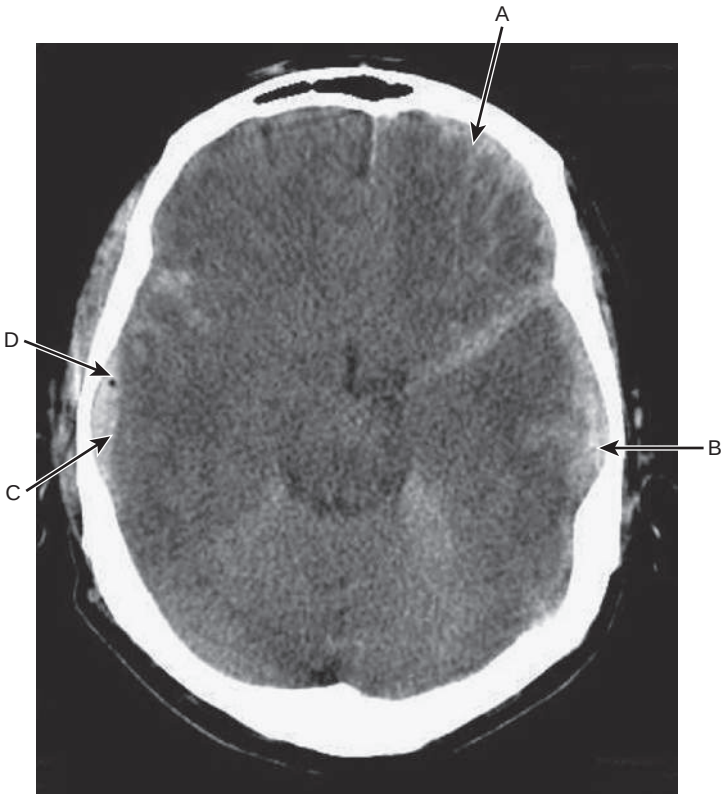

Figure 84-1. CT scan of the head showing subarachnoid hemorrhage *(A),* subdural hemorrhage *(B),* and subdural hemorrhage *(C)* with intracranial free air *(D, black dot).*

10. 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.

11. 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 brain stem.

12. List the four types of herniation syndrome.

1. Uncal herniation
2. Central herniation
3. Cingulate herniation
4. Posterior fossa herniation

13. 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 brain stem compression with respiratory and cardiac arrest. Transtentorial herniation of this type is the most common variety.

14. 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.

15. 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.

16. 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 brain stem dysfunction may occur rapidly and with little warning.

17. What is the ED treatment for increased ICP?

- Maintain adequate cerebral perfusion pressure[5,6]: Although there is often misguided reluctance to vigorously hydrate 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 neurologic 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 five *Hs* (**h**ypotension, **h**ypoxia, **h**ypercarbia, **h**ypoglycemia, and **h**yperthermia) 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 patients with head injury who experience hypoxia (partial pressure of oxygen [PO_2] <60 mm Hg) also have a twofold increase in mortality rate. 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 decreases the vascular compartment of the brain and may "buy some time" for definitive surgical interventions. Unfortunately, when blood flow to the brain decreases, delivery of oxygen and glucose also decreases, resulting in ischemic injury and worse edema, so this intervention is used only in patients who are rapidly deteriorating neurologically (i.e., herniating). The optimal level of hypocarbia is uncertain at present, but most clinicians recommend moderate short-term hyperventilation, with a partial pressure of carbon dioxide (PCO_2) level no less than 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's airway with rapid-sequence intubation (RSI) and mechanically ventilate with settings determined by arterial blood gases to maintain the PCO_2 at or near 35 mm Hg. Hyperventilation is never used prophylactically.
- 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.[7] It has been shown to be at least as effective as mannitol in treating elevated ICP and may have a more prolonged effect than mannitol. It is preferred over mannitol in hypotensive patients. 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 an external ventricular drain (EVD) is occasionally implemented in the ED and is perhaps the most effective way of monitoring and rapidly lowering ICP.
- Sedation: 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 0.05 to 0.2 mg/kg, or midazolam 0.1 mg/kg followed by an infusion at 0.01 to 0.2 mg/kg/hr, 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. Propofol (2 to 2.5 mg/kg induction dose, followed by an infusion of 0.1 to 0.4 mg/kg/min) is often used for induction of anesthesia and ongoing sedation. Lower doses should be used to avoid hypotension or apnea in older or debilitated patients or in those with liver failure. Fentanyl 0.1 to 0.3 μg/kg causes a slight increase in ICP and is not the preferred agent for sedation of a patient with head injury.

18. Is there any role for therapeutic hypothermia in patients with TBI?

Reducing a patient's body temperature to 32°C to 33°C (90°F to 93°F) for 24 to 48 hours has shown some benefit in preserving neurologic function in survivors of cardiac arrest, and it was hoped that it would show the same benefits in patients with brain injury. However, the results of several trials have been conflicting.[8] If any benefit occurs, it is likely in those who have a Glasgow Coma Scale (GCS) score of 5 to 8, but even in these patients, the treatment should be considered experimental.[5] 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 intravenously) or levetiracetam (Keppra).
5. Hyperventilate to pCO_2 of 35 mm Hg only if patient has elevated ICP and is clinically herniating.
6. Osmotic therapy should done with either mannitol or hypertonic saline.
7. Correct coagulopathy.

19. 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.[9]

20. What are the indications for a repeat head CT scan?

Some centers routinely schedule a repeat head CT scan after the initial scan is positive for intracranial hemorrhage (ICH), although this practice is not recommended based on the evidence.[10] The indications for repeat head CT scan are as follows:

- Clinical deterioration as indicated by worsening mental status, progression of focal neurologic deficits, or declining GCS score should prompt a repeat scan.
- Patients taking coumadin or other anticoagulant drugs have approximately a 2% to 3% chance of developing a delayed ICH after head trauma, even if the initial head CT is normal. A repeat head CT 6 hours after the initial study is indicated. Patients taking antiplatelet agents, such as aspirin or clopidogrel, have a higher overall risk of ICH after head trauma and a worse prognosis if bleeding occurs. However, the hemorrhage is typically present on the initial head CT and patients do not appear to have an increased risk of delayed bleeding; thus a repeat CT is not necessary in these patients.

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QUESTIONS

1. In a patient with head trauma who has a normal neurologic examination and normal head CT scan, which of the following is an indication for routine repeat head CT scan 6 hours after injury?
   a. Anticoagulation with coumadin
   b. History of chronic alcohol abuse
   c. History of an early posttraumatic seizure
   d. LOC at the time of head impact
   e. Use of antiplatelet agents, such as aspirin or clopidogrel
   The correct answer is *a*. Studies of delayed intracranial bleeding after head trauma have identified only use of coumadin as an increased risk (2% to 3% incidence) of delayed intracranial bleeding in patients with a normal initial head CT scan. Patients taking antiplatelet agents have an increased risk of bleeding, but the hemorrhage in these cases is apparent on the initial head CT scan. Such patients do not appear to have an increased risk of delayed ICH if the initial head CT is normal.
2. Which of the following symptoms is consistent with postconcussion syndrome?
   a. Diplopia
   b. Facial numbness
   c. Hand weakness
   d. Urinary incontinence
   e. Vomiting
   The correct answer is *e*. Postconcussion syndrome includes a wide variety of neuropsychiatric symptoms, such as headache, nausea and vomiting, insomnia, lethargy, and difficulty concentrating, to name a few. Hard findings of brain injury, such as focal numbness or weakness or cranial nerve dysfunction are not part of postconcussion syndrome and are suggestive of gross cerebral pathology.
3. A 14-year-old football player is brought to the ED after sustaining a brief LOC after a collision on the field. He regained full alertness en route, but after a brief observation period, he developed sleepiness and ptosis of his left eyelid. Examination reveals weakness of the right arm and leg and a sluggish and dilated left pupil. This is most consistent with which of the following?
   a. Central herniation
   b. Cingulate herniation
   c. Cerebellar herniation
   d. Spinal cord injury
   e. Uncal herniation
   The correct answer is *e*. The symptoms described fit with uncal herniation, in which the third cranial nerve is compressed, producing ipsilateral ptosis, gaze paresis, and papillary dilation, as well as contralateral paresis. Central herniation causes bilateral motor weakness; posterior fossa herniation causes a sudden apnea, bradycardia or asystole, hypotension, loss of motor tone, and unresponsiveness. Spinal cord injury does not cause cranial nerve or mental status changes.

TRAUMATIC OPHTHALMOLOGIC EMERGENCIES

Edited by Peter T. Pons, MD, FACEP

1. **Name the two most time-critical emergencies in ophthalmology.**
   Central retinal artery occlusion and chemical burns to the eyes are the most time-critical of ophthalmologic emergencies.

2. **What is the treatment for a chemical burn of the eye?**
   The eyes should receive immediate copious irrigation for at least 20 minutes. Irrigation should be initiated before transport to the ED. Tap water may be more effective than normal saline.

3. **How do I know when I 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. High-flow oxygen is also thought to improve visual outcomes.

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?**
   The most common eye injury seen in the ED is corneal abrasion with or without a superficial foreign body.

7. **How is corneal abrasion diagnosed?**
   The anesthetized eye is 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 commonly overlooked aspect of therapy is the instillation of a cycloplegic agent, usually cyclopentolate (Cyclogyl), to relieve the ciliary spasm that often accompanies this injury. Patients also need evaluation for tetanus prophylaxis. Most should receive topical antibiotics, drops, or ointment. Nonsteroidal antiinflammatory eye drops have also been proven to be useful.

Abstract

This chapter will discuss the most important emergent diagnoses and treatment for traumatic eye injuries. Indications for emergent ophthalmology consultation and emergency treatment will be emphasized.

Keywords:

eye trauma, corneal abrasion, penetrating global injuries, hyphema, corneal foreign body, blow-out fracture, traumatic iritis, traumatic mydriasis, solar keratitis

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 they also 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 or use heavy machinery, 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 given patches. 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, anterior hyphema, is seen as a layering of red blood 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 should be covered with a patch, and ophthalmologic consultation should be initiated, at least by phone to arrange for prompt follow-up treatment. 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 of 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
3. Paralysis or limitation of upward gaze (manifested as diplopia), resulting from entrapment of the inferior rectus muscle

16. What is traumatic mydriasis?

Traumatic mydriasis is 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 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?

An ophthalmologist or plastic surgeon should repair injuries 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 to 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 retroorbital hematoma?

Bleeding behind the globe (retroorbital 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 retroorbital 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 the result of 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 to time of exposure to time initiating irrigation; do not wait for the patient to arrive at the hospital.
2. Never put a patch on 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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QUESTIONS

1. Which of the following requires immediate intervention in the ED?
   a. Chemical exposure to the eye
   b. Corneal ulcer with hypopyon
   c. Orbital floor fracture
   d. Ruptured globe
   The correct answer is *a*.
2. What is the value of an eye patch in a patient with a corneal abrasion?
   a. Improves healing time
   b. None
   c. Prevents secondary infection
   d. Relieves pain
   The correct answer is *b*.
3. What should be done to manage a traumatic retrobulbar hematoma?
   a. Emergent lateral canthotomy
   b. Emergent ophthalmology consultation
   c. Emergent paracentesis of anterior chamber
   d. Increase ambient carbon dioxide
   The correct answer is *a*.

NECK TRAUMA

Christina H. Georgopoulos, MD

CHAPTER 86

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, respiratory, and gastrointestinal systems, provides a fertile ground for debate and myriad opinions about modality of treatment.

2. 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 and tracheal trauma causes voice alteration, airway compromise, subcutaneous emphysema, crepitus, and hemoptysis.
- Injury to the esophagus causes pain, neck tenderness, subcutaneous emphysema, dysphagia, and bleeding from the mouth or nasogastric tube.

3. What are the most urgent concerns in the initial management of neck trauma?

Airway and hemorrhage control must be accomplished first. Airway management comes before anything else discussed in this chapter. Early endotracheal intubation is indicated for any patient with signs of existing or potential airway compromise, including altered mental status, expanding hematoma, hypoventilation or hypoxia, or direct trauma to the trachea or larynx. Delay in airway management increases the difficulty of intubation because of swelling, distortion, and compression of the anatomic structures. Bleeding should be controlled with direct pressure, using two fingers over the source of the bleed, as opposed to blind pressure over large areas or blind clamping. Once the airway and bleeding are controlled, the wound should be examined to determine whether it has violated the platysma. Avoid injudicious probing of the wound, however, because a vascular structure that has ceased to bleed may resume with massive hemorrhage and disastrous consequences when its tamponade is released.

4. What is the preferred method to secure the airway?

Rapid-sequence intubation (RSI) with oral tracheal intubation should be the initial airway approach in patients with no damage to minimal distortion of their airway. Patients with airway distortion with anticipated difficult bag-valve-mask ventilation should have their airways managed with local airway anesthesia or sedative-assisted oral tracheal intubation. The preferred sedative medications include midazolam and fentanyl, because they are reversible, or ketamine, because it does not depress spontaneous respirations. Equipment for a surgical airway should be immediately available at the bedside, and cricothyrotomy should be employed if endotracheal intubation is unsuccessful. Tracheostomy by an appropriately trained physician is preferred over cricothyrotomy if there is an anterior hematoma or visible damage to the larynx and cricoid cartilage.

5. What are the indications for cervical spine immobilization in neck trauma?

In patients with blunt trauma, the indications for cervical spine immobilization include posterior neck tenderness, pain with motion of the neck, or focal neurologic findings on examination. In patients with penetrating trauma, the chance of spinal injury is extremely rare. Cervical spine immobilization in these patients is only required if the patient is obtunded, or if there is altered mental status or a focal neurologic finding on examination. For a patient requiring immobilization, be sure to maintain inline stabilization while the airway is being secured.

6. What are the three anatomic zones of the neck?

1. Zone I is the area below the cricoid cartilage. With injuries to this area, trajectory may involve other anatomic areas, including the chest and cardiac box.
2. Zone II extends from the cricoid cartilage to the angle of the mandible. This has classically been the most surgically accessible zone of the neck.

Abstract

This is a review of the evaluation and management of penetrating and blunt cervical trauma. This chapter covers the procedure for securing the airway, the anatomic zones of the neck, the hard and soft signs of penetrating neck trauma, the imaging modalities for penetrating neck trauma, the indications and modalities to image for blunt neck trauma, and the management of blunt vascular injuries.

Keywords:

penetrating neck trauma, blunt neck trauma, cervical vascular injury, aerodigestive injury

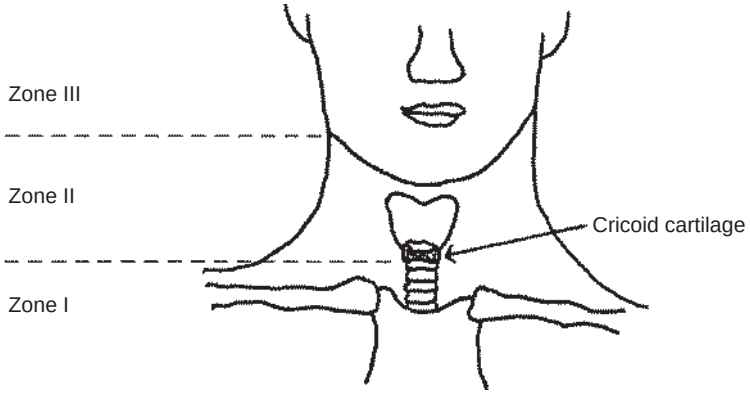

Figure 86-1. Zones of the neck.

3. Zone III extends from the angle of the mandible to the base of the skull. With these injuries, trajectory may involve the head.

Figure 86-1 illustrates the zones of the neck.

7. Why is the neck divided into three zones?

Traditionally, the workup for penetrating neck injuries in stable patients required conventional angiography for zone I and III injuries, because the complex anatomy in these zones did not allow for easy surgical exposure. With the improvement of helical computed tomography angiography (CTA) over the past 2 decades, there is less of a role for conventional angiography to evaluate injuries in these zones. Further, there is less of a role for formal surgical exploration in stable patients with zone II injuries. Therefore the separation of the neck into zones has become less important. Currently, the workup is not decided upon by the zone of the neck involved in the injury, but rather by the stability of the patient and the presence of specific findings on physical examination.

8. What is the main controversy regarding management of penetrating neck trauma.

In the 1990s, surgeons changed from the mandatory exploration of all penetrating neck wounds to a selective management approach. Earlier in the 1940s, mandatory exploration was instituted for all penetrating wounds that violated the platysma. This policy reduced the mortality rate significantly, and remained the only mode of therapy until the mid-1970s. In this setting, the negative exploration rate was 50%, and therefore the cost of the operation and the added length of stay were unwarranted. Many of these operations could be avoided with a more selective approach to neck exploration. With the improved sensitivity and specificity of ancillary diagnostic testing (mainly CTA, as well as esophagography, esophagoscopy, and laryngoscopy), a nonoperative approach to a select group of patients is safe. The selective approach has reduced the negative exploration rate from 50% to 30%.

9. What is the current algorithm for the workup of penetrating neck trauma?

Patients who are hemodynamically unstable or have findings of significant neck injury (see Question 2) are taken immediately for surgical exploration. Given the clarity provided by modern helical CTA, stable patients are now evaluated by CTA for any occult injury to the vascular or aerodigestive structures, rather than being taken for surgical exploration. Patients with a normal CTA but with soft signs on physical examination can be further evaluated with ancillary tests, including esophagography, esophagoscopy, laryngoscopy, and, rarely, formal angiography.

10. What are the hard and soft signs of penetrating neck trauma?

Hard vascular signs include significant bleeding, bruits and thrills, large or pulsatile hematomas, altered mental status, and shock. Hard aerodigestive signs include airway compromise, a laceration visibly involving the trachea, hemoptysis, hematemesis, and air bubbling through the wound. Hard

Table 86-1. Signs and Symptoms of System Injuries

VASCULAR	AERODIGESTIVE
Hematoma	Respiratory distress
Hemorrhage	Stridor
Neurologic deficit	Cyanosis
Pulse deficit	Hemoptysis
Horner 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

physical examination signs require immediate surgical exploration. Soft physical examination signs, on the other hand, require CTA and ancillary tests. These signs include small bleeds and hematomas, mild dysphagia and dysphonia, mild subcutaneous emphysema, and tenderness in the neck.

For more details, see Table 86-1.

11. Can CTA replace conventional angiography for detection of vascular injuries in penetrating neck injuries?

In the more current 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.

12. Which diagnostic studies are important in suspected laryngeal injuries?

- Soft-tissue cervical radiographs may show a fractured larynx, subcutaneous air, or prevertebral air.
- Computed tomograph (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.

13. Which diagnostic studies are important in suspected esophageal injuries?

Soft-tissue cervical radiographs may show subcutaneous emphysema or an increased prevertebral soft-tissue shadow. Chest radiograph findings may include pleural effusion, pneumothorax, mediastinal air, and mediastinal widening, or be normal in appearance if obtained early in the patient's course. Esophageal contrast studies (esophagography) should be done initially with radiopaque contrast medium (Gastrografin); if negative, studies should be repeated with barium to increase diagnostic yield. These studies have a 30% to 50% false-negative rate and should be followed by esophagoscopy in patients with suspected esophageal injury. 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.

14. What are the signs and symptoms of blunt carotid or vertebral 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 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 syndrome, transient ischemic attack, aphasia, or hemiparesis. The clinical manifestations of vertebral artery injury include ataxia, vertigo, nystagmus, hemiparesis, dysarthria, and diplopia.

15. What are the indications for imaging to evaluate for vascular injury in patients with blunt cervical trauma?

Classically, the mechanisms for carotid injury include cervical hyperextension and rotation, hyperflexion, or direct blow. Indications for emergent imaging are arterial hemorrhage or expanding hematoma in the head or neck, cervical bruit in patients younger than 50 years, and focal neurologic findings. Imaging should also be considered in patients to screen for vascular injury if they have the following findings:

- Cervical hyperextension and rotation or hyperflexion injury
- Le Fort II or III midface fracture
- Basilar skull fracture
- Closed head injury with Glasgow Coma Scale (GCS) score less than 6
- Cervical vertebral fracture
- Near-hanging incident

16. What diagnostic testing is preferred in the 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% of patients. 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 (2006) found that CTA had a 97% sensitivity and 100% specificity. CTA has been shown to decrease significantly the time to diagnose the injury.

17. Is there any role for Doppler ultrasound in blunt vascular trauma?

Color-flow Doppler ultrasound provides rapid identification and quantification of arterial dissection in the carotid artery, but it is unable to assess the distal upper extracranial and intracranial internal carotid artery and is highly operator dependent. Although some authors suggest that with an experienced operator, ultrasound can be used as a screening test in lower-risk patients, the Eastern Association for the Surgery of Trauma (Bromberg 2010) published a guideline stating that, because of its limitations, duplex ultrasound cannot be recommended to screen for blunt cerebrovascular injury.

18. How about MRI? Can it be used to identify blunt vascular injury?

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 examination or for stable patients; MRA is difficult to perform in an acutely injured unstable patient.

19. What is the appropriate management of blunt vascular injuries?

For patients with an ischemic stroke related to a carotid or vertebral artery dissection, antiplatelet or anticoagulation therapy should be given for 3 to 6 months. Because this duration is arbitrary, many physicians prefer to repeat CT angiography to confirm recanalization of the vessel before cessation of these medications. For a patient who has recurrent cerebral ischemic symptoms in spite of medical therapy, endovascular stenting should be considered. If stenting fails in the patient or is not a candidate for endovascular therapy, he or she should be considered for surgical intervention.

KEY POINTS: MANAGEMENT OF NECK TRAUMA

1. Manage airway early before airway distortion occurs.
2. Use oral tracheal intubation with RSI as an initial airway management option.
3. Always set up for a backup surgical airway at bedside while attempting oral tracheal intubation.
4. Do not forget to work up anatomic areas adjacent to zone I and zone III.
5. Patients with continued suspicion for occult injury after a negative CTA should undergo ancillary tests, including conventional angiogram, esophagoscopy/esophagography, and/or laryngoscopy.
6. CTA is a reliable test for the workup and screening of blunt cervical vascular injury.

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QUESTIONS

1. Which of the following is not the correct description of a zone of the neck?
   a. Zone II extends from the cricoid cartilage to the angle of the mandible.
   b. Zone I is the area below the cricoid cartilage.
   c. Zone III is the area below the cricoid cartilage.
   d. Zone III extends form the angle of the mandible to the base of the skull.
   The correct answer is *c.*
2. Which of the following is a hard sign for penetrating neck trauma?
   a. Dysphagia
   b. Neck tenderness
   c. Neck bruit
   d. Subcutaneous emphysema
   The correct answer is *c.*
3. Which of the following is not a sign for emergent imaging in blunt neck trauma?
   a. Enlarging neck hematoma
   b. Cervical bruit in a 55-year-old patient
   c. Unilateral upper extremity weakness
   d. Epistaxis
   The correct answer is *b.*

CHAPTER 87

CHEST TRAUMA

Jennifer A. Salotto, MD, and Robert T. Stovall, MD

1. What is the initial approach to the patient with chest trauma?

The initial approach to the patient with chest trauma should follow standard Advanced Trauma Life Support (ATLS) protocol and includes assessing the airway, breathing, circulation, and disability, and exposing the patient.

- The airway is first assessed for patency or obstruction, air movement, stridor, and need for intubation. Patients with a Glasgow Coma Scale score less than 8, massive hemoptysis, or difficulty oxygenating or ventilating should have their airways intubated for definitive airway control.
- The lungs are auscultated for the presence of bilateral breath sounds.
- Peripheral pulses are identified, intravenous (IV) access is established, and fluid or blood is infused as indicated. The patient is placed on a monitor for continuous measurements of vital signs. External bleeding is controlled with direct pressure.
- A neurologic examination should be performed before any sedation.
- The patient should be completely undressed for a thorough examination. Details from the trauma scene, as well as any known medical history, should be taken from the patient, police, or prehospital provider who brought the patient from the scene.

2. What is the proper way to examine the chest during the trauma survey?

Remember to look, listen, and feel.

- Look (inspect): After undressing the patient, the entire chest, including the axillae and back, are inspected. Patients with penetrating injuries should be rolled early to identify any life-threatening wounds on the back. Bruises or lacerations should be noted, and gunshot wounds or stab wounds should be identified. Abnormal chest rise or paradoxic movements may indicate rib fractures or a flail segment.
- Listen (auscultate): The examiner should listen for bilateral breath sounds. Decreased or absent breath sounds can indicate a pneumothorax, hemothorax, or diaphragm injury. If an endotracheal tube has been placed, the absence of breath sounds on one side may indicate that main stem bronchus intubation.
- Feel (palpate): The chest should be palpated, specifically looking for any subcutaneous emphysema (potentially an indication of underlying pneumothorax), fractures, or tenderness. The patient should be rolled, using in-line stabilization of the spine as necessary, and the back and thoracic spine inspected and palpated.

3. What are the immediate threats to life after thoracic trauma, and how are they identified?

The immediate threats to life include airway obstruction, tension pneumothorax, open pneumothorax, flail chest, massive hemothorax, and cardiac tamponade. Airway obstruction, tension pneumothorax, open pneumothorax, and severe flail chest are diagnosed with physical examination. Radiography of the chest can diagnose massive hemothorax and flail chest. The focused assessment with sonography for trauma (FAST) pericardial view can be used to diagnose cardiac tamponade.

4. Which major organs may be injured in blunt or penetrating chest trauma?

- Lungs and tracheobronchial tree
- Heart and great vessels: Aorta, vena cavae, and pulmonary, axillary, and subclavian vessels
- Esophagus, diaphragm, and thoracic duct
- Chest wall: Ribs, clavicle, scapula, sternum, and thoracic spine

Abstract

This chapter describes the initial assessment of a patient with suspected thoracic trauma and details the diagnosis and treatment of key injuries sustained after blunt and penetrating chest trauma.

Keywords:

chest trauma, tension pneumothorax, pneumothorax, flail chest, hemothorax, tamponade, focused assessment with sonography for trauma (FAST) examination, tube thoracostomy, rib fractures, open pneumothorax, blunt aortic injury, blunt cardiac injury, pulmonary contusion, resuscitative thoracotomy, thoracic spine fracture, neurogenic shock

KEY POINTS: INITIAL ASSESSMENT CHEST TRAUMA

1. Evaluation of thoracic trauma begins with assessment of the airway, breathing, circulation, and disability, and exposure of the patient.
2. To fully assess the chest, one must look, listen, and feel.
3. Immediate threats to life that must be identified and treated in the primary survey include airway obstruction, tension pneumothorax, open pneumothorax, flail chest, massive hemothorax, and cardiac tamponade.

5. What is a pneumothorax, and what may commonly cause it?

A pneumothorax is air in the pleural space. It is caused by any opening that allows air into the pleural space via the tracheobronchial tree, lung, mediastinum, or chest wall.

6. What are the signs and symptoms of a pneumothorax?

Signs and symptoms of a pneumothorax may include shortness of breath, chest pain, hypoxia, tachypnea, tachycardia, diminished breath sounds on the affected side, and subcutaneous emphysema. In some cases, the patient may appear asymptomatic.

7. How is a pneumothorax diagnosed?

Most commonly a pneumothorax is diagnosed with a chest radiograph as the initial adjunct to the primary and secondary survey. Computed tomography (CT) scan may diagnose smaller pneumothoraces not seen on plain film. The use of ultrasound to assess for pneumothorax has been shown to be useful in skilled hands. Ultrasound is helpful when it shows a pneumothorax; however, a negative ultrasound does not rule out the diagnosis. Chest radiography remains the imaging modality of choice.

8. How do I treat a pneumothorax?

A pneumothorax is treated with tube thoracostomy. Smaller chest tubes are felt to be adequate for the treatment of a pneumothorax without hemothorax. Small, asymptomatic pneumothoraces may be observed with no intervention. A follow-up radiograph of the chest is prudent to ensure stability in cases where no intervention is made.

9. What is a tension pneumothorax?

A tension pneumothorax results from the accumulation of air in the pleural space under pressure. When pressure reaches a critical level, displacement of the heart impairs venous return to the heart, leading to decreased filling of the heart, decreased cardiac output, and potential cardiovascular collapse.

10. What are the possible signs and symptoms of a tension pneumothorax?

All of the signs of a simple pneumothorax may be present, but with a tension pneumothorax, tachycardia, distended neck veins, hypotension, tracheal deviation (late sign), and even cardiovascular collapse may be present.

11. How should a tension pneumothorax be diagnosed?

A tension pneumothorax is a life-threatening emergency and should be diagnosed clinically.

12. How is a tension pneumothorax treated?

It is treated with immediate decompression of the pleural space. This is at times both diagnostic (with a positive rush of air) and therapeutic. Temporary needle decompression with a large-bore needle is performed in the second or third intercostal space anteriorly, followed by tube thoracostomy. A rapidly placed chest tube may also be an appropriate first choice, depending on the setting and available equipment. In a true crisis situation, a scalpel is sufficient to enter the pleural space for adequate and life-saving temporary decompression of the pleural space.

KEY POINTS: TENSION PNEUMOTHORAX

1. Tension pneumothorax is a clinical diagnosis.
2. Absent breath sounds, hypotension, and distended neck veins should point to the diagnosis.
3. Treatment includes immediate decompression of the chest with a large-bore needle, chest tube, or knife.

13. What is an open pneumothorax, and how is it diagnosed?

Also known as a *sucking chest wound,* an open pneumothorax is a direct and open communication of the pleural space with the atmosphere. An open pneumothorax can be an immediate threat to life, in that it may compromise ventilation and oxygenation. It is usually diagnosed clinically on physical examination.

14. How should an open pneumothorax be treated?

A three-sided dressing is traditionally described in the prehospital setting. This prevents the accumulation of pressure in the pleural space and the development of a tension pneumothorax while improving the patient's respiratory dynamics. Purpose-made vented chest seals may also be used. Once in the hospital, a chest tube is placed and the open wound is covered with an occlusive dressing. Definitive operative repair is done as necessary.

15. What is a hemothorax, and how is it diagnosed?

A hemothorax is blood in the pleural space. This blood may come from a variety of sources, including trauma to the lung, intrathoracic blood vessels, or heart. A hemothorax is commonly diagnosed with chest radiography and, at times, CT scan. Signs and symptoms are similar to those of pneumothorax, including shortness of breath, hypoxia, respiratory compromise, and decreased breath sounds. If the volume of hemothorax is great enough, signs of hypovolemia may be evident.

16. What is the treatment of hemothorax?

For the hemodynamically stable patient, drainage is the standard treatment, initially with tube thoracostomy. There is some debate as to exactly how large a hemothorax should be before attempting drainage. Moderate to large hemothoraces should be drained, and some advocate that any hemothorax seen on a chest radiograph should be drained. Traditionally, a large-bore (32 French or greater) chest tube has been used to drain a hemothorax, but this concept is being challenged, and it is possible that a smaller size chest tube is adequate. For the hemodynamically unstable patient, ongoing resuscitation with drainage is still the first option, but surgery for hemorrhage control is paramount to patient survival.

17. What should I do if my chest tube does not completely drain the hemothorax?

A second chest tube is a possible consideration. However, with available thoracoscopic techniques, early video-assisted thoracoscopic washout of a retained hemothorax is a good option. If after the second chest tube a residual hemothorax exists, proceeding to thoracoscopic washout is preferred if the patient is stable.

18. What is a massive hemothorax?

A massive hemothorax occurs when more than 1500 mL of blood, or a third of a patient's blood volume, accumulates in the chest. Those with massive hemothorax usually show signs of hemorrhagic shock and respiratory compromise. Initial treatment consists of tube thoracostomy and rapid resuscitation with IV fluids and blood products. This is often followed by definitive surgery.

19. How much ongoing blood loss out of the chest tube is an indication for operative exploration?

If the patient is hemodynamically unstable with a likely chest source, he or she should be in the operating room. If a patient is hemodynamically stable after blunt trauma, the immediate drainage of 1500 mL of blood with ongoing bleeding is an indication for operative intervention. For penetrating trauma, surgical intervention is indicated after the immediate drainage of 1000 mL of blood with ongoing bleeding. Finally, greater than 200 mL/h for more than 2 hours is also an indication for exploration in either blunt or penetrating trauma.

20. What other fluids may fill the pleural space after trauma?

Some examples are chyle, serous fluid, pancreatic fluid, and bile, although almost any fluid can be present; however, these are usually rare.

21. What is the best management of an asymptomatic, hemodynamically stable patient after penetrating thoracic trauma with no pneumothorax or hemothorax on initial chest radiography?

Keep the patient in the ED for observation. If no other physical abnormalities are encountered, obtain a chest radiograph at 3 hours, and if still normal, the patient may be discharged home.

22. What is a pulmonary contusion, and how is it diagnosed?

A pulmonary contusion is an injury to the lung parenchyma that causes accumulation of blood and fluid in the airspaces of the lung. Severity may range from asymptomatic to severe respiratory failure. Signs include hypoxia and shortness of breath, hemoptysis, and chest wall pain. Diagnosis is made with a chest radiograph or CT scan. A pulmonary contusion appears as ground glass or consolidation not confined to lobar designations of the lung. It is common for a pulmonary contusion to "blossom" 24 to 48 hours after injury, resulting in a worse respiratory status than on presentation. Pulmonary contusion should be suspected after almost any blunt chest trauma; however, it is commonly associated with rib fractures and especially with flail segments.

23. How is a pulmonary contusion managed?

Pain control, supplemental oxygen, and pulmonary toilet are the mainstays of therapy. Mechanical ventilation may be required in severe cases. Observation, continuous pulse oximetry monitoring, and expectant management are key, because the clinical signs and symptoms may worsen after the initial presentation.

24. What are the signs and symptoms of an intrathoracic tracheobronchial tree injury?

Signs of injury to the tracheobronchial tree include shortness of breath, hypoxia, persistent pneumothorax after a chest tube has been inserted, or a large persistent air leak through the chest tube. Small injuries are easily missed and can present in a delayed fashion as pneumonia, pulmonary abscess, mediastinitis, or sepsis.

25. How are tracheobronchial tree injuries diagnosed and treated?

Tracheobronchial tree injuries are diagnosed at surgical exploration or with bronchoscopy. Bronchial injuries most commonly occur on the right within 2.5 cm of the carina. Imaging studies may suggest an injury, but bronchoscopic confirmation is often necessary. Radiographic findings that may point to this injury include tracheal and bronchial extraluminal air, the fallen lung sign, subcutaneous emphysema, and pneumomediastinum. Small injuries may be observed. Large injuries require operative repair.

26. What are the signs and symptoms of cardiac tamponade?

Cardiac tamponade occurs when the pericardial sac fills with fluid or blood. In its most severe form, cardiac tamponade can cause cardiogenic shock and hemodynamic collapse. Earlier signs of tamponade include tachycardia, *pulsus paradoxus*, and elevated jugular venous pressure. The combination of muffled heart sounds, jugular venous distention, and hypotension is called the *Beck triad* and is associated with cardiac tamponade.

27. How can cardiac tamponade be diagnosed?

It can be quickly diagnosed with a transthoracic ultrasound (FAST examination) in the trauma bay. This test can be quickly repeated with any signs of decompensation and is highly sensitive and specific in capable hands.

28. How is cardiac tamponade treated?

A positive FAST examination should prompt transfer to the operating room for a pericardial window. Pericardiocentesis is only recommended for decompression of a traumatic cardiac tamponade if surgical intervention is not possible or not rapidly available. Pericardiocentesis is not definitive treatment of the tamponade but does allow stabilization of a patient's hemodynamics before definitive treatment. Tamponade leading to loss of vital signs is an indication for resuscitative thoracotomy and opening of the pericardium.

KEY POINTS: TRAUMATIC CARDIAC TAMPONADE

1. Cardiac tamponade occurs when the pericardial sac fills with blood.
2. Signs and symptoms include dyspnea, tachycardia, elevated jugular venous pressure, and hypotension.
3. Diagnosis can be made quickly at the bedside when a trained provider performs ultrasound.
4. Optimal treatment is operative decompression.

29. What is blunt cardiac injury (BCI)?

BCI occurs when blunt force to the chest results in altered structure or function of the heart. BCI is a spectrum of disease that includes cardiac contusion, coronary artery thrombosis, cardiac rupture, pericardial rupture, and commotio cordis (ventricular tachycardia or fibrillation after a sudden blow to the chest). Clinically, these entities may manifest as chest pain, electrocardiographic changes, or a new dysrhythmia.

30. What is the appropriate management for suspected BCI?

An admission electrocardiogram (ECG) should be performed on any patient with suspected BCI. The most common ECG finding after BCI is sinus tachycardia or premature contractions. Unfortunately, ECG alone does not rule out BCI. If an ECG shows a new dysrhythmia, a new heart block, or ischemic changes, the patient should be admitted for continuous cardiac monitoring. Echocardiography (ECHO) should be performed on any symptomatic patient or any patient with any new dysrhythmias or ischemic changes on ECG.

31. When should a penetrating cardiac injury be suspected, and how is it diagnosed?

A penetrating cardiac injury should be suspected with any penetrating injury in proximity to the heart. If the patient is stable, a FAST examination should be performed to evaluate for pericardial blood, and if positive, the patient should undergo subsequent pericardial window, usually in the operating room. A chest radiograph may demonstrate a hemothorax from a cardiac injury that is decompressing into the thorax. Diagnosis and treatment should occur emergently in the operating room for those who are hemodynamically unstable.

32. How does the management of a suspected transmediastinal gunshot wound differ in a hemodynamically stable patient from the management of an unstable patient?

Any patient with a missile trajectory that crosses the midline should be evaluated for potential injury to any of the mediastinal structures.

- In the hemodynamically stable patient, the primary and secondary surveys are performed, IV access is obtained, and tube thoracostomy is performed as needed. Imaging should include chest radiography with radiolucent markers over wounds to identify trajectory, a FAST examination, and a thoracic CT scan with IV contrast. Depending on missile trajectory, adjuncts to diagnosis include angiography, bronchoscopy, esophagoscopy, or esophagogram as indicated.
- An unstable patient with a suspected transmediastinal gunshot wound should be taken emergently to the operating room. Management in the trauma bay should be limited to establishing an airway and IV access, decompression of the chest if needed, sending a blood sample for type and cross-match, and performing chest radiography. A resuscitative thoracotomy should be performed for a loss of vital signs within 15 minutes of arrival.

33. What is blunt aortic injury, and how does it occur?

Blunt aortic injury occurs when shear, torsion, or compressive forces are applied to the trunk and aorta, most commonly after a deceleration force in a motor vehicle collision. Tears of the intima or media can lead to dissection or pseudoaneurysm and predispose that area to rupture. Aortic rupture is a common cause of death after blunt thoracic trauma before the arrival to the hospital.

34. At which anatomic location is the aorta most commonly injured after blunt trauma?

Blunt aortic injury occurs in areas where the aorta is fixed in place, including at the aortic root, at the ligamentum arteriosum, and at the diaphragm. The most common location for blunt aortic injury is at the ligamentum arteriosum, just distal to the take-off of the left subclavian artery.

35. How do blunt aortic injuries present, and how are they diagnosed?

Blunt aortic injuries are often clinically occult, in that the patient may be asymptomatic with no external signs of trauma. Chest pain, shortness of breath, or back pain may be presenting signs. On physical examination, the patient may have tenderness or bruising over the sternum or anterior ribs and an abnormal upper extremity arterial-arterial gradient is sometimes present. A high index of suspicion with the appropriate mechanism of injury is paramount to timely diagnosis.

36. Which modalities are used to diagnose blunt aortic injury, and what are the radiographic findings?

Chest radiography and CT angiography (CTA) are first-line imaging modalities in the workup of any patient with a high-mechanism blunt trauma. Findings on chest radiograph can include a widened mediastinum, an obscured aortic knob, or hemothorax, but these findings are not always present. CTA findings include contrast extravasation, dissection, pseudoaneurysm, an intimal flap, aortic thrombus, and periaortic hematoma. Transthoracic echocardiogram can be performed if the patient is going urgently to the operating room for other findings.

37. How are blunt aortic injuries treated?

Repair with endovascular or open surgical techniques is recommended. Until repair is accomplished, strict blood pressure control should be maintained with short-acting agents, such as esmolol or nicardipine. Goals of hemodynamic control include a heart rate less than 100 beats per minute and a systolic blood pressure of 100 mm Hg.

38. How do penetrating injuries to the great vessels present?

The great vessels include the aorta; the subclavian, axillary, and pulmonary arteries and veins; and the superior and inferior vena cavae. Hard signs of vascular injury indicating need for emergent repair include pulsatile bleeding, an expanding hematoma, absent distal pulses, a cold limb, or cardiovascular collapse. Hemothorax, hemoperitoneum, tamponade, or hemoptysis may be present. Great vessel injuries can present with exsanguination, leading to a loss of vital signs. In these cases, resuscitative thoracotomy is indicated for direct occlusion of the injured vessel.

39. How are great vessel injuries evaluated, and what is the treatment?

CTA is the best study to evaluate great vessel injuries in stable patients. CTA may demonstrate contrast extravasation, an intimal flap, a pseudoaneurysm, or hematoma. Patients may be treated with endovascular or open operative techniques. Unstable patients should be taken directly to the operating room for definitive surgical repair

40. What are the goals of ED resuscitative thoracotomy?

A resuscitative thoracotomy is a life-saving procedure that is performed when a patient loses vital signs after trauma. Goals of resuscitative thoracotomy include:

- Decompress a tamponade.
- Maintain cerebral perfusion pressure with aortic occlusion as a means to decrease circulating volume.
- Gain control of intrathoracic hemorrhage.
- Perform effective cardiac massage.
- Temporarily control intraabdominal hemorrhage with aortic occlusion.

41. What are the contraindications for ED resuscitative thoracotomy?

The contraindications for ED resuscitative thoracotomy include:

- Prehospital cardiopulmonary resuscitation exceeds 10 minutes after blunt trauma with no signs of life.
- Prehospital cardiopulmonary resuscitation exceeds 15 minutes after penetrating trauma with no signs of life.
- Asystole is the presenting rhythm in the absence of pericardial tamponade.

42. Which is the more common mechanism for thoracic esophageal injury: blunt or penetrating?

Penetrating injury more commonly causes esophageal injury. Blunt injury to the esophagus is rarely seen, likely because of its relatively protected location in the posterior mediastinum.

43. What are the signs and symptoms of esophageal injury and rupture?

Symptoms are difficult to ascribe specifically to the esophagus, and commonly patients with esophageal injury are multiply injured and may be obtunded, but pain in the chest is a likely symptom. Signs of inflammation and air around the esophagus may be seen on imaging. A unilateral or bilateral pleural effusion may be present. Unexplained pneumomediastinum is suggestive and should be further investigated. After penetrating trauma, a trajectory in proximity to the esophagus should raise the index of suspicion for an esophageal injury. A chest tube placed for pneumothorax or hemothorax may drain gastric contents.

44. How should a suspected thoracic esophageal injury be investigated?

The ideal study for diagnosis is a Gastrografin swallow study. If this study is normal, a repeat evaluation using thin barium may be performed if a question persists. Suggestive findings on thoracic CT scan include pneumomediastinum, periesophageal air, and a new plural effusion. If a patient must go to the operating room emergently, direct esophagoscopy is helpful in diagnosis, but should not replace a formal swallow study when it can be done.

45. What is the treatment of esophageal injury?

The treatment of esophageal injury is drainage and prompt surgical repair.

46. Why are diaphragm injuries important to recognize?

A diaphragm injury occurs after a sudden increase in intraabdominal pressure against a fixed diaphragm, which leads to a tear. Diaphragm injury is three times more common on the left because of the protection afforded by the liver. A tear in the diaphragm may allow abdominal contents to herniate into the chest. These injuries have potential for high morbidity and mortality when herniated intraabdominal organs torse, strangulate, or perforate.

47. How is a diaphragm injury diagnosed?

A diaphragm injury can be clinically silent with no physical findings or symptoms. Chest pain and shortness of breath may be present. In larger injuries, a chest radiograph may demonstrate a gastric bubble in the chest, a nasogastric tube above the diaphragm, or intraabdominal contents above the diaphragm. Smaller defects may have no radiographic findings. When in doubt, definitive diagnosis should be made with laparoscopy or thoracoscopy.

48. What are the manifestations of a chyle leak resulting from blunt thoracic trauma, and how do I confirm the diagnosis?

A chyle leak, usually resulting from trauma to the thoracic duct, can present as a painless supraclavicular mass, a cervical fistula, or as chylothorax. The diagnosis is made by sending a sample of the fluid for triglyceride level.

49. What are the signs and symptoms of rib fractures, and how are they diagnosed?

Signs and symptoms of rib fractures include pain and tenderness over the fracture, referred pain to the fracture site upon compression of the fractured rib away from the fracture, mobility of the ribs, boney crepitance, and bruising over the ribs. Chest radiography and CT scan are the imaging studies of choice to confirm the diagnosis.

50. Name the potential complications after rib fractures.

- Pneumonia
- Empyema
- Hemothorax
- Ventilator dependence
- Chronic pain
- Decreased exercise tolerance

51. How are rib fractures treated?

Rib fractures are treated with supportive care, including pain control and pulmonary toilet. Major morbidity comes from inadequate pain control with subsequent impairment of pulmonary toilet, resulting in pneumonia. Multimodality pain therapy can be beneficial in these patients. Narcotics, nonsteroidal antiinflammatory drugs (NSAIDs), and muscle relaxants may all be of benefit. For patients with multiple rib fractures, local and regional anesthetic techniques, including epidural and paracostal pain catheters, can be used.

52. What are the risk factors for increased morbidity and mortality from rib fractures?

Increasing age and increasing number of rib fractures are associated with morbidity and mortality. Underlying comorbidities also play a role in clinical significance of these fractures.

53. What is a flail chest?

A flail chest occurs when multiple contiguous ribs are broken in more than one location, causing a portion of the rib cage to be completely detached from the remainder of the bony thorax. Paradoxic motion occurs with respiration. Nearly half of the patients with flail chest require mechanical ventilation, because the flail is often associated with significant underlying pulmonary contusion.

54. What is the treatment of choice for flail chest?

Similar to isolated rib fractures, treatment goals include maximizing pain control and pulmonary toilet. Ongoing investigations into surgical rib fracture stabilization suggest a benefit in repairing certain fracture patterns, but this has not been fully demonstrated at this time.

KEY POINTS: RIB FRACTURES AND FLAIL CHEST

1. Rib fractures are a common injury after thoracic trauma.
2. Diagnosis starts with the physical examination, with signs and symptoms including crepitance, chest wall instability, tenderness, or bruising over the affected rib.
3. A flail chest occurs when multiple contiguous ribs are broken in more than one place, causing a portion of the rib cage to be detached from the remainder of the bony thorax.
4. Treatment for both entities includes pain control, supplementary oxygenation, and aggressive pulmonary toilet.
5. Advanced age and multiple rib fractures are risk factors for morbidity and mortality.

55. What injuries are associated with a posterior sternoclavicular dissociation and scapular fracture?

Posterior sternoclavicular dissociation may cause injury to the great vessels, the trachea, and the esophagus. Injuries associated with a scapular fracture include hemopneumothorax, a lung contusion or laceration, spinal fractures, subclavian vessel injury, and brachial plexus injury.

56. What is the significance of a sternal fracture, and how is it diagnosed?

Similar to first and second rib fractures, sternal fractures are considered markers of high-energy transfer and should raise the concern for undiagnosed intrathoracic injuries. Sternal fractures may be seen on lateral chest radiography but are commonly diagnosed with CT scan.

57. What is the imaging modality of choice for suspected thoracic spinal injuries?

Thoracic CT scan is more sensitive than plain films in identifying thoracolumbar spine fractures. Patients with back pain, tenderness, altered mental status, distracting injuries, or high-energy mechanisms should undergo a thoracic CT scan with reconstruction of the spine. Imaging of the thoracic spine should be performed in patients with a known cervical or lumbar fracture to rule out a concomitant thoracic spine fracture.

58. What is neurogenic shock, and how does it manifest?

Neurogenic shock occurs after a cervical or high thoracic injury and causes impairment of sympathetic stimulation of the heart and periphery, with resulting vasodilation, hypotension, and absence of compensatory tachycardia. Extremities are warm secondary to the peripheral vasodilation. Management is fluid optimization with subsequent use of a peripheral vasoconstrictor, such as dopamine or norepinephrine, as needed.

59. How does thoracic trauma in children differ from thoracic trauma in adults?

Children are different from adult trauma patients in many ways. Hemodynamically, they are able to maintain normal vital signs in the face of significant injury for a longer time. Tachycardia and hypotension are late findings that may quickly proceed to hemodynamic collapse and arrest. An increased probability for tension pneumothorax results from the increased mobility of the pediatric mediastinum. Pediatric bones are more pliable, so any visible rib fractures indicate a high-energy force, and conversely, the absence of rib fractures does not preclude underlying organ injury. Surface area is proportionally larger in a child; therefore children have a greater predisposition for hypothermia.

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QUESTIONS

1. Which is not appropriate in the initial management of a stable patient with a massive hemothorax?
   a. Needle thoracostomy
   b. Establish an airway
   c. Establish IV access
   d. Tube thoracostomy and measure output
   The correct answer is *a*.
2. What is the appropriate treatment of an open pneumothorax?
   a. Suture the open wound
   b. Needle decompression
   c. Cover the wound with a three-sided dressing and place a chest tube
   d. 100% oxygen via face mask
   The correct answer is *c*.
3. At which anatomic location is the aorta most commonly injured?
   a. The ligamentum arteriosum
   b. The diaphragm
   c. The aortic root
   d. After the takeoff of the left carotid artery
   The correct answer is *a*.

ABDOMINAL TRAUMA

Alexander P. Morton, MD, and Ernest E. Moore, MD

CHAPTER 88

1. What is ABCDE, and why is it relevant to the evaluation of significant abdominal trauma?

Airway
Breathing
Circulation
Disability
Exposure

This acronym represents the primary survey employed to identify and treat the most life-threatening injuries in a systematic fashion. Airway, breathing, circulation, disability, and exposure are the important elements in the initial evaluation of a trauma patient. Circulation involves evaluating hemodynamics and identifying active hemorrhage. Persistent hemodynamic instability in the setting of abdominal trauma is an indication for emergent laparotomy. Exposure and examination of the abdomen, pelvis, back, buttock, and perineum may yield important findings, including penetrating trauma, active hemorrhage, evidence of significant blunt trauma (e.g., seat belt sign, unstable pelvis), abdominal tenderness, or diffuse peritonitis. Significant disability (i.e., neurologic injury) can render the clinical examination less effective and thus warrant more diagnostic studies.

2. Discuss the key aspects of the secondary survey in the evaluation of abdominal trauma.

The secondary survey is in essence a thorough history and complete physical examination, important in establishing the sequence and extent of early diagnostic efforts. Prehospital providers can relay invaluable information regarding the mechanism and force of the accident or injury, time from injury, use of seat belts, air bag deployment, ejection from a vehicle, drug and alcohol use, and the trend in a patient's clinical status (getting better versus getting worse). 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 anteriorly or tip of the scapula posteriorly. With significant injury, abdominal tenderness and guarding may be early signs, but rebound tenderness and rigidity are relatively uncommon. Most important, 20% to 40% of patients with serious intraabdominal injury may appear asymptomatic. Imaging during the secondary survey should include repeated focused assessment with sonography for trauma (FAST) examinations of the chest and abdomen. Chest and pelvis radiographs are often included in cases of major blunt trauma, but close proximity of a computed tomography (CT) scanner may negate the need for these studies.

3. What are some of the biomechanical principles in blunt and penetrating trauma?

Trauma represents the result of the transfer of kinetic energy from the colliding object (e.g., bullet, car) to the patient. The severity and location of blunt trauma depends on the type of impact (e.g., compression versus shear forces), momentum of the involved objects, and properties of the affected tissues (Fig. 88-1). In penetrating trauma, damage is caused by the dissipation of energy as the knife, bullet, or other object traverses tissues. The injury pattern is dependent both on the momentum of the projectile and its trajectory through the body. Because of significantly increased momentum and unpredictable intracavitary trajectories, gunshot wounds can produce extensive damage with unexpected injury patterns when compared with stab wounds. Therefore penetrating abdominal gunshot wounds typically require laparotomy, and maintenance of a broad differential for sources of intraabdominal hemorrhage or injury; whereas stab wounds can often be managed selectively (Fig. 88-2).

Abstract

This chapter discusses the management of blunt and penetrating abdominal trauma in the ED. Key topics include the initial assessment, physical examination, imaging modalities, indications for emergent laparotomy, and information on unique patient populations in trauma.

Keywords:

trauma, abdomen, penetrating trauma, blunt trauma, stab wound, gunshot wound, laparotomy, focused assessment with sonography for trauma (FAST)

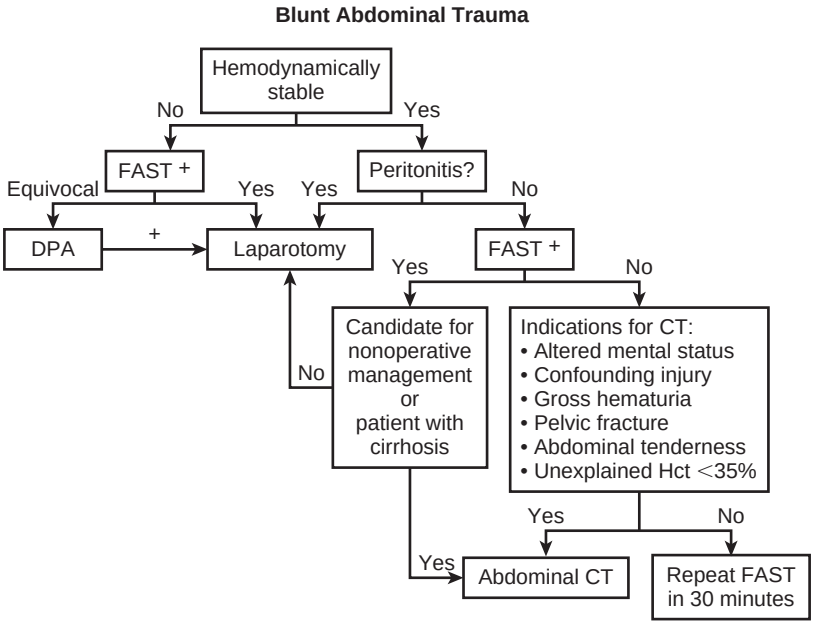


Figure 88-1. An algorithm for the management of blunt abdominal trauma. Peritonitis and hemodynamic instability with positive FAST or DPA are indications for immediate laparotomy. *CT,* Computed tomography; *DPA,* diagnostic peritoneal aspiration; *FAST,* focused assessment with sonography for trauma; *Hct,* hematocrit.

KEY POINTS: INDICATIONS FOR LAPAROTOMY

1. Diffuse peritonitis
2. Hemodynamic instability with evidence of abdominal injury
3. Penetrating gunshot wounds with peritoneal violation
4. Penetrating stab wounds with evisceration

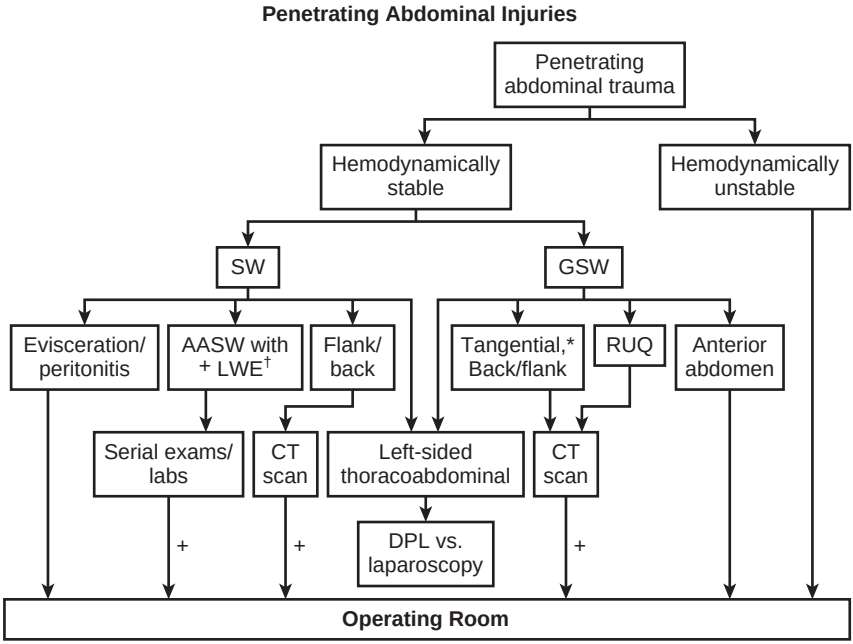
4. What are the most commonly injured abdominal organs?
The liver and spleen are the most commonly injured abdominal solid organs. The stomach and small bowel are the most commonly injured abdominal hollow viscera.

5. What is a seat belt sign?
It is an ecchymotic imprint of the seat belt or shoulder strap on the anterior chest or abdomen of a restrained passenger, indicating rapid deceleration from a motor vehicle crash. The presence of a seat belt sign is associated with a 20% incidence of intraabdominal injury.

6. Lower rib fractures are typically associated with what intraabdominal injuries?
Lower rib fractures are associated with injuries to the liver and spleen.

7. What is a Chance fracture?
A Chance fracture is a transverse fracture of a low thoracic or lumbar vertebra that is caused by flexion of the back, and is associated with two-point seat belt use. The incidence of associated intraabdominal injuries with a Chance fracture approaches 50%, and includes the small bowel and abdominal aorta.

8. What abdominal injuries are associated with pelvic fractures?
Pelvic fractures are associated with injuries to the solid (11%) and hollow (4%) intraabdominal organs, the diaphragm (2%), and the bladder and urethra (6%).



*Tangential GSWs may also be evaluated with diagnostic laparoscopy.
†A positive local wound LWE is defined as violation of the peritoneum.

Anterior abdomen: From inguinal ligaments to costal margin, from anterior axillary line to anterior axillary line
Flank/back: From anterior axillary line around posteriorly to opposite anterior axillary line below costal margin and above pelvis
LWE: Positive if there is peritoneal penetration
Serial examinations: Documented by upper level every 2 hours
Consider CT A/P if Hgb drops without peritonitis in stable patient
Consider CT A/P if RUQ SW to evaluate isolated liver injury

Figure 88-2. An algorithm for the management of penetrating abdominal trauma. Penetrating trauma with hemodynamic instability, anterior abdominal gunshot wounds, and abdominal stab wounds with evisceration are indications for immediate laparotomy. *AASW,* Anterior abdominal stab wound; *A/P,* abdomen/pelvis; *CT,* computed tomography; *DPL,* diagnostic peritoneal lavage; *GSW,* gunshot wound; *Hgb,* hemoglobin; *LWE,* local wound exploration; *RUQ,* right upper quadrant; *SW,* stab wound.

9. In the setting of trauma, what is the significance of gross hematuria?

Hematuria suggests injury to the genitourinary system, including the kidneys, ureters, bladder, or urethra. Physical examination can provide key information regarding the location of injury, such as flank ecchymosis, penetrating injuries near the kidneys or bladder, perineal laceration, or a high-riding prostate. Important diagnostic modalities include retrograde cystourethrography to identify bladder rupture and urethral injuries, as well as CT with contrast, including the excretory phase to identify renal and ureteral injuries.

10. Describe the incidence of diaphragmatic rupture in trauma and how it can be diagnosed on a chest radiograph.

Diaphragm injuries occur in 1% to 7% of blunt trauma and 10% to 15% of penetrating trauma. A displaced nasogastric tube representing the stomach through the left hemithorax reveals a diaphragm rupture. However, the chest radiograph is normal in up to half of patients with left diaphragmatic injury and is often normal with right-sided injuries. Left-sided injuries are more

common than right-sided injuries, because the liver absorbs more energy during right-sided injuries, protecting the diaphragm. High clinical suspicion for diaphragm injury should be maintained in the setting of penetrating thoracoabdominal trauma.

11. Does a normal serum amylase test exclude pancreatic injury?

No, the initial serum amylase test is neither a sensitive nor specific test for pancreatic injury (i.e., a normal amylase result does not exclude pancreatic injury), and an elevated amylase may be the result of an increase in salivary amylase. Pancreatic injuries are found in 3% to 6% of patients undergoing laparotomy for trauma. Because of its close association with many vital structures, pancreatic injury is associated with other injuries in more than 90% of cases.

KEY POINTS

1. Certain injuries should increase clinical suspicion for intraabdominal injury after blunt trauma, including a seat belt sign, lower rib fractures, major pelvic fractures, and Chance fractures.
2. The most commonly injured solid organs are the liver and spleen. The most commonly injured hollow viscera are the stomach and small bowel.
3. Gross hematuria is a sign of genitourinary trauma, including the urethra, bladder, ureters, and kidneys.

12. What is the initial imaging modality of choice to evaluate for evidence of abdominal trauma?

FAST is 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 intraabdominal fluid. If the test is initially negative, repeating the examination in an unstable patient is imperative because more than 250 mL of blood must accumulate within the Morrison pouch before a fluid stripe will appear on the FAST test. A single negative FAST test cannot rule out abdominal injury.

13. What are the four locations evaluated during FAST, and in which order should they be evaluated?

1. Pericardium: Presence of pericardial fluid
2. Right upper quadrant: Morrison pouch (most common location of positive FAST, regardless of injury location)
3. Left upper quadrant: Splenorenal space and subphrenic space
4. Suprapubic area: Bladder and rectovesical space

14. What is the role of CT scanning?

Abdominopelvic CT is the test of choice for evaluating the abdomen of patients with significant blunt abdominal trauma who are hemodynamically stable. Indications include signs or symptoms of abdominal injury, patients with significant disability or distracting injury (i.e., femur fracture), and injuries detected on diagnostic peritoneal lavage (DPL), FAST, or radiograph. Abdominal CT serves a major role in the decision to manage the injured spleen, liver, or kidney nonoperatively by allowing for injury grading and identification of active arterial extravasation.

15. What is the role of DPL?

The major advantage of DPL is a sensitivity rate greater than 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. DPL is used predominantly if the FAST results are negative but there is no other source to account for a patient's acute blood loss. The DPL is often done without the infusion of fluid (i.e., diagnostic peritoneal aspirate [DPA]). If the patient is hemodynamically unstable because of intraabdominal hemorrhage, a gross blood sample should be retrieved on insertion of the catheter.

16. How are DPL results interpreted?

DPA is considered positive if more than 10 mL of free blood or any enteric contents are 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. The test is positive for the following results:

- Greater than 100,000 RBC/mL
- Greater than 500 WBC/mL
- Greater than 175 IU/mL amylase
- Any bile, bacteria, or food particles

A positive DPL is an indication for laparotomy.

KEY POINTS: DIAGNOSTIC TOOLS FOR BLUNT TRAUMA

1. FAST examination should be done as soon as possible and repeated.
2. CT scanning is preferred in the hemodynamically stable patient, even if there is a positive FAST test.
3. DPL is useful to exclude major abdominal hemorrhage if the FAST test is normal.
4. Serial physical examination is an important aspect of initial management.

17. What are the unique concerns in a pregnant patient with abdominal trauma?

Physiologic changes occur in the pregnant patient, including hypervolemia, a decrease in peripheral vascular resistance, venous return, and blood pressure, which can mask signs of shock or lead to supine hypotensive syndrome. Pregnant patients' blood becomes hypercoagulable, leading to increased risks of venous thromboembolism, and they have displacement of their abdominal organs, making the physical examination less reliable. Optimal care of the mother ensures the best outcome for the fetus; therefore the primary survey should focus on her care. It is important to place the patient in left lateral tilt position to avoid or relieve caval compression and impaired venous return. After the primary survey is complete, continuous noninvasive fetal monitoring should be used if the fetus is greater than 24 weeks' gestational age to evaluate for signs of fetal distress. Conservative management of blunt abdominal injuries is the treatment of choice in stable, pregnant trauma patients. Hemodynamic instability, uterine rupture, placental abruption, and other injuries requiring immediate repair are indications for abdominal exploration. Patients requiring laparotomy have an increased risk of preterm labor that increases with gestational age.

18. What are the general principles of trauma in the elderly population?

The combination of chronic medical conditions, limited organ reserve, and atherosclerosis 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. Age-related cardiac dysfunction can result in a relatively fixed cardiac output and heart rate, leading to vasoconstriction as the sole response to hypovolemia.

Because of the masking of traditional signs of hemorrhagic shock, this group is more susceptible to failure of conservative management and has a higher mortality rate than younger patients with similar injuries. Therefore a high index of suspicion for intraabdominal hemorrhage must be maintained with the understanding that traditional markers of hypovolemia and failure of conservative management may not be accurate.

19. In the management of abdominal trauma, are children really just small adults?

No, injury patterns are different in children because of their size and tissue elasticity. They can have minimal external signs of injury, even in the setting of significant blunt trauma and intraabdominal injuries. A thin abdominal wall and close proximity of organs leaves children at increased risk of multiple organ injuries even from a single blow. External signs of injury, such as a seat belt sign or abdominal tenderness, significantly increase the likelihood of abdominal injury. Although blunt injuries to solid abdominal organs tend to be self-limited in children, those with hemodynamic instability or ongoing/progressive blood loss should undergo operative management. Additionally, pediatric blood pressure and heart rate can be falsely reassuring while compensatory mechanisms remain intact. Hemodynamic instability can be masked until hemorrhagic shock becomes severe and cardiovascular collapse occurs. As with any pediatric injury, clinical suspicion for abuse should be maintained in the setting of a discrepant history or a physical examination that shows signs of abuse, such as bruises in multiple stages of healing, evidence of old fractures, or abdominal organ injury without a history of abdominal trauma.

KEY POINTS: ABDOMINAL TRAUMA

1. Patients who have persistent hemodynamic instability or peritonitis after abdominal trauma require emergent laparotomy.
2. A detailed history and physical examination are key elements in the evaluation of the lucid trauma patient.
3. A single 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. Pregnant, elderly, and pediatric trauma patients have unique anatomy and physiology that affects injury patterns, physiologic response to trauma, and treatment algorithms.

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QUESTIONS

1. Which of the following is not an indication for emergent laparotomy?
   a. 35-year-old male in a motor vehicle crash with a seat belt sign and diffuse peritonitis on examination
   b. 72-year-old female victim of an auto-pedestrian accident with normal chest and pelvis radiographs, abdominal tenderness, and a systolic blood pressure of 80 mm Hg
   c. 50-year-old male with a stab wound to the anterior abdomen and a normal systolic blood pressure and heart rate, and normal FAST test
   d. 21-year-old male with a gunshot wound to the anterior abdomen and a normal systolic blood pressure and heart rate, and positive FAST test

   The correct answer is *c*. The indications for emergent laparotomy are diffuse peritonitis, hemodynamic instability (e.g., systolic blood pressure <90), abdominal gunshot wound with peritoneal violation, or abdominal stab wounds with evisceration. In this case, the patient in scenario *c* has a stab wound without evidence of evisceration, intraabdominal fluid, peritonitis, or hemodynamic instability, and can initially be managed conservatively.
2. Where is fluid most likely to accumulate and be visible on FAST examination?
   a. Pericardium
   b. Right upper quadrant
   c. Left upper quadrant
   d. Posterior to bladder

   The correct answer is *b*. The Morrison pouch (the right upper quadrant) is the most common place for a positive FAST examination, regardless of injury location. Greater than 250 mL of blood must accumulate before it can be visualized on FAST. A single negative FAST examination does not rule out intraabdominal injury.
3. The following DPL results are considered positive except:
   a. 5500 RBC/mL
   b. 550 WBC/mL
   c. 200 IU/mL amylase
   d. Positive bile

   The correct answer is *a*. Positive DPL criteria are greater than 500 WBC/mL, greater than 100,000 RBC/mL, greater than 175 IU/mL amylase, or any bile, bacteria, or food particles. Aspiration of greater than 10 mL of frank blood before infusion of 1 L normal saline is also considered a positive test.

CHAPTER 89

PELVIC FRACTURES AND GENITOURINARY TRAUMA

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 of airway, breathing, and circulation (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 trauma surgeon and orthopedic surgeon in the ED is warranted (Fig. 89-1).

3. How do I examine the patient with a pelvic fracture?

Examine the patient very carefully. The physical examination of 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 can lead 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 radiography (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

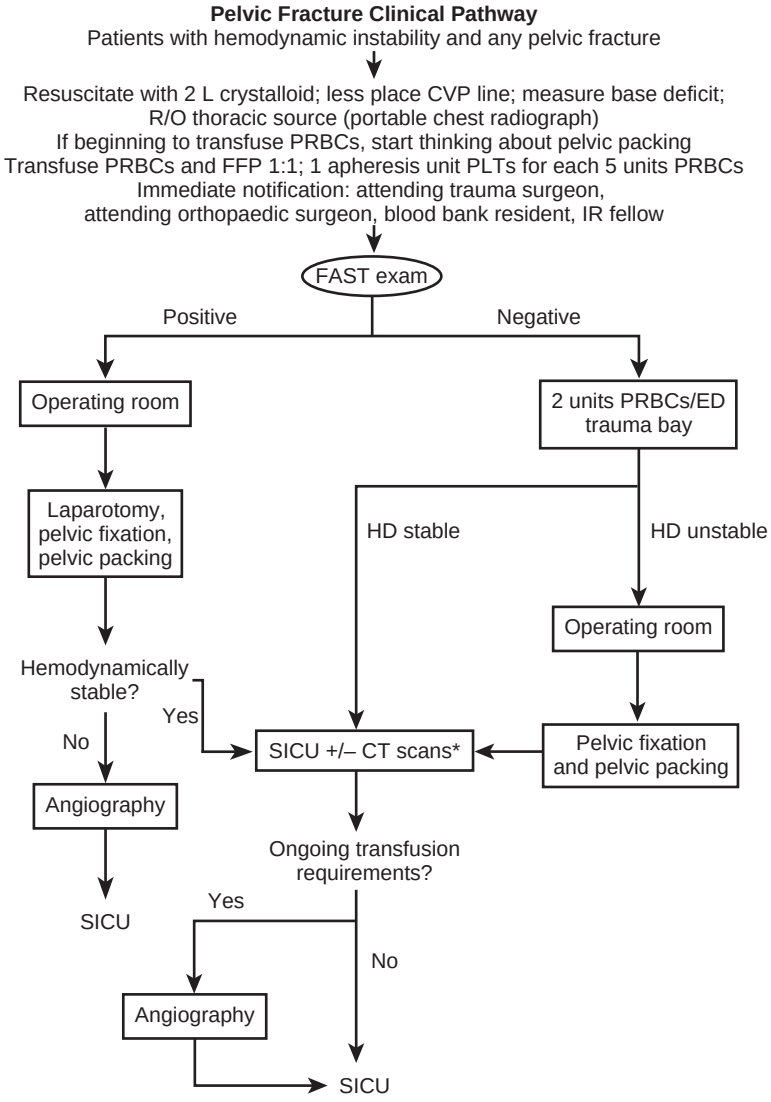
Vertical shear injuries consist of displaced fractures of the anterior rami and posterior columns, including SI dislocation.

Abstract

Pelvic fractures are potentially lethal. In the unstable patient, multidisciplinary management with critical decision making is the key to survival.

Keywords:

pelvic fracture, pelvic packing, angioembolization, genitourinary trauma, renal injury, bladder injury, urethral injury



*Normalize coagulation status, consider factor VIIA if recalcitrant.
Abdominal CT scan if no laparotomy done.

Figure 89-1. Management of patients with a pelvic fracture. *CT,* Computed tomography; *CVP,* central venous pressure; *FAST,* focused assessment with sonography for trauma; *FFP,* fresh-frozen plasma; *HD,* hemodynamically; *IR,* interventional radiology; *PLT,* platelet; *PRBC,* packed red blood cell; *R/O,* rule out; *SICU,* surgical intensive care unit.

5. What are the sources of bleeding from major pelvic fractures?

The most common 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 symphyseal 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.

6. Name three goals of mechanical pelvic stabilization.

1. Reduce pelvic volume.
2. Promote tamponade of bleeding bone and vessels.
3. Prevent further fracture motion.

7. Discuss four methods of acute pelvic stabilization.

1. Wrapping the pelvis: This intervention should be performed immediately on discovery of an unstable pelvic fracture, particularly before patient transport. It may be accomplished with a bedsheet; alternatively, a proprietary device (e.g., T-Pod pelvic binder) may be used. It is important to wrap at the level of the greater trochanter of the femur. Binding at the level of the knees and ankles may further compress the pelvic volume. Prolonged use of pelvic binding may result in extremity or abdominal compartment syndrome.
2. Anterior external fixation: This is the standard intermediate-term 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.
3. Pelvic C-clamp: This intervention is more effective than a standard anterior frame in stabilizing the posterior pelvis.
4. 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 used 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 that is 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. 89-1).

9. How often are rectal injuries associated with pelvic injuries, and 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. 89-1). 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. If a laparotomy is needed, it may be performed rapidly. More prospective studies are needed for this to gain wide acceptance in the United States.

KEY POINTS: APPROACH TO PATIENTS WITH A PELVIC FRACTURE

1. Mechanical stabilization of the pelvis, reversal of shock, and correction of coagulopathy are critical early steps to avoid exsanguination.
2. Associated injuries are common and should be diagnosed promptly.
3. Intraabdominal bleeding must be excluded before sending a patient to interventional radiology.
4. Prompt, definitive decision making is the key to survival.

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 and 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 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 angiographic 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 intraabdominal 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 too has 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 caused by lap belt or steering wheel injuries. Gross hematuria is present in more 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 to 30 mL) is injected under gentle pressure as the anteroposterior and oblique views are taken.

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 monitoring 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 penile fracture is 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.

KEY POINTS: UROLOGIC TRAUMA

1. Renal injury is the most common urologic trauma.
2. Renal pedicle injury can lead to uncontrolled hemorrhage or ischemia.
3. Gross hematuria or persistent microhematuria warrant evaluation.
4. Urologic injury may be present in the absence of hematuria.

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QUESTIONS

1. Which of the following fracture types is least likely to be associated with exsanguinating hemorrhage?
   a. Tile C
   b. APC III
   c. LC I
   d. Vertical shear
   The correct answer is *c*.
2. All of the following are considered appropriate methods of acute pelvic stabilization in unstable patients except:
   a. Pelvic binding with sheet
   b. Anterior external fixation device
   c. Open reduction and internal fixation of pelvis
   d. Pelvic C-clamp
   The correct answer is *c*.
3. All of the following are true regarding genitourinary injuries except:
   a. Hematuria may be absent when the ureter is completely transected.
   b. Gross hematuria is present in greater than 95% of patients with bladder rupture.
   c. The ED management of complete urethral disruption is placement of a large Foley catheter.
   d. Asymptomatic microscopic hematuria is not a good predictor of genitourinary tract injury.
   The correct answer is *c*.

CHAPTER 90

TRAUMA IN PREGNANCY

Jedd Roe, MD, MBA, FACEP

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 7% of pregnancies are complicated by trauma. In blunt abdominal trauma, the usual causes are falls (52%), motor vehicle accidents (MVAs; 34%), and intentionally inflicted injuries (9.5%). Immediate complications are seen in 3.2% of pregnant trauma patients, most commonly preterm labor and placental abruption. 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' gestation. In hospitalized fall patients, there are eight times the number of placental abruptions, and more than four times as many women experience preterm labor.

3. **Is physical or sexual abuse often seen 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, and the maternal and fetal mortality rates associated with domestic violence are 3% and 16%, respectively.

4. **Given the impact of domestic violence, what can be done in the ED?**
   Effective screening is essential and can be done in an abbreviated fashion. One 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).

5. **What are the implications of MVC mechanisms of injury for pregnant patients?**
   MVCs are one of the leading causes of maternal and fetal mortality, and 87% of pregnant women in an MVC receive medical care. One of the major risk factors for adverse outcomes is improper seat belt placement, and only half of patients reported receiving prenatal counseling from their provider regarding appropriate seat belt placement. Another risk factor is the use of alcohol and other intoxicants, because 40% to 45% of pregnant patients in an MVC test positive for such substances. Placental abruption may be seen in up to 8.5% of uninjured pregnant women involved in an MVC, and 13% of those who are severely injured.

6. **How do physiologic changes in pregnancy affect the evaluation of the trauma victim?**
   Whereas decreasing blood pressure and rising heart rate might indicate hypovolemic shock in a nonpregnant woman, 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.

Abstract

There are unique aspects to managing trauma in a pregnant woman, because there are two lives to consider. The possible injuries to the fetus and mother are discussed in this chapter.

Keywords:

placental abruption, fetomaternal hemorrhage, perimortem cesarean section, preterm labor, cardiotocographic monitoring (CTM), domestic violence

7. 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 hematocrit levels of 32% to 34% by the third trimester. Fibrinogen levels are double those seen in other trauma patients. Therefore disseminated intravascular coagulation (DIC) may be seen with normal fibrinogen levels. Because of hormonal stimulation of the central respiratory drive, the partial pressure of carbon dioxide (PCO_2) falls to 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.

8. 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.

9. Name the most common causes of fetal death.

- Maternal death
- Maternal shock
- Placental abruption

10. How does placental abruption occur?

Abruption results from the separation of the relatively inelastic placenta from an elastic uterus secondary 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.

11. What are the findings of abruption after trauma?

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 for a serum fibrinogen level, with low levels being the indication for obtaining a complete DIC panel.

12. How often does ultrasound detect placental abruption?

Because a large separation must be present for ultrasound to be diagnostic, it detects only about half of all cases. In many instances, fetal distress is present before the clear visualization of an abruption by ultrasound. The fetal mortality rate 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.

13. Are radiologic investigations harmful to the fetus?

The fundamental effects of radiation on the developing fetus are intrauterine growth retardation, defects in the central nervous system (microcephaly, mental retardation), and risk of cancer. The most vulnerable period is between 2 and 15 weeks' gestation. Cumulative exposure of less than 5 rads (50 mGy) during pregnancy has not been shown to affect the outcome of pregnancy compared with control populations. In general, all necessary radiographic studies should be undertaken with appropriate fetal shielding. All clinically indicated studies should be done regardless of any radiation concerns. Furthermore, there have been no reported adverse effects on neonatal thyroid function with the use of iodinated contrast, and it should be administered if absolutely necessary. Evaluation should begin with the nonradiographic alternative of ED ultrasound (focused assessment with sonography for trauma [FAST]) to rapidly determine the presence of intraperitoneal hemorrhage, pericardial effusion, or pneumothorax. See Chapter 9 for radiation exposures from diagnostic imaging studies.

14. 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.

15. What are the priorities for ED management?

The prehospital therapies mentioned previously should be continued. Of particular importance 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 or blood, opening of the cervical os, and genital tract trauma. Continued aggressive resuscitation with warmed lactated Ringer solution (less acidotic, more physiologic than normal saline) and blood is especially important, given the physiologic changes mentioned previously.

16. How do I begin to evaluate the fetus?

First, determine the size of the uterus and the presence or absence 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.

17. What is fetomaternal hemorrhage (FMH)?

FMH is 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 Rhesus factor (Rh) sensitization, fetal anemia, and fetal death can occur. Laboratory techniques are not sensitive enough to diagnose FMH accurately.

18. How is FMH managed?

The prudent course is to give Rh immunoglobulin (RhIG) to all Rh-negative patients who are suspected of having abdominal trauma, because RhIG given within 72 hours of antigenic exposure prevents Rh isoimmunization. 50 μg of RhIG is the dosage used in the first trimester, and this is increased to 300 μg after 12 weeks' gestation. 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, although in the setting of massive transfusion, the KB test can be used to determine whether the patient requires more than 300 μg of RhIG.

19. When is emergency 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 g, are predicted to have a 50% survival rate in the neonatal intensive care unit (NICU) 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.

20. When should perimortem cesarean section be performed?

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 30 minutes after maternal decompensation. Delivery of the near-term fetus improves maternal cardiac output by 30% to 80%, and emptying the uterus also improves the effectiveness of CPR. Thus maternal outcomes have been shown to improve with perimortem delivery as well.

21. Which pregnant patients with abdominal trauma require admission for fetal monitoring?

Any viable (>23 to 24 weeks' gestation) 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 contractions, amniotic membrane rupture, vaginal bleeding, serious maternal injury, significant abdominal pain, and nonreassuring fetal heart rate variability, 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

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QUESTIONS

1. Which of the following is not a physiologic change commonly seen with pregnancy?
   a. Increased oxygen demand
   b. A fall in the PCO_2 to 27 to 32 mm Hg
   c. Hemoconcentration, manifested by a hematocrit level of 47 to 49
   d. An increase in maternal blood volume of 50%
   The correct answer is *c*.
2. Which is the most appropriate statement with regard to placental abruption?
   a. Patients usually present with vaginal bleeding or uterine tenderness.
   b. A major mechanism of injury is needed to cause an abruption.
   c. Fetal distress may be the only presenting sign of abruption.
   d. Ultrasound is highly sensitive and accurate in detecting abruption.
   The correct answer is *c*.
3. Which is an important consideration with respect to radiologic investigations and the pregnant trauma patient?
   a. Ultrasound should be avoided because of its low-resolution images.
   b. Cumulative exposure of 10 rads (100 mGy) during pregnancy has not been shown to affect the outcome of pregnancy when compared with controls.
   c. Iodinated contrast should be avoided because of its effect of the neonatal thyroid.
   d. All clinically indicated studies should be done regardless of any radiation concerns.
   The correct answer is *d*.

CHAPTER 91

PEDIATRIC TRAUMA

Mariah H. Bellinger, MD, and Patrick J. Maloney, MD, FACEP

1. Which children get injured? How do they do it?

Every year, nearly one in three children is injured. Fifty percent of all pediatric ED visits in the United States are related to trauma. Fortunately, most of these injuries are minor. However, according to the most recent data by the Centers for Disease Control and Prevention (CDC) in 2011, trauma is not only the leading cause of mortality in children older than 1 year, but trauma accounts for more deaths among children in the United States than all other causes combined. Motor vehicle collisions are the most common cause of trauma-related death in all age groups (about 50% overall), followed by drownings, house fires, homicides, and falls. A very common site of pediatric trauma is the home. Boys are injured twice as often as girls, and this disparity widens as children get older.

2. Are children just little adults?

No, unique anatomic characteristics in children require special consideration. This is especially true for infants, toddlers, and smaller children. Once a child reaches the age of about 12 years, the anatomy begins to resemble that of adults.

3. What are some of the anatomic differences?

- 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 the Waddell triad: a femur fracture, truncal trauma (i.e., intraabdominal or intrathoracic injury), and head injury, typically occurring after a child is struck by an automobile at high speed.
- Because of a greater head-to-body ratio, thinner cranial bones, and less myelinated brain tissue, intracranial injury is more common and often more debilitating than in older populations. In fact, head injuries are the leading cause of morbidity and mortality 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, make intraabdominal solid organ injuries more common.
- A child's incompletely calcified and thus more compliant skeleton allows internal organ damage without visible overlying fractures. This is most commonly seen with pediatric thoracic injuries. Children are much less likely to suffer rib fractures but much more likely to sustain pulmonary contusions.
- A high body surface area–to-volume ratio results in significant thermal energy loss and early hypothermia in a child.

4. How does prioritization of the resuscitation ABCs (airway, breathing, and circulation) differ between children and adults?

There is no difference in the prioritization of the ABCs when evaluating and managing children. The ABCs always take priority in that order, whether in an adult or a child. It is important to remember, however, that most traumatic arrests in children result from respiratory arrest. Therefore special attention should always be directed at a child's airway and breathing. In addition, recognizing hypovolemic shock in a child may be more difficult than in adults because hypotension is a late and ominous finding in children (see Question 8).

- Tachycardia may be the result of pain, anxiety, and other emotional factors in the injured child but should always alert the provider to possible blood loss and shock. A child's mental status, skin examination (including capillary refill time), and sometimes urine output are clues, in addition to serial vital signs, that help evaluate a child's hemodynamic status in the ED.
- 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.

Abstract

Fifty percent of all pediatric ED visits in the United States are related to trauma. Most injuries are minor, but trauma is the leading cause of death among children after the first year of life. Children are not just little adults, and providers must take into account specific anatomic and physiologic differences when evaluating pediatric injuries. Head injury is the leading cause of morbidity and mortality. Cervical spine injuries, while more rare, more often occur higher in the spine (C1 to C3) than in adults. Rib fractures are less common, but pulmonary contusions occur more often. The most common cause of death in pediatric trauma is respiratory arrest. Therefore special attention should be given to good airway and respiratory support. Hypotension is a late and ominous finding in the pediatric trauma patient. Tachycardia and signs of poor perfusion are earlier and more reliable clues.

Keywords:

trauma, injury, motor vehicle collisions, cervical spine injuries, respiratory arrest, pulmonary contusions, aortic injuries, nonaccidental trauma, concussion, toddler's fracture, greenstick fracture, torus fracture, buckle fracture, growth plate facture, spinal cord injury without radiographic abnormalities (SCIWORA), psuedosubluxation, line of Swischuk, Pediatric Emergency Care Applied Research Network (PECARN), intraosseous (IO) line, focused assessment with sonography for trauma (FAST) examination

- Hypotension is a very late finding in shock in a child. One way to quickly estimate the lower limit of normal blood pressure (lowest fifth percentile) in children of different ages is as follows:
  - Age younger than 1 month: Greater than 50 mm Hg
  - Age 1 month to 1 year: Greater than 60 mm Hg
  - Age older than 1 year: [70 + (2 × Age in years)] mm Hg

5. Which factors affect the patency of a child's airway?

There are a number of anatomic factors related to the child's airway that make children particularly prone to obstruction.

- 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, especially on a rigid spinal immobilization board. To align the oral, pharyngeal, and tracheal axes, a towel roll should 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 because they may induce vomiting.

6. Which factors affect endotracheal intubation of a child?

See Chapter 68.

7. What are my options if I cannot endotracheally intubate the patient's airway?

- When bag-valve-mask ventilation is inadequate or unsuccessful and endotracheal intubation fails or is not possible, a laryngeal mask airway (LMA) may be inserted by an experienced provider. It should be noted that there is an increased rate of complications, particularly upper airway obstruction, caused by the LMA folding the larger epiglottis into the larynx, when used in smaller children.
- In children older than 8 years, 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, the cricothyroid membrane is too small and structures are 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. Because of limitations in adequate ventilation with needle cricothyrotomy, the treating provider should immediately consult with a surgeon to perform an emergent tracheostomy.

8. How do I recognize shock in a pediatric patient?

Children have increased physiologic reserve and robust hemodynamic compensatory mechanisms. As a result, they often maintain a blood pressure in the normal range even in the presence of significant volume loss (this is referred to as *compensated shock*). Young children are less able to increase their cardiac contractility, and therefore maintain their cardiac output and blood pressure in the presence of blood loss by increasing their heart rate and systemic vascular resistance (SVR). For these reasons, poor skin perfusion (mottling of the skin, cool extremities, capillary refill greater than 2 seconds), decreased pulse pressure, increased work of breathing, and abnormal mental status (depressed level of consciousness or agitation) are more reliable signs of hemodynamic instability than blood pressure in a child. Hypotension is a very late and ominous finding in a child, and typically indicates a loss of at least 25% to 40% of blood volume and may be accompanied by bradycardia.

9. Name the preferred sites for venous access.

In decreasing order of preference:

- Peripheral
- Intraosseous (IO)
- Central venous (femoral, subclavian, or internal jugular veins)
- Saphenous vein cutdown at the ankle

In the unstable patient, IO line insertion should not be delayed by multiple peripheral attempts. Ultrasound guidance should be utilized for central venous line access, because its use results in a decreased number of access attempts, as well as fewer arterial punctures in the pediatric population.

10. What are some considerations regarding an IO line?

IO lines typically can be inserted easily and quickly. IO line insertion should be considered in any critically ill patient if a peripheral line cannot be established immediately. IO lines are safe for administration of virtually any fluid, blood product, or drug. The preferred site is the proximal tibia below the tibial tuberosity. Other potential sites include the distal femur, medial malleolus, proximal humerus, iliac crest, and sternum. IO lines should not be placed distal to a fracture and should be removed once peripheral or central intravenous access is secured. Complications include cellulitis, osteomyelitis, growth plate injury, fat microembolism, compartment syndrome, and iatrogenic fractures.

11. What is a child's normal blood volume?

Approximately 80 mL/kg

12. How should I resuscitate a pediatric trauma patient?

Once a child's airway has been evaluated and managed, and adequate oxygenation and ventilation has been confirmed, attention should quickly be focused on the injured child's circulatory status. Direct manual pressure should be applied to any sites of significant external bleeding. Special attention should be given to the back of the scalp, because this is a common site of significant bleeding. In general, an approximately 25% reduction in blood volume is required to manifest signs of compensated shock. Warmed crystalloid fluids (normal saline or lactated Ringer solution) should be given in 20 mL/kg boluses. After each bolus, the child's hemodynamic status should be reevaluated. The child with compensated shock will often require 40 to 60 mL/kg of crystalloid fluid. If the child continues to show signs of shock after crystalloid fluid administration, warmed packed red blood cells (10 to 20 mL/kg) should be transfused. There is a growing body of evidence suggesting that early blood product transfusion results in better outcomes. In children receiving large volumes of packed red blood cells (approaching half of their total blood volume), consideration should be given to utilizing a massive transfusion protocol in which packed red blood cells, platelets, and fresh frozen plasma are given in a 1 : 1 : 1 ratio.

13. Why are children prone to head trauma?

A child's head mass is disproportionately large compared with his or her body. When a child falls, the tendency is to fall head first. One way to remember this is to think: Children's bodies are like darts; they lead with their heads.

14. Which kinds of head injuries do children get?

Compared with adults, mass lesions (such as epidural and subdural hematomas) are less common, but cerebral edema and postinjury seizures are more common. Neonates and young infants have a relatively large intracranial volume, as well as open cranial bone sutures and fontanels. As a result, these very young children differ from most other children and adults in that they can actually develop hemorrhagic shock secondary to blood loss within the intracranial and subgaleal spaces. Bulging sutures or fontanelles suggest a significant brain injury and/or cerebral edema and warrant aggressive management and emergent neurosurgical consultation. 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 (see Chapter 66).

15. 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, whereas others are overly sensitive but lack specificity and, therefore, if used alone, result in increased rates of computed tomography (CT) utilization and unnecessary exposure to radiation. In an attempt to identify patients who can be safely discharged from the ED without imaging, the Pediatric Emergency Care Applied Research Network (PECARN) published a large series (more than 42,000 patients) describing children with minor head injuries in 2009.

- In patients younger than 2 years, the following characteristics gave a negative predictive value (NPV) of 100% (95% CI, 99.7% to 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, the following criteria generated an NPV of 99.95% (95% CI, 99.81% to 99.99%): normal mental status, no loss of consciousness, no vomiting, nonsevere mechanism, no signs of basilar skull fracture, and no severe headache.

16. How do I recognize a concussion in a child?

Concussions are generally defined as head injuries with some form of alteration in mental status without focal neurologic deficits. Concussions may occur with initial loss of consciousness after blunt head trauma, but children can sustain a concussion without ever losing consciousness. Initially, a child may have amnesia, headache, dizziness, nausea/vomiting, blurry vision, or confusion. Brain imaging is usually normal, but subtle diffuse axonal abnormalities may be seen on magnetic resonance imaging (MRI). The mainstay of management is "brain rest," in addition to symptomatic care of headaches and nausea. Long-term sequelae can include chronic headaches, cognitive impairment, disrupted sleep, and behavioral or psychological issues. The average length of symptoms from a concussion is 5 to 7 days, but in some children, especially younger children, symptoms may last weeks to months.

17. How do cervical spine injures in children differ from those in adults?

- Because the fulcrum of a child's cervical spine is higher (around C2 to C3) than adults (around C6 to C7), children tend to sustain high cervical injuries (C1 to C3) as opposed to adults who more commonly injure their lower cervical spines (C7 to T1).
- Children are more likely to suffer primarily ligamentous injuries without fractures. This is the result of more horizontally situated facet joints, incomplete spinal ossification, and immature ligamental support structures.
- Pediatric cervical spine injuries are often associated with severe brain injury and respiratory arrest. Unfortunately, many children suffering these injuries die at the scene of the accident.

18. What is SCIWORA?

Spinal cord injury without radiographic abnormality (see Chapter 83)

19. What is pseudosubluxation of the cervical spine, and how common is it?

Pseudosubluxation of the cervical spine is the anatomic normal variant in children in which the C2 vertebral body is slightly anteriorly displaced relative to the C3 body (C2 on C3 pseudosubluxation). This is because of the normal mobility of the vertebral bodies in young children. About 40% of children younger than 7 years and 20% up to the age of 16 years demonstrate pseudosubluxation. Pseudosubluxation can be differentiated from true subluxation by evaluating the line of Swischuk, which is drawn along the anterior edge of the spinous processes of C1 and C3. Injury, and not normal psuedosubluxation, is suspected if the line passes at a distance greater than 1.5 mm from the anterior spinous process of C2.

20. What is the most common upper extremity fracture in children?

Supracondylar fracture is the most common upper extremity fracture, which is a fracture of the distal humerus. This fracture is very common in children younger than 8 years, because the tensile strength of the ligaments and joint capsule is greater than that of the bone itself. Children can sustain supracondylar fractures from falling onto a hyperextended or flexed arm. Providers should suspect this injury in a child with signs of elbow effusion, even if no obvious fracture is seen on plain radiograph. It is important that these children are monitored closely to prevent an ischemic injury know as *Volkmann ischemic contracture.*

21. What are other examples of fractures that are more common in children than adults?

Common pediatric fractures include toddler's fractures, greenstick fractures, torus or buckle fractures, and fractures through the growth plate.

- Toddler's fractures are oblique nondisplaced fractures of the tibia and occur commonly in children younger than 2.5 years. These are relatively low-force injuries. Common mechanisms include running, twisting of the leg, or a fall from an insignificant height. A child may have a limp or refuse to walk.
- Greenstick fractures are incomplete fractures of long bones at the diaphyseal-metaphyseal junction. The bony cortex remains intact on one side.
- Torus (buckle) fractures occur in the metaphyseal region of bone after a compressive load. The bony cortex buckles in a small area.
- The Salter-Harris classification system is used to describe fractures involving the growth plate (see Chapter 92).

22. How common are rib fractures in children?

Rib fractures are not very common. The compliant chest wall allows unimpeded transmission of energy to the underlying thoracic organs, potentially resulting in life-threatening pulmonary 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.

23. How common are mediastinal (great vessel) injuries in children?

Mediastinal injuries are very rare in children. Because of the high compliance of the chest wall and laxity of the ligamentous structures of the mediastinum, aortic injuries as a result of sudden deceleration are rare in children compared with adults.

24. What are predictors of pediatric intraabdominal injuries?

Multiple investigators have attempted to identify predictors of intraabdominal injury in children. High-risk mechanisms for intraabdominal injury should be taken into consideration. These include high-speed motor vehicle collisions, pedestrians struck by automobiles, bicycle accidents (including handlebar injuries), and direct blows to the abdomen. Findings on physical examination that are associated with significant intraabdominal injuries include tenderness on palpation, seat belt bruising, and hemodynamic instability in the prehospital setting. In 2009, Holmes and co-workers published a prospectively validated prediction instrument that identified six criteria, wherein the absence of all of the following factors identified pediatric patients at low risk for intraabdominal injury (95% sensitivity and 37% specificity):

1. Low age-adjusted systolic blood pressure
2. Abdominal tenderness
3. Femur fracture
4. Increased liver enzyme levels (serum aspartate aminotransferase >200 U/L or serum alanine aminotransferase >125 U/L)
5. Microscopic hematuria (>5 red blood cells/high-powered field)
6. Initial hematocrit level less than 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.

25. Compare and contrast the primary diagnostic modalities for evaluating children for abdominal trauma.

CT and ultrasonography (focused assessment with sonography for trauma [FAST] examination) are the primary diagnostic tests. CT is the most sensitive and specific test, identifying solid-organ and (less accurately) hollow-viscus injuries. It also evaluates the retroperitoneum. However, CT imaging is relatively time consuming, requires the patient to be hemodynamically stable for transfer to the radiology suite, and occasionally requires sedation in young children. Therefore it is not appropriate in unstable patients. Ultrasonography (FAST examination) is simple, rapid, repeatable, and noninvasive. It has the advantage of being performed at the bedside. The main objective when performing the FAST examination is to quickly identify free fluid (blood) in the intraabdominal cavity, pelvic cavity, or pericardium. As with adult patients, it may be used to quickly triage unstable pediatric patients to the operating room or to rapidly exclude the abdomen as a source of significant blood loss. Although it is not as sensitive for identifying injury as CT, in the hands of skilled practitioners, FAST is an important and useful adjunct to physical examination and CT imaging. If equivocal, further imaging may be required, depending on the urgency of the situation. Diagnostic peritoneal lavage (DPL), although traditionally used to identify hemoperitoneum or other signs of intraabdominal injuries, is no longer routinely performed.

26. What is a handlebar injury?

The term *handlebar injury* refers to an intraabdominal 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.

27. What is the lap belt syndrome?

Lap belt syndrome, or seat belt syndrome, is the classic triad of abdominal wall ecchymoses or abrasions, flexion-distraction injury of the lumbar spine (Chance fracture), and intestinal or

mesenteric injury. It is usually associated with motor vehicle collisions in which a child is wearing a seat belt improperly (lap belt positioned high over the abdomen rather than low over the pelvis, and/or shoulder belt positioned behind child's back). Properly secured and used car seats, including booster seats, are by the far the most important factor in reducing morbidity and mortality rates among children in motor vehicle collisions.

28. How much of a problem is nonaccidental trauma?

Unfortunately this is an all-too-common condition see in all EDs (see Chapter 66).

29. What are some examples of specific childhood fractures and fracture patterns that are highly suspicious for nonaccidental trauma?

Clinicians should always have a high index of suspicion for abuse whenever a child has multiple fractures or injuries at different stages of healing. Certain fractures should also raise suspicion for nonaccidental trauma. These include "bucket-handle" fractures (metaphyseal corner avulsion fractures), which are avulsed bone fragments typically seen in the proximal tibia, distal femur, or proximal humerus. These fractures are often the result of significant shaking or pulling/twisting of an infant's limb. Seventy percent of femoral fractures in children younger than 1 year (nonambulatory) are associated with abuse. Humerus fractures (with the exception of supracondylar fractures) are strongly suggestive of abuse in children younger than 3 years. Scapular and rib fractures, which require a high amount of force, are also commonly seen with nonaccidental trauma (see Chapter 66).

KEY POINTS: PEDIATRIC TRAUMA

1. Fifty percent of all pediatric ED visits in the United States are related to trauma. Trauma is the leading cause of death among children after the first year of life.
2. Motor vehicle collisions are responsible for the most morbidity and mortality among children.
3. Children are not just little adults. Providers must take into account specific anatomic and physiologic differences when evaluating for pediatric injuries.
4. In the pediatric trauma population, head injury is the leading cause of morbidity and mortality.
5. Hypotension is a late and ominous finding in the pediatric trauma patient. Tachycardia and signs of poor perfusion are earlier and more reliable clues.
6. The most common cause of death in pediatric trauma is respiratory arrest. Therefore special attention should be given to good airway and respiratory support.
7. Because of the laxity of children's ligamentous structures, cervical fractures, rib fractures, and aortic injuries are less common, but cervical ligamentous injuries and pulmonary contusions are more common.
8. Young children are more likely to injury their cervical spines around C1 to C3 compared with adults.

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QUESTIONS

1. When evaluating a child who has been seriously injured, which of the following is the best early indicator of hemorrhagic shock?
   a. Abdominal tenderness
   b. Hypotension
   c. Poor skin perfusion
   d. Crying
   The correct answer is *c.*
2. Which of the following statements is true with regards to pediatric cervical spine injuries?
   a. Children commonly suffer cervical spine vertebral fractures.
   b. Children more commonly suffer spinal cord injuries at the level of C1 to C3 compared with adults.
   c. *Pseudosubluxation* refers to the posterior displacement of the C2 vertebral body compared with the C3 vertebral body.
   d. Plain radiographs and CT imaging will clearly identify all cervical spine injuries.
   The correct answer is *b.*
3. *Lap belt syndrome* refers to a triad of injuries, including all of the following except:
   a. Intestinal injury
   b. Splenic injury
   c. Abdominal wall bruising/contusion
   d. Thoracolumbar vertebral injury
   The correct answer is *b.*

MUSCULOSKELETAL TRAUMA AND HAND INJURIES

CHAPTER 92

Kyros Ipaktchi, MD, FACS, and Philip F. Stahel, MD, FACS

GENERAL PRINCIPLES

1. What are immediate treatment priorities in open fractures?

Open fractures warrant an immediate orthopedic consultation. After ruling out associated vital organ injuries per Advanced Trauma Life Support (ATLS) protocol, open fractures are assessed during the secondary survey. Any skin break near a fracture site should be presumed to communicate with the fracture until proved otherwise. After careful examination, including neurologic and vascular assessment, the wound should be cleaned of gross contamination, and a sterile dressing applied. Wound probing in the ED should be discouraged in general. 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. Wound cultures, extensive irrigation, and multiple examinations of the wound must be avoided because of the increased potential for secondary contamination and soft-tissue damage. Tetanus prophylaxis and intravenous (IV) antibiotics have to be 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 agricultural settings, penicillin is added to cover for the increased risk of anaerobic organisms. Figure 92-1 depicts the institutional multidisciplinary protocol for the initial management of open fractures.

2. What percentage of polytrauma patients have unrecognized fractures at time of admission?

Up to 20% of all multiply injured patients have unrecognized fractures at the time of initial assessment. These occult injuries are located most commonly around the wrist, hand, ankle, and foot. This important fact highlights the need for a tertiary survey of multiply injured patients. The patient's family should be informed from the beginning of the patient's care about the potential that initially unrecognized injuries and fractures may be found in later surveys.

3. What is compartment syndrome?

Acute compartment syndrome (ACS) develops when the pressure within the confined space of the muscle compartment exceeds capillary filling pressures, resulting in muscle ischemia and edema. Muscle ischemia in turn increases intracompartmental pressure and leads to a vicious cycle, resulting in muscle and nerve necrosis. Any progressive mismatch between size of a compartment and its contents can result in ACS.

4. What causes compartment syndrome?

Common causes include fractures, crush injuries, extravasated IV fluids, hemorrhage, postischemic swelling after vascular injury repair, tight-fitting casts or dressings, and circumferential burns. Importantly, ACS can result from minor injury mechanisms as well, and a high level of suspicion should be raised in any patient with pain out of proportion to the clinical findings after a soft-tissue injury, with or without associated underlying fracture or joint dislocation. A high level of suspicion for ACS must also be raised after peripheral ischemic events, such as those secondary to vascular injuries by direct trauma or indirectly by kinking of a critical vascular structure (e.g., popliteal artery kinking after prolonged surgical procedures in lithotomy position, prolonged travels by airplane or motor vehicle with flexed knees, superficial femoral artery [SFA] injuries associated with femur fractures). As a rare variant, "exertional compartment syndrome" is characterized by exercise-induced compartmental pain and swelling that resolves with rest. This is most typically seen in horseback riders, cyclists, and runners. As a general rule of thumb, it is prudent to exclude presence of ACS in any patient with painful extremities of uncertain etiology.

Abstract

This chapter provides a summary of common musculoskeletal injuries and acute traumatic and nontraumatic conditions of the hand to support the ED physician's understanding of these acute entities and to guide appropriate prioritization of initial management in the ED.

Keywords:

musculoskeletal trauma, hand injury, soft-tissue infection, compartment syndrome, pelvic fracture, femur fracture, pediatric fracture

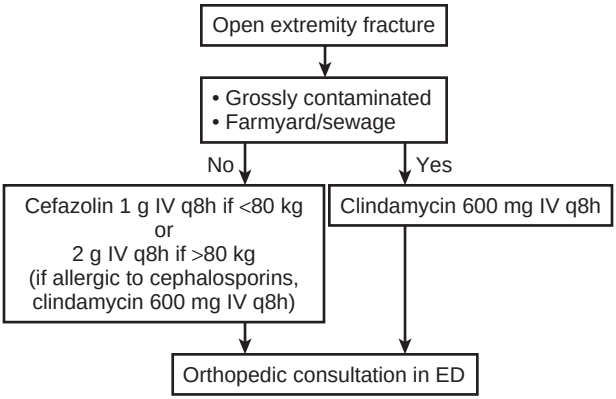

Figure 92-1. ED management of open fractures. *IV,* Intravenous; *q8h,* every 8 hours. *(Modified from Mauffrey C, Bailey JR, Bowles RJ, et al: Acute management of open fractures: proposal of a new multidisciplinary algorithm.* Orthopedics *35:877–881, 2012. Available at Healio.com; accessed 4-10-15.)*

5. What are the clinical signs and symptoms of ACS?

The classic 5 Ps to reflect symptoms of ACS (**p**ain, **p**aresthesia, **p**allor, **p**ulselessness, **p**aralysis) should be revisited to exclusively reflect the main cardinal symptom of ACS: *Pain, pain, pain, pain, pain!* In fact, pain out of proportion to the clinical examination represents the earliest and most common symptom associated with ACS. Pain is typically exacerbated when passively stretching the muscles of the involved compartment (e.g., passive extension of great toe and ankle for assessment of suspected lower limb ACS). Pain is typically ischemic in nature and often not relieved by narcotics. The other 4 Ps represent late symptoms of a "missed" compartment syndrome and typically reflect the extent of an irreversible ischemic injury.

Although there are various methods of measuring intracompartmental pressures, the diagnostic gold standard is represented by a positive clinical examination in conjunction with pain out of proportion and a plausible history (as outlined).

6. What are the most common sites for compartment syndrome?

The leg is the most common anatomic location for ACS, most often associated with proximal tibia fractures and tibia shaft fractures. The anterior compartment of the leg is most commonly affected, and the deep posterior compartment of the leg is the site that is most often missed. Thigh compartment syndrome is less common, and therefore more at risk for being missed. Similarly, upper extremity ACS requires a high level of suspicion, because volar compartment (carpal tunnel syndrome) and forearm compartment syndromes are often missed. These are often associated with both-bone forearm fractures, high-energy distal radius fractures, radiocarpal dislocations, and pediatric supracondylar humerus fractures.

7. How do I treat compartment syndrome?

Detailed documentation and timing of physical findings are of essential importance. The only valid and curative treatment option for ACS is the immediate surgical release of all involved compartments by fasciotomies. Nonoperative measures are justified for monitoring suspected compartment syndrome in equivocal cases. Removal of all circumferential dressings and maintenance of normal limb perfusion by maintaining a normotensive blood pressure are important adjuncts to managing limbs at risk. When in doubt, surgical compartment release by fasciotomies represents the treatment of choice. Rare exceptions to this rule include the presence of crush syndrome and nonviable compartments, and this difficult judgment requires a decision by a qualified surgeon.

8. Describe the joint fluid analysis consistent with septic arthritis.

See Chapter 54.

9. How do I diagnose a traumatic arthrotomy (open joint)?
Probing of a wound in proximity to a joint is inadvisable, because this may increase the risk of a spreading deep infection and septic arthritis. Radiographs of the involved joint may reveal an air arthrogram. A saline challenge will help in diagnosing a traumatic arthrotomy. Larger joints, such as the knee joint, may be injected with up to 150 mL sterile saline. The traumatic wound is inspected for egress of the injected fluid. A positive saline challenge mandates a formal surgical washout of the traumatic open joint, with exploration and closure of the joint capsule.

10. When should I order radiographs, and how many should I order?
Radiographic diagnostics should not delay resuscitation in a multiply injured patient. Whenever existing limb deformity results in vascular compromise or may devitalize overlying skin, radiographs should be delayed, pending emergent realignment and splinting or application of traction to the involved extremity. Radiographs should be ordered based on physical examination findings. Three-way view radiographs should include the joints above and below the perceived area of injury.

KEY POINTS: MUSCULOSKELETAL TRAUMA—GENERAL PRINCIPLES

1. Open fractures require immediate orthopedic consultation and must be recognized and managed urgently.
2. ACS must be identified early and managed by urgent surgical fasciotomies because of the risk of irreversible long-term sequelae with loss of function and potential loss of limb.
3. Suspected septic joints require immediate diagnostic workup and surgical management for positive cases.

HAND AND FOREARM INJURIES

11. What is the incidence of hand injuries seen in EDs?
At least one of every 8 to 10 injury-related ED visits is for a hand or wrist injury. As people reach out to interact with the environment, they use their hands, which makes them prone to injury.

12. List the essential elements of the history in hand injuries.
- Age
- Handedness
- Occupation
- Injury details (how, when, where)
- Tetanus status
- Prior hand injury
- Preexisting disability

13. List the elements of a complete hand examination.
Inspect skin, soft-tissue, and skeletal components, assess neurovascular function, and examine tendon function. Detailed documentation and timing of physical findings are of essential importance.

14. What is the best method to control bleeding in lacerations of hand and forearm?
Apply direct pressure. Tourniquets are rarely necessary. Blood vessels are in close anatomic association with nerves in neurovascular bundles; blindly placing surgical clamps or ligation sutures into the wound is discouraged in general, because this can damage adjacent nerves.

15. What is the normal posture of the hand at rest, and what is the tenodesis test?
When the wrist is held in slight extension, the fingers cascade progressively with a more flexed position from index to small finger. Passive wrist extension will cause tension in the flexor tendons and more cascading; in contrast, wrist flexion will extend the fingers as the flexor tendons relax. Any alteration in this normal posture should raise suspicion for the diagnosis of a tendon injury.

16. Does dorsal hand swelling always signify a dorsal hand injury or infection?
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 has dorsal swelling.

17. What is the Allen test, and how is it performed?

The Allen test verifies patency of the radial and ulnar arteries and is performed as follows:

- Occlude radial and ulnar arteries at the wrist by applying firm finger pressure.
- Have the patient hold the hand in a tight fist for about 5 seconds.
- Ask the patient to open the hand while holding compression. The palmar aspect of the hand will be blanched.
- Release the ulnar artery; this should lead to full digital and thenar reperfusion in 3 to 5 seconds.
- Repeat the test, releasing the radial artery instead of the ulnar artery and look for any delay in reperfusion.

18. How is function of the flexor digitorum superficialis (FDS) tendon tested?

The FDS inserts on the middle phalanx and flexes only the proximal interphalangeal (PIP) joint. The flexor digitorum profundus (FDP) inserts on the distal phalanx; its contraction flexes both the PIP (by contribution only) and the distal interphalangeal (DIP) joints. The FDS muscle-tendon units are independent of one another, whereas the FDP tendons (with the exception of the index finger) arise from a common muscle belly. To test FDS function of a finger, the patient is asked to flex the PIP joint of an isolated finger while blocking the other fingers in extension, thereby disabling the FDP flexion contribution. Because the FDP to the index finger can be independent of the other profundi, the FDS test is unreliable in the index finger.

19. How do I test the extrinsic extensor tendons?

The extrinsic extensors extend the metacarpophalangeal (MCP) joints. They combine with interosseous and lumbrical tendons to form the extensor mechanism for the interphalangeal (IP) joints. To test the extrinsic extensor, ensure that the patient can extend at the MCP joint.

20. Can extensor function to a finger be intact despite complete laceration of the extensor digitorum communis (EDC) to that finger?

Yes, the juncturae intertendineum connect the EDC tendons at the midmetacarpal level. If the EDC to a finger is completely lacerated on the dorsum of the hand, proximal to the juncturae, extension at the MCP joint can still be possible. Careful and complete wound exploration is necessary to identify this injury.

21. How do I test sensory nerve function?

Assess and document, in writing, motor and sensory nerve function before injecting local anesthetic. Test digital nerves by checking two-point discrimination on the volar pad using calipers or self-fabricated opened paper clips. The two points should be less than 5 mm apart.

22. Describe the sensory distributions of the median, ulnar, and radial nerves

See Figure 92-2.

23. How is the motor function for the median, ulnar, and radial nerves tested?

- Median (abductor pollicis brevis [APB]): Abduct the thumb against resistance while palpating the APB muscle belly; touch the tip of the small finger with the thumb ("OK" sign).
- Ulnar (first dorsal interosseous): Abduct the index finger against resistance.
- Radial (no intrinsics; extensor pollicis longus [EPL]): Extend the thumb IP joint against resistance.

24. Name the carpal bones, including the most commonly dislocated carpal bone?

The proximal carpal row contains from radial to ulnar side the following bones:

- Scaphoid
- Lunate
- Triquetrum
- Pisiform

The distal row consists of:

- Trapezium
- Trapezoid
- Capitate
- Hamate

The lunate bone is most commonly dislocated, such as in perilunate injury patterns. See Figures 92-3 and 92-4.

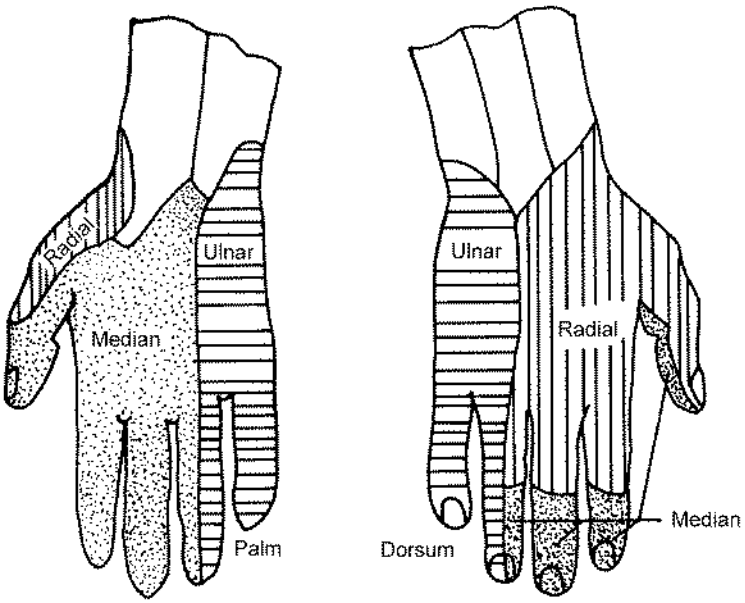

Figure 92-2. Sensory distribution of the median, ulnar, and radial nerves.

25. Which is the most commonly fractured carpal bone?

The scaphoid is the most commonly fractured carpal bone. Its predominant distal blood supply increases the likelihood of avascular necrosis (AVN) in the proximal segment after a proximal pole fracture.

26. How much deformity can be tolerated in metacarpal fractures?

Rotational deformity does not correct itself and is an indication for surgery. Metacarpal neck or shaft rotation may cause finger scissoring when attempting to make a fist. Apex dorsal flexion deformity is common and usually functionally well tolerated. Twenty, 30, 40, and 50 degrees of flexion in the index through small fingers can be accepted without functional deficiency in most patients. A greater degree of deformity is tolerated at the small finger, because of the increased motion at the carpometacarpal (CMC) and MCP joints. The same is true for a thumb metacarpal fracture, in which 40 degrees of angular deformity can be accepted.

27. What are Rolando and Bennett fractures?

These eponyms describe intraarticular fractures of the base of the first metacarpal bone. A Rolando fracture is seen after axial trauma and results in a three-part Y pattern, whereas a Bennett fracture is seen in eccentric shear injuries, where the volar beak of the metacarpal tears off as an avulsion piece held by the volar oblique ligament. Both fractures are surgical indications. Prognosis of Rolando fractures is worse than for Bennett fractures.

28. 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 lies at the floor of the anatomic snuffbox of the wrist, which is bordered by the EPL and the extensor pollicis brevis tendons. Tenderness in this area is suggestive of a scaphoid fracture, which can be undetectable on initial radiographs. Trabecular resorption at the fracture site aids secondary radiographic detection approximately 10 to 14 days after injury. Patients with a concern for a possible occult scaphoid fracture 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 setting.

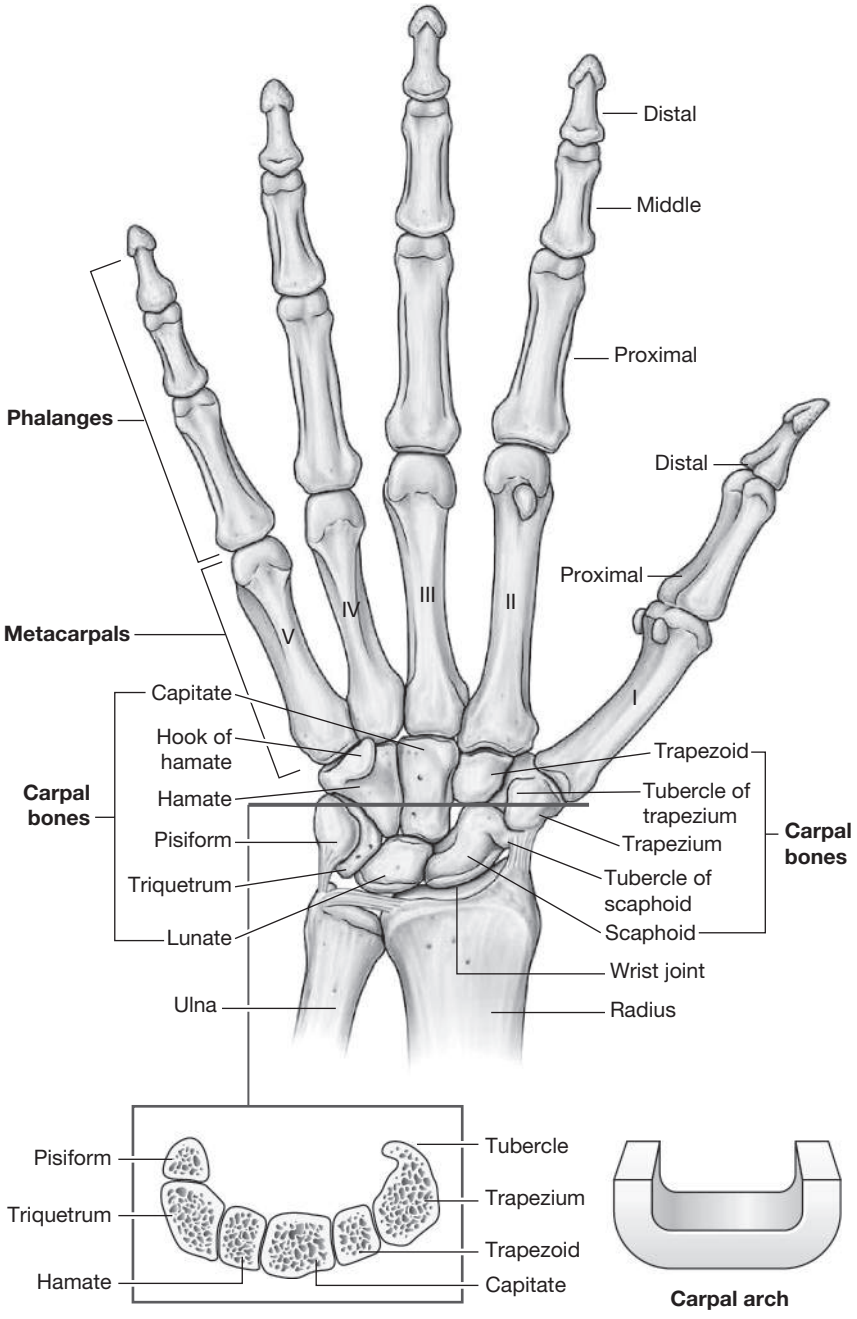

Figure 92-3. Carpal bones of the wrist. *(From Drake R, Vogl AW, Mitchell A:* Gray's Anatomy for Students, *Philadelphia, 2005, Saunders, Fig. 7-91.)*

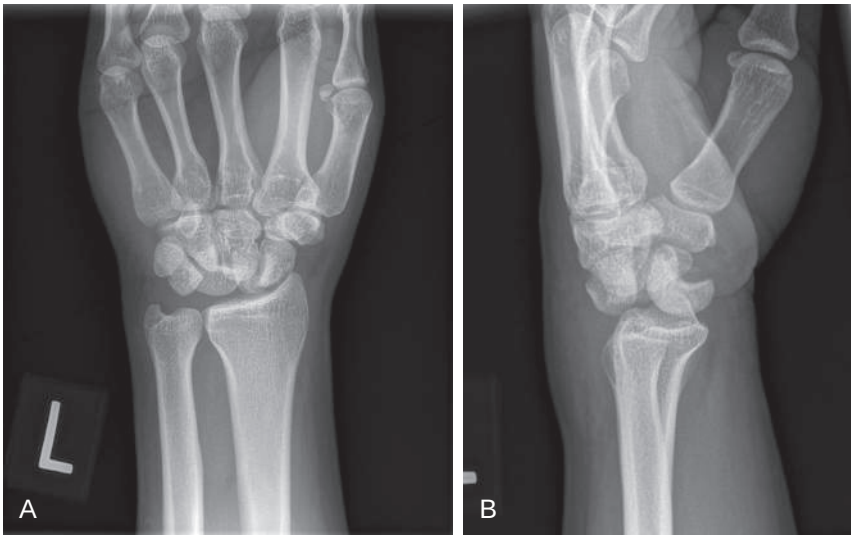

Figure 92-4. Perilunate transscaphoid fracture dislocation with associated lunate fracture. *(From Marx J, Hockberger R, Wall R:* Rosen's Emergency Medicine—Concepts and Clinical Practice, *ed 8, Philadelphia, 2013, Saunders, Fig. 51-22B.)*

29. What is the difference between a "nightstick" fracture and a Monteggia fracture?

Proximal-third ulna fractures with associated radial head dislocation are usual presentations of a Monteggia fracture. This fracture can occur from a fall on an outstretched hand with associated valgus force on the extremity. The treatment requires internal fixation of the ulnar fracture. Nightstick fractures of the ulna result from a direct blow to the ulnar shaft. There is no associated injury to the proximal radioulnar 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.

30. What nerve can be injured in a Monteggia fracture?

The posterior interosseous nerve (PIN) lies in close proximity to the neck of the radius. The PIN can be acutely stretched during dislocation or sustain a chronic traction neuroma in persisting dislocations. The patient usually has an inability to extend the thumb or wrist.

31. Why are high-pressure injections serious hand injuries?

High-pressure injection injuries (from paint or grease guns) initially often may seem innocuous. Whereas there is usually only a small entry point, the underlying tissue destruction can be extensive and disastrous. Emergent surgical debridement and release is indicated. Loss of function, even loss of the finger, is well described in literature.

32. List Kanavel's four cardinal signs of flexor tenosynovitis.

1. Flexed finger posture
2. Fusiform finger swelling
3. Pain on passive IP joint extension
4. Percussive tenderness along the flexor tendon sheath

Flexor tenosynovitis requires urgent surgical management.

33. What is a paronychia, and how is it treated?

Paronychia is a common infection involving the nail fold. Although *Staphylococcus aureus* is the usual pathogen in acute presentations, fungal infections are common pathogens in chronic infections. In the absence of visible pus or fluctuance, treatment consists of warm moist compresses, elevation, and antistaphylococcal antibiotics. If there is fluctuance or pus present, irrigation and debridement is indicated. This may consists of simply elevating the eponychial fold or

making a small longitudinal incision over the area of fluctuance. Removal of a longitudinal section of the nail plate may be necessary.

34. How is whitlow different from a paronychia?

Whitlow is a herpes simplex virus infection with burning pain and erythema, followed by a vesicular rash with serous drainage on the distal phalanx. Whitlow can be seen more commonly in health care workers and in preschoolers. A Tzanck smear can be diagnostic. The patient also may have perioral cold sores. The treatment consists of observation and acyclovir administration. Surgery is not necessary or indicated.

35. What is a felon, and how is it treated?

A felon is a painful and potentially disabling infection of the fingertip pulp.

The finger pulp anatomy features fibrous septae. These structures complicate treatment of the infection and preclude full drainage unless properly debrided. Midlateral incisions, including J-shaped incisions are favored. Older fish-mouth or double-longitudinal incisions have fallen out of favor because of excessive scarring and tissue necrosis risk. Care must be taken not to convert a felon into a flexor tendon sheath infection by opening up the flexor sheath. Cultures should be obtained, and wounds kept open by a wick to facilitate drainage, followed by daily soaks.

36. What is a jersey finger, and how is it treated?

Jersey finger describes a rupture of the FDP in sudden forceful flexion against resistance, such as occurs when a football player snags a finger on an opponent's jersey. The tendon is avulsed from its insertion at the palmar base of the distal phalanx and can take a bone fragment along. Surgical repair within the next several days is indicated.

37. What is a mallet finger, and how is it treated?

A mallet finger may be seen as an opposite of the jersey finger. In this case the insertion of the extensor 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 neutral position for 8 to 10 weeks in a DIP blocking (Stack) splint while allowing PIP joint motion to prevent a swan neck deformity. A hand specialist should do the follow-up consultation and aftercare of this injury.

38. Describe a subungual hematoma. How is it treated?

Subungual hematomas are painful collections of blood under the nail plate. Relieving the pressure by nail trephination will result in immediate pain relief. Use a red-hot paperclip to burn a hole in the nail or an 18-gauge needle to gently drill a hole through the nail to allow drainage of the accumulated blood. Removal of an intact nail plate is almost never indicated.

39. What is a gamekeeper's thumb, and how is it diagnosed?

Gamekeepers thumb describes a torn ulnar collateral ligament (UCL) of the thumb's MCP joint, resulting from forceful abduction of the thumb. The name arose from an initial description in 24 Scottish game wardens who sustained UCL injuries while breaking the necks of wounded rabbits. The injury, also called *skier's thumb,* can be disabling because of chronic instability of the thumb at the MCP joint and ensuing arthritis. One way to test for injury to the UCL of the thumb MCP joint is to hand the patient a heavy can or bottle. If the MCP joint is unstable, the patient will be unable to hold the object without supinating to balance the object in the palm or dropping it. Complete rupture of the ligament usually requires surgical repair. ED treatment consists of application of a thumb spica splint and referral.

40. What is a boxer's fracture?

Boxer's fractures of the small or ring finger metacarpal neck typically occur as a result of a fist fight. Because of increased small- and ring-finger metacarpal mobility in the CMC joint and greater MCP joint motion, fracture deformities up to 60 degrees flexion can be well tolerated, especially in the small finger. Nevertheless, attempts to correct significant angulation of an acute boxer's fracture are indicated. Of particular importance is that any rotational deformity must be corrected. A laceration accompanying a boxer's fracture is assumed to be a fight bite.

41. What is a fight bite?

As the name implies, the injury occurs when a punch is thrown at an opponent's mouth, resulting in the hand striking the teeth. Because of the close proximity of the underlying MCP joint, any resulting skin laceration must be assumed to be a traumatic arthrotomy. All suspected fight bites

require formal irrigation and debridement in the operating room to prevent septic arthritis. This includes extending the skin laceration to visualize tendon and joint injuries. Underlying cartilaginous injuries can be present and escape radiographic detection. Associated tendon injuries and metacarpal fractures must be ruled out. Incisions are loosely approximated or left open to allow drainage. Patients should be admitted for overnight hospitalization for IV antibiotics and wound care.

42. Name six hand emergencies.

1. Partial or complete amputations with acute vascular compromise
2. ACS
3. Third-degree and circumferential burns
4. High-pressure injection injury
5. Flexor tenosynovitis
6. Septic joint

43. Name indications and contraindications for a microvascular replantation

Indications:

- All pediatric amputations
- Multiple finger amputations
- Thumb amputation proximal to the nail fold
- Whole-hand or midhand amputation
- Any major upper extremity limb amputation

Contraindications:

- Patients who have experienced multiple trauma
- Severe crush or avulsion injuries
- Multilevel injuries
- Heavy contamination
- Single-finger amputations in adults
- Severe associated medical problems

The ultimate decision should be deferred to a hand or microvascular surgeon, including discussion of options and outcomes with patient and family.

44. How should an amputated part be handled and stored for transport?

- Remove gross contamination with saline irrigation.
- Wrap the part in a saline-moistened (not soaked) sterile gauze.
- Place the wrapped part into a sealed plastic bag or container.
- Place the bag or container into an ice water bath.

Never put the amputated part directly onto ice or immerse it in disinfection solution.

45. What should be done with a devascularized but still partially attached digit?

Leave the part attached (preserves veins for replantation), gently wrap it in moist gauze, and apply an immobilizing bulky dressing.

KEY POINTS: HAND AND FOREARM INJURIES

1. Scaphoid fractures can escape radiographic detection in the acute setting. Patients with tenderness to palpation of the anatomic snuffbox should be treated with a thumb spica splint and undergo repeat evaluation in 1 to 2 weeks under the assumption they may have a fracture.
2. Extensive tissue destruction as seen in high-pressure injections can be missed because of an innocuous entry wound. These injuries require urgent surgery.
3. Any laceration over the dorsal MCP joint is suspicious for a fight bite. Fight bites require meticulous exploration and wound care in the operating room. If the wound penetrates the extensor hood, thorough joint washout and IV antibiotics are required.
4. Bleeding from a wound is best controlled with direct pressure and not clamps or ligature stitch.
5. Never place an amputated part directly on ice or immerse in water; always double bag amputated parts.

SHOULDER AND UPPER ARM INJURIES

46. How can I detect anterior and posterior shoulder dislocations on radiographic film?

Anteroposterior (AP) radiographs may show an inferomedial bony overlap in anterior dislocations. In AP radiographs, a posterior shoulder dislocation can be diagnosed by a vacant glenoid sign, because the humeral head fails to fill most of the glenoid. There is also a positive rim sign, with space between the anterior rim of the glenoid and the humeral head exceeding 6 mm.

47. What is the incidence and what are common causes of posterior shoulder dislocations?

Posterior dislocations account for 5% of shoulder dislocations. The usual mechanism is a fall onto the outstretched hand. Other causes 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, followed by external rotation of the arm until the humeral head has cleared the glenoid rim. A sling should be applied in neutral position to 5 to 10 degrees of external rotation and slight abduction.

48. What percentage of patients with anterior shoulder dislocations experience recurrent dislocations?

In patients aged 30 years or younger, 90% experience a recurrent dislocation; in older patients the percentage is lower and more variable, depending on the injury mechanism.

49. What are potential complications of anterior shoulder dislocations?

The axillary nerve can be injured at the time of dislocation. Examination of the deltoid muscle motor function and sensation to the lateral aspect of the shoulder are mandatory. Additionally, rotator cuff tears can occur, especially in first-time dislocations in patients older than 40 years.

50. How is a rotator cuff tear diagnosed?

Pain with overhead activity, night pain, and pain with abduction of the arm are typical symptoms of a rotator cuff injury. Patients have difficulty abducting the arm and are often 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 (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. If injecting 10 mL 1% lidocaine into the subacromial space relieves these findings, then subacromial impingement rather than a pure rotator cuff tear is more likely.

51. What is the most common neurologic deficit seen with humeral shaft fractures?

The radial nerve may be contused, stretched, or, rarely, lacerated. This condition typically occurs with fractures involving the distal third of the humerus, where the radial nerve passes through the intermuscular septum (Holstein-Lewis fracture) and is prone to injury. Disability includes inability to extend the wrist and fingers at the MCP joints and numbness on the dorsum of the radial side of the hand. IP extension, through ulnar and median nerve function, may be preserved. Triceps function is usually intact because radial nerve branches exiting proximal to the radial groove power it.

52. What about clavicle fractures?

Clavicle fractures result mainly from indirect trauma forces as the most common etiology, typically from a fall onto the shoulder or on the outstretched hand (e.g., bicycle accident). Direct trauma, such as a blow to the clavicle during contact sports, represents a less common cause of clavicle fractures. Clavicle fracture is the most common fracture in pediatric patients. The most common site is the middle third of the clavicle, in approximately 75% of all cases.

53. How are clavicle fractures treated?

Most clavicle fractures are treated in the ED by placing the shoulder in a sling for comfort and pain control. The historic “figure-of-8” bandage is outdated and considered obsolete. Orthopedic consultation is appropriate for fractures that may require operative repair. These include completely displaced fracture ends, fractures with greater than 2 cm of shortening, displaced fractures causing skin tenting, open fractures, and fractures associated with neurovascular injuries.

54. What is a shoulder separation, and how does it occur?

The term *shoulder separation* more properly refers to a separation at the acromioclavicular (AC) joint. AC separations typically result from a direct blow to the point of the shoulder, such as occurs in high-energy contact sports (football). AC separations are divided into different types, depending on the degree of damage to the AC ligament and amount of displacement of the clavicular end.

55. How is an AC separation treated?

The large majority of AC separations are managed conservatively with a sling, ice packs, and analgesics. Surgery is usually reserved for significant displacement in young patients with high demands on their shoulders.

KEY POINTS: SHOULDER AND UPPER ARM

1. Shoulder dislocations can be associated with rotator cuff injuries in first-time dislocations in patients older than 40 years.
2. Document motor and sensory function of the axillary nerve in shoulder dislocations.

LOWER EXTREMITY AND PELVIC FRACTURES

56. Name major complications seen in pelvic fractures.

- Hemorrhagic shock
- Death from exsanguinating hemorrhage
- Urogenital and rectal injuries (see Chapter 89)

57. What is the mortality rate in patients with open pelvic fracture?

Mortality has decreased from 50% to 60% in the 1990s to around 10% to 25%, because of multidisciplinary approaches, proactive concepts of early hemorrhage control (including retroperitoneal pelvic packing), and advances in critical care.

58. What is the incidence and injury mechanism in posterior hip dislocation?

Around 80% of all hip dislocations are posterior dislocations; the mechanism is usually a posterior directed force applied to a flexed knee, as occurs when the knee strikes the dashboard in a head-on motor vehicle crash.

59. What complications can be seen in posterior hip dislocations?

Sciatic nerve injuries are found in 10% of patients, resulting in weakness or loss of hamstring function in the thigh and leg.

AVN of the femoral head is seen in 10% to 15% of patients. The risk of AVN increases 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.

60. How are posterior hip dislocations clinically differentiated from femoral neck fractures?

Both result in lower extremity shortening. In posterior hip dislocation, the hip is flexed, adducted, and internally rotated. With a femoral neck or intertrochanteric fracture, the lower extremity is not flexed but shortened, abducted, and externally rotated.

61. How much blood loss can be expected from a femoral shaft fracture?

Patients typically lose 1500 to 2000 mL of blood.

62. How are femoral shaft fractures best stabilized in the ED?

Stabilization is best achieved with immediate application of longitudinal traction, using a self-contained traction unit. Most emergency medical service providers carry these and can apply them in the field or ambulance. Another option in the hospital is placement of a distal femoral traction pin and in-line traction connected to the bed or gurney. Conventional splinting is ineffective and contraindicated. Femoral traction dramatically reduces the mortality and risk of remote organ injury (brain, lungs) from femoral shaft fractures.

63. Why can patients with pathologic conditions of the hip experience 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 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.

64. Name the most common injury associated with traumatic hemarthrosis of the knee joint.

Anterior cruciate ligament (ACL) ruptures are most commonly causes for knee hemarthrosis. If fat globules are noted in the joint aspirate, the possibility of an associated intraarticular fracture should be pursued.

65. Name the most commonly injured ligament seen in an inversion-type ankle sprain.

The most commonly injured ligament is the anterior talofibular ligament (ATFL). The calcaneofibular ligament can also be injured in more severe sprains.

66. 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.

67. What is a locked knee, and 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 medial meniscus tears, a loose body such as an osteochondral fragment in the knee joint, or a dislocated patella.

68. What is the most common direction of a knee dislocation?

The direction is determined by the position of the tibia relative to the femur. Anterior dislocations are seen in around 40% of cases, followed by posterior dislocations in 25%. Hyperextension trauma causes anterior dislocations, and dashboard mechanisms often result in posterior dislocations. Forty percent to 50% of all anterior/posterior dislocations are associated with a popliteal artery injury. Postreduction angiography should be considered for all patients with abnormal distal pulses or ankle-brachial index (ABI).

69. What direction is associated with irreducible knee dislocations?

Posterolateral dislocations are irreducible. Here the medial femoral condyle produces a dimple sign as it buttonholes through the anteromedial joint capsule, thus becoming entrapped. An open reduction in the operating room is required.

70. How is the ABI calculated?

The ABI is calculated by dividing the Doppler systolic arterial pressure measured in the injured leg by the pressure measured in an uninjured arm. An ABI value of greater than 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. An ABI less than 0.9 must trigger immediate further diagnostic workup and/or surgical exploration in the operating room.

71. What injuries are often associated with calcaneal fractures?

Depending on injury mechanism and type of calcaneal fracture, up to 50% of patients may have an associated compression fracture of the lumbar or lower thoracic spine. Ten percent of all calcaneal injuries are bilateral, and about 25% are associated with other lower extremity injuries.

KEY POINTS: LOWER EXTREMITY AND PELVIS

1. A multidisciplinary approach is required for the successful management of acute traumatic pelvic hemorrhage.
2. ACL injuries are common causes for a hemarthrosis of the knee.
3. All knee dislocations require a thorough vascular examination, including ABI measurement, to rule out an associated vascular injury.

PEDIATRIC ORTHOPEDICS

72. What is a torus or buckle fracture?

This fracture is typically seen in the radial metaphysis. Torus, or buckling, describes the pediatric plastic bone deformity, resulting in a round swelling or protuberance, which happens when bone fails in compression, while the opposite cortex remains intact. Because the opposite cortex remains intact, these fractures are stable and require cast immobilization for 4 weeks.

73. 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 forearm and may require reduction, in which case the fracture should be completed to achieve an adequate reduction. Immobilization in a cast is required for 6 weeks.

74. What is the Salter-Harris classification?

Fractures involving the physeal zone may result in growth disturbance, and parents must be informed accordingly. 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 worse prognosis. Displaced Salter-Harris types III and IV fractures may require open reduction to restore the normal anatomic physeal relationship.

The five types of fracture according to the Salter-Harris classification are (Fig. 92-5):

- Type I: Physeal separation; this may appear as a widening of the radiolucent area representing the growth plate.
- Type II: Fracture traverses the physis and exits on the metaphyseal side.
- Type III: Fracture traverses the physis and exits on the epiphyseal side.
- Type IV: Fracture traverses through epiphysis, physis, and metaphysis.
- Type V: Physeal crush injury; this may be difficult to determine on plain radiographs.

75. Which vascular complication is associated with pediatric supracondylar humerus fractures?

Displaced pediatric supracondylar humerus fractures have a 5% incidence of vascular injuries. The brachial artery can be compressed or lacerated by the anteriorly displaced humeral shaft. Posterolateral displacement of the supracondylar fracture is the pattern most likely to result in vascular injury. Children with pink, pulseless hands should undergo prompt reduction and fracture fixation in the operating room, with subsequent reexamination and vascular surgery consultation.

76. Describe the neurologic complications associated with pediatric supracondylar humerus fractures.

The anterior interosseous nerve (AIN; branch of the median nerve) is the most commonly injured nerve. It innervates muscles in the deep flexor compartment of the forearm: radial part of the FDP, the pronator quadratus, and flexor pollicis longus (FPL). AIN function is tested, evaluating FPL and FDP function at the thumb and index finger IP joint (OK sign). 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.

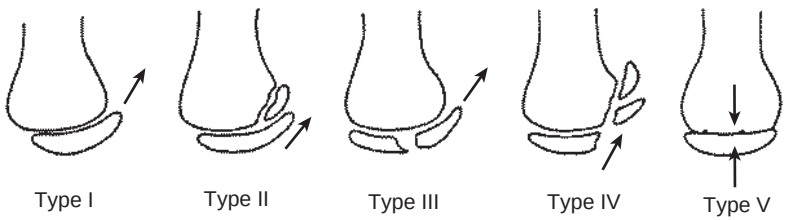

Figure 92-5. Salter-Harris classification of physeal injuries.

77. What is a nursemaid's or pulled elbow, and what is its management?

A longitudinal pull on the outstretched arm of a small child may subluxate the cartilaginous radial head out of the annular ligament. The child typically experiences 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 click over the radial head signifies reduction. The child often begins using the extremity within minutes of reduction. The parent or caregiver should be educated to avoid longitudinal traction on the arm to prevent this from reoccurring.

78. Describe the potential implications of long bone fractures in a small child.

In nonambulating children with long bone fractures, nonaccidental trauma (child abuse) must be considered (see Chapter 66).

79. What is the Waddell triad?

The Waddell triad describes the injury constellation of a child struck by a car sustaining a femur fracture, intrathoracic or intraabdominal injury, and a head injury.

80. Which nontraumatic hip disorders cause a limp in a child?

- Septic arthritis
- Transient synovitis
- Slipped capital femoral epiphysis (SCFE)
- Idiopathic AVN
- Perthes disease
- Juvenile rheumatoid arthritis

Among these uncommon diagnoses, transient synovitis is probably the most prevalent cause of a nontraumatic limp in a child and a diagnosis of exclusion. Symptomatic treatment is prescribed for transient synovitis, including nonsteroidal antiinflammatory drugs and non–weight bearing or bed rest. Untreated or delayed treatment of septic arthritis can irreversibly damage articular cartilage. Infection in a child with atraumatic hip pain must be ruled out. The white blood cell count, erythrocyte sedimentation rate, and body temperature commonly are elevated in cases of infection. If doubt persists, a hip aspiration is the gold standard and is usually performed in the operating room. Standard AP and lateral radiographs of the hip help differentiate between Perthes disease and an SCFE.

81. 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 radiographic view. If AP 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. Further diagnostics, such as MRI, can diagnose preslip conditions if radiographs are negative. MRI examinations are usually ordered through a pediatric orthopedic consultation.

82. What is the ED management of a child with injury and tenderness over an open epiphysis but a normal radiograph?

In this circumstance, assuming there is an occult physeal injury is prudent. Splint the extremity 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 consultation with a specialist 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.

KEY POINTS: PEDIATRIC ORTHOPEDICS

1. Rule out septic arthritis in a child with hip pain independent of a history of trauma, because septic seeding of a joint can be facilitated by a preceding trauma.
2. Consider nonaccidental trauma (child abuse) in nonambulating children with long bone fractures.

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QUESTIONS

1. All of the following represent a condition requiring urgent orthopedic consultation except:
   a. Suspected septic arthritis
   b. Scaphoid fracture
   c. Suspected compartment syndrome
   d. High-energy pelvic fracture
   The correct answer is *b*.
2. How are femoral shaft fractures best managed initially in the ED?
   a. In-situ splinting in a posterior splint, as long as distal pulses are normal
   b. Immediate application of axial traction
   c. Bedside application of an external fixator
   d. Radiographic documentation in three views before initiating treatment
   The correct answer is *b*.
3. Which of the following injuries allow definitive management in the ED?
   a. Flexor tendon injury of the hand
   b. Tenosynovitis of the hand
   c. Nondisplaced ulnar nightstick fracture
   d. Displaced pediatric supracondylar humerus fracture
   The correct answer is *c*.
4. Which statement about clavicle fractures is correct?
   a. Clavicle fractures typically occur from a direct trauma mechanism or penetrating injuries.
   b. Most clavicle fractures require surgical management.
   c. Clavicle fractures are most common in the middle third (midshaft).
   d. Clavicle fractures are most common in the lateral third.
   The correct answer is *c*.
5. Which statement about traumatic AC joint separations is correct?
   a. AC joint separations are rare injuries that are mainly seen in the elderly population after minor falls.
   b. Most AC joint separations are managed operatively.
   c. AC joint separations are commonly caused by a direct trauma mechanism, such as a direct hit or blow to the shoulder in contact sports.
   d. AC joint separations are treated with a figure-8 bandage
   The correct answer is *c*.

CHAPTER 93

BURNS

Michael C. Overbeck, MD

KEY POINTS

1. Physicians should be facile in estimating burn depth and the percentage of total body surface area (%TBSA) involved, because these drive early resuscitation, management, and referral decisions.
2. Airway compromise can evolve quickly in the burned patient. Careful attention to historical features of the injury, subjective symptoms, and objective signs suggesting significant airway involvement can inform the decision to intubate the patient's airway.
3. Consider consultation and transfer to a burn center in patients with larger, deeper burns, extremes of age, or burns complicated by significant trauma.
4. A majority of burns seen in the ED can be managed in the outpatient setting. Elements of an outpatient management plan include pain control, wound care, close initial follow-up care, return criteria, and in pediatric patients, a reliable and safe home environment.

1. Immediately after a thermal injury, what first aid should be offered?

Primarily, the burn process should be stopped (i.e., either removal from heat source, extinguishing the flame, or diluting any chemical). Next, thermal burns should be cooled for a minimum of 20 minutes with cool tap water. This may have important and positive effects when applied up to 3 hours after the burn. Ice may provide analgesia but can extend tissue damage through tissue necrosis and is not recommended.

2. How is the burn patient evaluated on arrival?

The physician should undertake a trauma evaluation, including primary and secondary survey, with careful attention to airway adequacy, ventilation, and oxygenation. In patients with limited and uncomplicated partial-thickness burns to an extremity, this process is often straightforward. Evaluate the burn depth and distribution of involved areas. Early intubation should be considered in cases of respiratory insufficiency or inhalation injury.

3. What should I do if there is both trauma and burns?

The combination of trauma and burns in a patient increases the chances of morbidity and mortality. This population often requires concomitant early fluid resuscitation while trauma evaluation and stabilization are ongoing. Because of the associated higher morbidity, coordination of care is important, and transfer to a burn center should be strongly considered after initial trauma stabilization.

4. What factors are important in assessment of the burn patient's airway?

It is difficult to predict which patients will go on to develop laryngeal edema and airway obstruction, and the evaluating physician must be alert to the rapidly evolving conditions that threaten upper airway patency. Increasing risk is noted in patients with larger %TBSA burns. Soot around the mouth or nares, hoarseness or stridor, facial burns, or carbonaceous sputum are suggestive of significant airway injury. Decreased oxygen saturation occurs late, if at all, in patients with significant airway involvement.

5. List the criteria for transfer to a burn center.

- Partial-thickness burns greater than 10% TBSA
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Full-thickness burns (of any size, as these will not heal on their own)
- Electrical burns, including lightning injury
- Chemical burns
- Inhalation injury

Abstract

The early care of patients with burns is in the ED. The initial approach and treatment of the burned patient, including wound care and fluid resuscitation is discussed in detail.

Keywords:

burn, airway, fluid management, trauma, child abuse, geriatrics, Parkland formula

- 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
- Burn injury in children at 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

6. What informs my decision to intubate the airway of a burn patient?

The likelihood of a patient's injury progressing to airway compromise is difficult to reliably estimate; therefore a low threshold for intubation must be maintained. Historical features of injury (confined space exposure, loss of consciousness, or advanced age) or physical examination findings (early oropharyngeal edema, facial or circumferential neck involvement, or stridor) can be important clues to impending airway obstruction. Increased cyanide or carbon monoxide (CO) levels should raise the suspicion for significant inhalation injury. Nasopharyngoscopy may yield supporting evidence of upper airway injury that can threaten airway patency in minutes or hours. Even in the absence of signs of inhalation injury, providers must maintain a high level of suspicion for evolving airway obstruction that, if occurring during transport, can have catastrophic consequences.

7. What dangers might be encountered with airway intubation of the burn patient?

Postburn facial oropharyngeal edema portends a difficult airway. The most skilled operator should perform the intubation, with a surgical airway considered the immediate alternative should oxygenation or ventilation suffer during initial intubation attempts. As with any anticipated difficult airway, cautious consideration of whether or not to administer paralytic agents should be given, to avoid the paralyzed, apneic patient that is subsequently unable to be ventilated. Additional attention should be given to the real possibility that a patient with significant burns can be hypovolemic and may become profoundly hypotensive after administration of sedative or paralytic medications employed in rapid sequence intubation.

8. Can succinylcholine be used safely in intubation of the burn patient?

Succinylcholine is commonly reported as contraindicated in the burn patient because of 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. Succinylcholine is considered to be safe to use up to 48 hours after the burn injury.

9. How is the burn depth categorized?

Classification of burns as "first-," "second-," or "third-degree" has transitioned to a more functional descriptive terminology. Physician awareness of this terminology is important in assessing, managing, and referring burn patients. The currently accepted terminology is *superficial* or *epidermal burns, superficial partial-thickness burns, deep partial-thickness burns,* and *full-thickness burns*.

10. What do superficial burns look like?

Superficial or epidermal burns, previously identified as *first-degree burns,* are characterized by red coloration, dry surface, and absence of blisters. The dermal-epidermal interface is the downward limit of damage, though irritation of dermal vessels and nerve endings projecting upward result in erythema and pain. Edema may result, particularly in sensitive areas such as the surrounding soft tissues of the eyes. This is the typical burn suffered after sun exposure, developing over hours and resolving in 3 to 5 days. This type of burn should not be included in the %TBSA calculation.

11. Describe superficial partial-thickness burns.

Superficial partial-thickness burns, previously identified as *second-degree burns,* are characterized by erythema, pain, and blisters. As blisters arise and rupture, exposed dermis with associated nerve endings for pain, temperature, and light touch make these injuries particularly sensitive. The loss of the covering of dermal papillae results in a glistening wound surface. Edema is typically present. The underlying capillary network remains intact, resulting in hyperemia and brisk capillary refill.

12. What are deep partial-thickness burns?

Deep partial-thickness burns, previously identified as more severe *second-degree burns*, extend down to the deep layer of the dermis. Capillary refill is slow or absent, and areas may be

erythematous or pale white. Sensation is altered/decreased, and the exposed surface is typically wet with prominent edema.

13. Describe full-thickness burns.

Full-thickness burns, previously described as *third-degree burns,* are burns that penetrate the full thickness of the epidermis and dermis down into the subcutaneous tissue. Capillary networks are cauterized and sensory nerve endings are obliterated, making these burns insensate, nonblanching, and initially dry. Subdermal burns, previously characterized as *fourth-degree burns*, involve subcutaneous structures such as muscle, bone, and the interstitium.

14. Why are circumferential full-thickness burns important to recognize?

Capillary leak and evolving edema of an extremity underneath overlying circumferential eschar can threaten limb perfusion and viability of distal tissues. Therefore in extremities with circumferential burns, adequacy of tissue perfusion should be closely evaluated, including temperature change, capillary refill, evolving paresthesias, and sensory examination. In affected extremities, hourly evaluation of pulses should be undertaken with Doppler ultrasound to ensure adequate perfusion. Hand or finger escharotomies are rarely necessary, and should only be undertaken in consultation with a burn center physician.

15. Why, when, and where are thoracic escharotomies done?

Circumferential partial- or full-thickness burns of the thorax can threaten mechanics of ventilation. Often the physician is alerted to the early evolution of this complication by worsening lung compliance parameters on the mechanical ventilator. After other causes of inability to ventilate the burn patient are evaluated (e.g., mucus plugging or kinked or disconnected tubing), thoracic escharotomy is curative and can be performed at the bedside to avoid unnecessary delays and complications associated with a formal trip to the operating room.

16. How does %TBSA impact patient care? How is it calculated?

Coupled with the depth of the burn, %TBSA drives management decisions, disposition, and impacts survival. Although limitations in physicians' ability to reliably estimate the %TBSA have been demonstrated, it remains an important parameter in patient care. In adults, Wallace's Rule of Nines (Fig. 93-1) can assist the provider in calculating the TBSA involved in partial- and full-thickness burns. Similarly, in children, Lund and Browder developed a standardized aid (Fig. 93-2) to incorporate the unique proportions of children when calculating TBSA involved in burns. By convention, superficial or first-degree burns are not included in the total.

17. How is %TBSA useful in planning fluid resuscitation in burn patients?

Immediately after a burn, an inflammatory response causes extravascular fluid shifts in the body, resulting in relative intravascular depletion. The Parkland formula is perhaps the best known way to calculate the fluid needs of the burned patient proportional to the %TBSA. The total volume required by the patient over the first 24 hours is calculated as:

$$\text{Total fluid in 24 hours} = 4\ \text{mL fluid} \times \text{kg body weight} \times \%\text{TBSA (partial- and full-thickness burns)}$$

Half of this volume is administered over the first 8 hours after the burn; the remainder is administered over the subsequent 16 hours.

18. Calculate a fluid regiment using the Parkland formula in a 70-kg adult suffering 20% TBSA burns.

The 24-hour fluid total is 4 mL/kg/%TBSA burned, or:

$$4\ \text{mL/kg/\%} \times 70\ \text{kg} \times 20\%, \text{ or } 4\ \text{mL/kg/\%} \times 70\ \text{kg} \times 20\%, \text{ or } 5600\ \text{mL given over 24 hours.}$$

Half of this volume is given over the first 8 hours, or $5600 \div 2 = 2800$ mL over 8 hours, or:

$$2800 \div 8 = 350\ \text{mL/h rate}$$

The second half is administered over the next 16 hours, or:

$$2800 \div 16 = 175\ \text{mL/h rate}$$

19. Are there any pitfalls with using the Parkland formula?

Evidence is beginning to surface suggesting that the Parkland formula underestimates volume requirements of adults with isolated severe burns. However, the Parkland formula should be

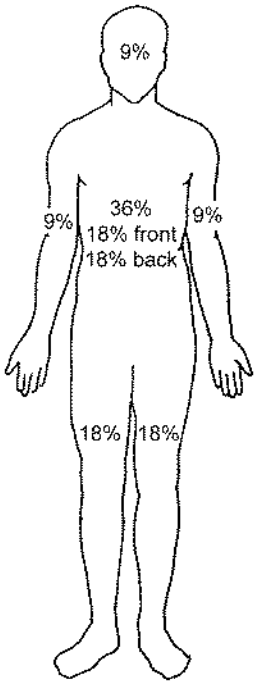

Figure 93-1. Percentages used in determining extent of burn by Rule of Nines. *(From Miller RH:* Textbook of basic emergency medicine*, ed 2, St. Louis, 1980, Mosby.)*

considered a starting point, with adjustments in fluid administration to maintain urine output in adults at a rate of 0.5 mL/kg/h or 1 to 2 mL/kg/h in children as monitored with an indwelling Foley catheter. It is additionally helpful in standardizing care when burn patients are initially evaluated and subsequently transferred to tertiary burn centers.

20. What is burn shock?

When a patient is given inadequate fluid resuscitation immediately after a burn, increased capillary permeability and insensitive losses combine to rapidly decrease circulating blood volume, threatening tissue perfusion and end-organ function, resulting in shock. Prompt intravenous (IV) volume resuscitation is essential to prevent burn shock. As renal failure with oliguria is a common and recognized consequence of underresuscitation, many protocols focus on maintenance of urine output to gauge adequacy of fluid management (*Advanced burn life support provider manual,* p 35)

21. What are three mechanisms by which smoke inhalation can cause injury?

1. Thermal injury to the mouth, tongue, oropharynx, and larynx (i.e., above the glottis)
2. Particulate and chemical injury to lower airways and lung parenchyma (i.e., below the glottis)
3. Metabolic derangements caused by disruption of oxygen kinetics at the cellular level (e.g., CO poisoning)

22. What are three ways smoke from a fire causes death by asphyxiation?

1. Consumption of oxygen by the fire, as well as products of combustion, can lead to asphyxiation. During fire in a closed space, room air oxygen concentration drops from 21% to between 10% and 15%, increasing asphyxiation risk.
2. CO is an odorless, colorless product of incomplete hydrocarbon combustion that rapidly transits the respiratory epithelium and out-competes oxygen for binding sites on hemoglobin, crippling oxygen delivery in the affected patient.

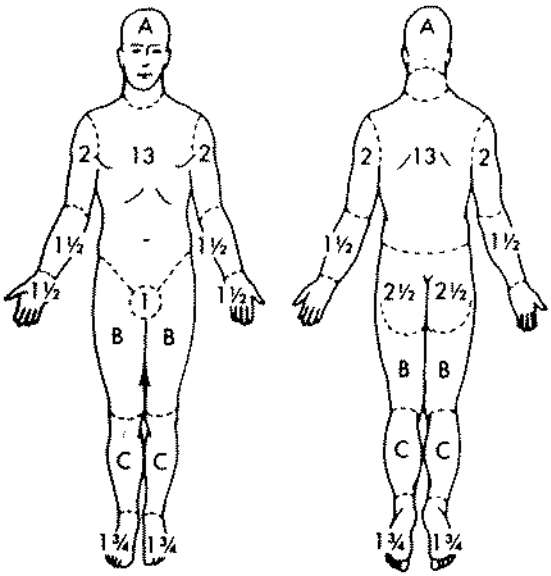

Figure 93-2. Classic Lund and Bowden chart. The best method for determining percentage of body surface burn is to mark areas of injury on a chart and then compute the total percentage according to patient's age. *(From Artz CP, Yarbrough DR 3rd: Burns, including cold, chemical, and electrical injuries. In Sabiston DC Jr, editor:* Textbook of surgery, *ed 11, Philadelphia, 1977, Saunders.)*

3. Cyanide gas is produced in the incomplete combustion of carbon- and nitrogen-containing materials such as wood, plastics, and synthetic polymers. The blood levels of cyanide are often fleeting and unreliable, making the diagnosis difficult, and a high index of suspicion should be maintained. Cellular oxygen utilization is impaired at the electron transport level, and despite normal arterial oxygen tension, patients suffer from tissue anoxia. Lactate levels above 8 mmol/L may be supportive in the diagnosis.

23. What should I look for in a patient with CO exposure?

Patients with significant CO exposure have nonspecific symptoms, such as nausea, headache, and presyncope with signs of compensation for cellular hypoxia, such as tachycardia and tachypnea. It is important to note that most pulse oximeters cannot distinguish CO-bound hemoglobin (CO-Hb) from oxyhemoglobin, and even in significant exposure, pulse oximetry readings are normal. Measurements of CO-Hb with cooximetry typically reveal levels as high as 5% in smokers, but levels in the range of 10% to 15% are consistent with CO poisoning. CO poisoning accounts for 80% of smoke inhalation deaths, with most patients succumbing within the first 24 hours after exposure.

24. How is CO poisoning treated?

Treatment is typically 100% oxygen therapy for hours, with hyperbaric oxygen therapy employed when CO-Hb levels exceed 25% (see Chapter 73).

25. How do I manage patients with cyanide toxicity?

Hydroxycobalamin given intravenously is the recommended treatment (see Chapter 73).

26. What are characteristics of patients whose burns can be managed in the outpatient setting?

- There is no question of airway compromise.
- The wound must be generally less than 10% TBSA, so fluid resuscitation is unnecessary.
- Children must be able to take in adequate fluid by mouth.
- The family must have the resources to support an outpatient care plan.

27. What are the elements of an outpatient management plan for the burn patient?

Once it is established that a burn patient can be managed as an outpatient, a thoughtful, individualized care plan should be formulated. The elements of the plan of care include pain control, wound cleansing, and topical wound care. Education of the patient and caregiver on explicit return precautions, routine burn clinic follow-up visits, and expected long-term course should be covered. Mandatory 24-hour recheck with a physician skilled in burn management or in the ED should be emphasized. Documentation of this management and education is important.

28. What about tetanus prophylaxis?

Tetanus should be updated for all burn patients, and if uncertainty exists as to the interval since the last tetanus immunization has been given, it is advised that the patient receive a tetanus booster at the time of initial evaluation.

29. How are children who suffer burns different from adults?

Children younger than 2 years have increased morbidity and mortality from burns, and require a modified approach. Resuscitation fluids in children are calculated differently. Total fluid administered over the first 24 hours is calculated as:

$$3 \text{ to } 4 \text{ mL} \times \text{kg body weight} \times \%\text{TBSA burned}$$

This is given (as in adults) as 50% over the first 8 hours after the burn, and the remainder over the subsequent 16 hours. In children weighing less than 30 kg, the intravenous (IV) flow rate should be adjusted to maintain slightly more brisk urine output at a rate of 1 mL/kg/h. Additionally, children younger than 2 years are particularly susceptible to hypoglycemia as glycogen stores are depleted in a postburn hyperadrenergic state. Careful glucose monitoring is warranted, with a transition to dextrose-containing electrolyte solutions (e.g., half-normal saline with 5% dextrose) if hypoglycemia develops.

30. What characteristics of burns suggest nonaccidental trauma?

See Chapter 66.

31. In pediatric patients, what specific concerns should be considered in household electrical injuries?

See Chapter 56.

32. What about the child who bites an electrical cord and sustains a burn at the oral commissure?

Injuries at the oral commissure after a child bites an electrical cord typically require no immediate intervention beyond wound care with topical antibiotics. In these injuries, there is a small risk of lateral erosion into the labial artery in subsequent days that should be anticipated, discussed with caregivers, and prompt a return visit.

33. What are the special considerations in elderly adults suffering burns?

Elderly patients often have diminished mobility, reduced reaction time, and impaired alertness. This amounts to a decreased ability to recognize risk and avoid danger. Physiologic changes with aging, such as atrophic skin, slower epidermal turnover rates, and decreased perfusion, place elderly victims at increased risk for more severe burns, delayed healing, and prolonged recovery. Moreover, the aging of organ systems, such as pulmonary, renal, and cardiovascular systems, threaten the burn patient's ability to compensate and overcome the physiologic insult of the initial injury.

34. Are there any issues in treating elderly adults who sustain burns?

Fluid resuscitation is more difficult than in younger adults, with the typical goal of optimizing cardiac preload having been shown to result in excessive fluid administration in elderly populations. Large increases in mortality rates accompany inhalation injury in this population, and airway management is often required earlier.

The elderly typically suffer burns in the home from scalding, flame, or both. Solitary living conditions contribute to delay in seeking medical attention and jeopardize the success of an outpatient care plan. Pain management further challenges providers to adequately treat this population. Underlying renal or liver impairment may limit analgesic choices, and physicians have been shown to underestimate perceptions of pain in elderly populations, further complicating plans for outpatient analgesia.

35. What is special about treating facial burns?

Silver-containing topical antibiotics (silver sulfadiazine) should be avoided in treatment of facial wounds out of concern for pigmentation changes in the face with potential cosmetic consequences. Triple antibiotic ointments are adequate. Consider corneal injury in any burn involving the face, and examine the eyes early, because facial swelling may quickly preclude thorough evaluation. Pay careful attention to the airway for early signs of involvement, potentially indicating the need for escalation of the patient's care.

36. What are general principles in managing patients suffering chemical burns?

Patients suffering chemical burns should be considered for transfer to a burn center. However, initial steps should be taken to prevent further patient injury and protect providers. Members of the health care team should wear gloves, gowns, and facial protection while involved in patient care. Garments and jewelry that may contain chemical agents and prolong the damaging effects of exposure must be removed. Copious irrigation should continue until the patient is symptom free or transfer is undertaken. Specific chemical exposures, such as hydrofluoric acid, petrol products (gasoline and diesel fuel), or alcohols such as phenol, require special attention and specific management that should be initiated before transfer.

WEBSITE

American Burn Association: www.ameriburn.org; accessed 3-9-15.

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QUESTIONS

1. Which of the following is the correct hourly rate for a 100-kg male with 20% TBSA partial-thickness burn during the first hour of resuscitation?
   a. 4000 mL/h
   b. 1000 mL/h
   c. 500 mL/h
   d. 250 mL/h
   The correct answer is *c*.
2. Which of the following patients with 9% TBSA hot water burns can be treated as an outpatient?
   a. 2-year-old female with partial-thickness circumferential burn on the knee
   b. 19-year-old female with epidermal burns to right shoulder and back
   c. 41-year-old male paraplegic with partial-thickness burns to the buttock
   d. 78-year-old male with full-thickness burns to perineum and abdomen
   The correct answer is *b*.
3. Which of the following categories of burn depth is associated with erythema, pain, a glistening wound surface, and brisk capillary refill?
   a. Superficial or epidermal
   b. Superficial partial thickness
   c. Deep partial thickness
   d. Full thickness
   The correct answer is *b*.

WOUND MANAGEMENT

Maria E. Moreira, MD

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 function of the injured body part. A cosmetic repair prioritizes healing with the least amount of scarring.

3. How do I remember what steps to take when repairing a wound?

Use the mnemonic *LACERATE*:

Look: Evaluate the wound to determine the most appropriate closure. Examine thoroughly for movement, sensation, and pulsation distal to the wound.
Anesthetize
Clip and clean: Clipping hair leads to less infection than shaving. Methodical irrigation is the best way to decrease infection risk.
Equipment/**E**xplore: Have everything needed for repair at the bedside, including laceration kit, gloves, suture material, and dressing. All wounds should be explored, evaluating for tendon injury or presence of foreign body. Extremity injuries should be explored while putting the extremity through range of motion.
Repair: Perform the repair. Devitalized tissue may need to be debrided.
Assess results: Reevaluate the wound when the repair is near completion to determine the need for additional sutures.
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.
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, and 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 the patient's 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?

Familiarity with underlying anatomy, especially in the regions of the face, neck, hands, and feet is important. Begin by identifying any motor, sensory, and vascular deficits. With extremity 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.

Abstract

This chapters reviews the general principles of wound management.

Keywords:

wounds, wound repair, wound management, irrigation, sutures, lacerations, primary closure, delayed primary

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 and debridement, 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 of edges and 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 debridement
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 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.
H_2O_2, Hydrogen peroxide; *SPF*, sun protection factor.

7. What is the most important step I can take to prevent infection?

Irrigation with normal saline, generating a pressure of at least 8 psi, is crucial. This can be achieved by using 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 debridement considered. Tap water is a reasonable alternative to sterile saline for wound irrigation. Detergents, hydrogen peroxide, and concentrated povidone-iodine should not be used for irrigation because they are toxic to tissues. Exploration; debridement 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 debridement 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

Table 94-2. Maximum Dose and Duration of Action of Anesthetics

ANESTHETIC	CLASS	MAXIMUM DOSAGE	DURATION
Lidocaine	Amide	4.5 mg/kg (not to exceed 300 mg)	1-2 hours
Lidocaine with epinephrine	Amide	7 mg/kg (not to exceed 500 mg)	2-4 hours
Bupivacaine	Amide	2.5 mg/kg (not to exceed 175 mg)	4-8 hours
Bupivacaine with epinephrine	Amide	3 mg/kg (not to exceed 225 mg)*	8-16 hours
Procaine	Ester	8 mg/kg (not to exceed 1 g)	15-45 minutes
Procaine with epinephrine	Ester	10 mg/kg (not to exceed 1 g)	30-60 minutes

*Can repeat bupivacaine doses once every 3 hours but should not exceed 400 mg in 24-hour period.

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 dosage of lidocaine and bupivacaine?

Table 94-2 summarizes the maximum dosage and duration of action of lidocaine, bupivacaine, and procaine, alone and in combination with epinephrine. When calculating the dosage of milligrams infiltrated, 1 mL of 1% lidocaine equals 10 mg of lidocaine, and 1 mL of 0.25% bupivacaine equals 2.5 mg of bupivacaine. Lower maximum dosages 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 dosage is met or exceeded. The caveat to that statement is that toxicity may take place at lower than maximum dosages 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 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% 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?

LET is a topical anesthetic that consists of a mixture of lidocaine (4%), epinephrine 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. Onset of action is within 20 to 30 minutes.

15. What are the contraindications to LET?

They are the same as for lidocaine or bupivacaine with epinephrine.

16. When should regional anesthesia be used?

- In wounds that would require large toxic doses of anesthetic
- In wounds in which tissue distortion needs to be avoided (i.e., vermillion border)
- In wounds in which local infiltration is very painful (i.e., plantar surface of foot)

17. 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 67).

18. What is a contaminated wound?

Any wound that has a high inoculum of bacteria is contaminated. 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

19. 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

20. Is a dirty wound the same as a contaminated wound?

No, road rash, resulting from road gravel, can appear quite dirty but 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.

21. What causes tattooing?

Tattooing is caused by 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 debridement, 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.

22. How is road rash managed?

Anesthetize the area with viscous lidocaine and circumferential or field block anesthesia. Remove all foreign bodies with the methods described previously. Consider dressing the wound with silver sulfadiazine, which greatly reduces the pain and may obviate the need for potent oral analgesics for deep, extensive, painful abrasions.

23. When is obtaining a radiograph appropriate?

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 when 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.

24. Which types of foreign bodies found in wounds are visible on radiographs?

Glass, metal, and gravel are all visible. 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.

25. 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.

26. 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 to 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.

27. Which wounds should be closed primarily

- Wounds on the extremities or torso presenting within 6 hours of occurrence
- Wounds on the face and scalp presenting within 24 hours of occurrence

The patient's medical history should also be taken into consideration when making decisions on primary closure. The clinician may decide to close a wound on the leg of a healthy young patient after 10 hours and not close a wound on the leg of a patient with diabetes and receiving dialysis after 4 hours. When making decisions to close wounds, clinicians should discuss risks and benefits of primary closure with the patient.

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, debrided, 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 monitored 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. When should secondary closure be used?

Secondary closure should 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.

31. What is the most important step when closing a lip laceration through the vermilion border?

Place 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. Close the remainder of the lip with absorbable suture. Close the skin with nonabsorbable suture.

32. When are surgical staples indicated?

Surgical staples are used to reapproximate linear lacerations not involving 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.

33. What is surgical glue, and how is it used?

Surgical glue, 2-octyl cyanoacrylate (Dermabond), is a polymer available as an alternative method 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.

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

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.
CT, Computed tomography; *MRI*, magnetic resonance imaging.

34. 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.

35. Summarize the advantages and disadvantages of the available techniques for wound closure.

See Table 94-3.

36. Which sutures are used for specific locations, how is the wound repaired, and when do I remove the sutures?

See Table 94-4.

37. How are bites treated?

See Figure 94-1.

38. What should be included in all follow-up instructions?

Include instructions for local wound care, signs of infection, and recommended 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.

39. How do I remember the direction of the lines of skin tension?

You do not, unless you have a photographic memory. Refer to Figures 94-2 and 94-3.

40. 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

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 (SC, 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 SC 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
Nail beds	5-0 SA	Gentle, meticulous placement to obtain even edges. Replace nail under cuticle	Allow to dissolve

From Markovchick V: Soft tissue injury and wound repair. In Reisdorff EJ, Roberts MR, Wiegenstein JG, editors: *Pediatric emergency medicine*, Philadelphia, 1993, Saunders, pp 899–908.
SA, Synthetic absorbable sutures such as Vicryl and Dexon; *SC*, subcutaneous.

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 debridement; 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.

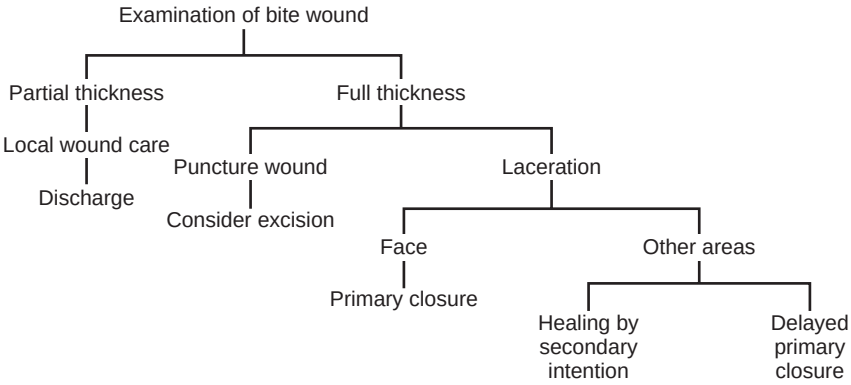

Figure 94-1. Algorithm for treatment of bites.

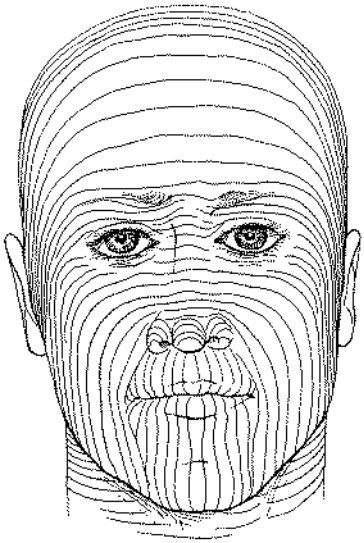

Figure 94-2. Direction of the lines of skin tension for the face. *(From Marx J, Hockberger R, Well R, et al: editors:* Rosen's emergency medicine: concepts and clinical practice, *ed 5, Philadelphia, 2002, Mosby, pp 738.)*

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. If soap is used, irrigation should follow.
5. A stiff brush (toothbrush) will remove ground-in dirt.

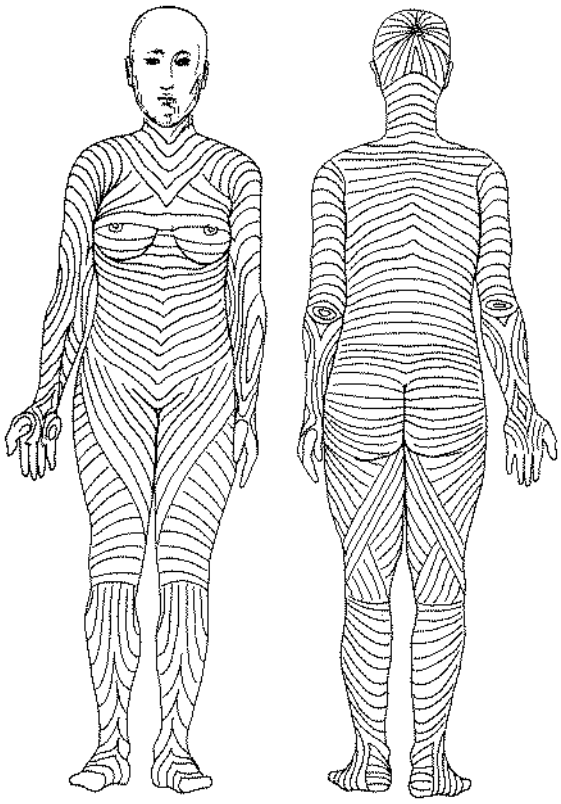

Figure 94-3. Direction of the lines of skin tension for the body. *(From Marx J, Hockberger R, Well R, et al: editors:* Rosen's emergency medicine: concepts and clinical practice*, ed 5, Philadelphia, 2002, Mosby, pp 739.)*

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QUESTIONS

1. Under general principles of wound closures, which of the following wounds would you decline to close on initial presentation?
   a. Leg laceration in a health 22-year-old patient within 6 hours of injury
   b. Clean arm laceration in a 45-year-old diabetic patient within 2 hours of injury
   c. Scalp laceration in a 45-year-old intoxicated male 19 hours after injury
   d. Leg laceration in a 22-year-old patient with end-stage renal disease 10 hours after injury
   The correct answer is *d*.
2. In which of the following clinical scenarios is a regional nerve block indicated?
   a. 2-cm laceration of the forearm
   b. 3-cm laceration of the dorsum of the foot
   c. 8-cm laceration of the scalp
   d. 2-cm laceration of the lip extending across the vermillion border
   The correct answer is *d*.
3. Which of the following factors does not increase the risk of wound infection?
   a. Advanced patient age
   b. Immunocompromised state
   c. Use of tap water for irrigation
   d. Foreign body present in the wound
   The correct answer is *c*.

CHAPTER 95

ACUTE PSYCHOSIS

Janetta Iwanicki, MD

1. What is psychosis?

Psychosis is a dysfunction of the perception of reality. According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5),* key features include delusions, hallucinations, and disorganized thinking and speech.

2. What are delusions?

From the *DSM-5* (2013), "delusions are fixed beliefs that are not amenable to change in light of conflicting evidence."

3. What are hallucinations?

"Hallucinations are sensory perceptions without external stimulation" (*DSM-5,* 2013). Hallucinations may occur in any of the five sensory modalities, although auditory hallucinations are most common.

4. How does a patient in a psychotic state typically appear upon arrival at the ED?

Patients in a psychotic state may act strangely, dress bizarrely, respond to hallucinations, harbor false and delusional beliefs, and consistently confuse the reality of events. They are commonly impulsive and in danger of acting on distorted perceptions or delusional ideas, resulting in injury or death. The patient is unable to discriminate whether the stimuli that he or she perceives are internal or external. Thinking and speech are often disorganized and incoherent. 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 airway, breathing, and circulation (ABCs), if necessary.
- Observe (quickly assess the patient's impulse control and tendency to physically act out).
- Control and manage psychotic behavior posing a danger to the patient or others, 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 in a psychotic state have little impulse control, and they cannot distinguish internal from external stimuli. Because of this dysfunction, they should always be considered a potential 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. Any history or comment that suggests violence should be taken seriously. The potential for violence 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, steps should be taken to avoid confrontation or escalation.

- Environmental: Keep the environment simple and stimulus 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.

Abstract

Acute psychosis is a cause of presentation to the ED for both organic and functional etiologies, and may represent a significant danger to the patient's health and safety.

Keywords:

psychosis, hallucinations, delusions, delirium, schizophrenia

9. How do I obtain a history for a psychotic patient?

Because acutely psychotic patients may not be able to provide an adequate history, all available collateral 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, psychosocial stressors, and psychiatric resources.
- Previous psychiatric disease: Determine whether there is organic brain disease, the use or misuse of medication, or a 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, a high percentage of missed organic diagnoses in psychotic patients was because of the lack of 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 provide structure for the psychotic patient and avoids confusion or misunderstanding.

12. What is the difference between organic and functional psychosis?

- Organic psychosis is 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., delirium, dementia, withdrawal states, and intoxications).
- Functional psychosis is a dysfunctional mental condition identified as schizophrenia, a major affective disorder, or other mental disorders with psychotic features.

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 (Wernicke encephalopathy)
- Alcohol-dependent withdrawal states
- Alcoholic ketoacidosis or hypoglycemia
- 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.* Patients with hysterical, borderline, and narcissistic personalities are prone to brief psychotic disorder, 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:

Drug toxicity
Emotional disorders
Metabolic disorders
Endocrine disorders
Nutritional disorders
Tumors and trauma

Table 95-1. *MADFOCS* Mnemonic

	ORGANIC	FUNCTIONAL
Memory deficit	Recently impaired	Remotely impaired
Activity	Hyperactivity and hypoactivity Tremor Ataxia	Repetitive activity Posturing Rocking
Distortions	Visual hallucinations	Auditory hallucinations
Feelings	Emotional lability	Flat affect
Orientation	Disoriented	Oriented
Cognition	Some lucid thoughts Perceives occasionally Attends occasionally Focuses occasionally	No lucid thoughts Unfiltered perceptions Unable to attend Unable to focus
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

Infection
Arteriosclerotic complications

17. Name the life-threatening causes of acute psychosis.

WHHHIMP mnemonic:

Wernicke encephalopathy
Hypoxia or hypoperfusion of the central nervous system
Hypoglycemia
Hypertensive encephalopathy
Intracranial hemorrhage
Meningitis/encephalitis
Poisonings

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 antiinflammatory drugs
- Anticancer agents
- Recreational drugs: Alcohol, cocaine, amphetamines

19. Is laboratory screening necessary in the workup of an acute psychotic patient?

Patients with established psychiatric diagnoses in 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 is experiencing his or her 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
- Computed tomography (CT) scan of the brain

Consider screening for toxic ingestions (e.g., acetaminophen, salicylates) in any patient who expresses suicidal ideation.

20. Are there any other clinical rules of thumb in the workup of the acute psychotic patient?

- Fever and psychosis = meningitis, encephalitis, or sepsis
- Acute psychosis and alcoholism = Wernicke encephalopathy
- Headache and psychosis = tumor or intracranial hemorrhage
- Abdominal pain and psychosis = porphyria
- Diaphoresis and psychosis = hypoglycemia, delirium tremens, sepsis, sympathomimetic intoxication
- 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 himself or herself appropriately
- If the patient has no social support system
- 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. Acute psychosis includes delusions or prominent hallucinations.
2. Use the least restrictive restraint (e.g., isolation, restraints, psychotropic medication).
3. A complete and thorough history and physical examination, including mental status examination, is imperative.
4. Distinguish between organic and functional disorders.

WEBSITE

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Acknowledgment

The editors gratefully acknowledge the contributions of Eugene E. Kerchner, MD, and Manish Amin, DO, authors of this chapter in the previous editions.

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QUESTIONS

1. Hallucinations may be:
   a. Auditory
   b. Visual
   c. Olfactory
   d. All of the above
   The correct answer is *d*.
2. Acute psychosis in a patient with a known history of schizophrenia should be evaluated with:
   a. CT of the head
   b. Lumbar puncture
   c. Complete physical examination
   d. Electrocardiogram (ECG)
   The correct answer is *c*.
3. Evaluation of a patient with acute psychosis should involve gathering information from:
   a. Only the patient
   b. The patient's family and friends
   c. EMS and police
   d. *b* and *c*
   The correct answer is *d*.

CHAPTER 96

DEPRESSION, SUICIDE, AND POSTTRAUMATIC STRESS DISORDER

Douglas A. Rund, MD

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)
- 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 symptoms are necessary for the diagnosis of depression, one of which must be the loss of interests or depressed mood:

SIG E CAPS
Sleep disturbance
Interests/mood
Guilt
Energy
Concentration
Appetite disturbance
Psychomotor changes
Suicidal 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* describes 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 syndrome

Abstract

Depression is a mood disorder characterized by a sad mood or loss of interest or enjoyment. Specific clinical features provide criteria to diagnose both unipolar and bipolar depressive disorders. Depressive disorders can be caused by a range of medical disorders and medications. Suicide is the most serious complication of depressive disorders. Evaluating suicide risk is an important part of the assessment of the patient with mood disorder or the patient who has attempted suicide. Posttraumatic stress disorder (PTSD) is a stressor-related disorder that can follow direct exposure to a severe traumatic event. Features include reexperiencing the trauma, avoiding stimuli that remind the person of the event, and marked alterations in arousal and reactivity.

Keywords:

depression, suicide, posttraumatic stress disorder (PTSD)

Neurologic disorders
- Cerebrovascular accidents
- Subdural hematoma
- Multiple sclerosis
- Brain neoplasm
- Parkinson 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 taking 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, emergent psychiatric consultation is warranted for consideration of inpatient psychiatric hospital admission. 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
- 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 serious

side effects (life-threatening TCA overdose) 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 safety profile, and ease of use with fewer adverse effects. These are fluoxetine (Prozac), paroxetine (Paxil), sertraline (Zoloft), citalopram (Celexa), fluvoxamine (Luvox), and escitalopram (Lexapro).

Newer 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 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 midpotency neuroleptics.
- Many other commonly used agents have dosage-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 schedule a follow-up visit with outpatient psychiatric care.

13. What is the most serious complication of depression?

Suicide is the most serious complication. 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. However, it is important 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
- 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
- Future plans (of the patient who has attempted suicide)

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 to 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 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 patients with HIV 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 much higher in the first year after an attempt, especially for people older than 45 years. 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 attempts 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 and co-workers 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 (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.

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 repeated 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

Modified from Hockberger RS, Rothstein RJ: Assessment of suicide potential by non-psychiatrists using the SAD PERSONS score. *J Emerg Med* 6:99–107, 1988; and Hockberger RS, Smith M: Depression and suicide ideation. In Wolfson AB, editor: *Clinical practice of emergency medicine*, ed 4, Philadelphia, 2005, Lippincott Williams & Wilkins, pp 637–639.

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 years; high risk-to-rescue ratio; serious mental illness; alcoholism; drug addiction; living alone with poor social support; and hopelessness, helplessness, or exhaustion.

KEY 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

POSTTRAUMATIC STRESS DISORDER

33. What are the clinical features of posttraumatic stress disorder (PTSD)?

- Exposure to a traumatic event
- Intrusive thoughts or events
- Avoidance of reminders of the event
- Negative effects on cognition and mood
- Marked alteration of arousal

34. What kind of traumatic events can cause PTSD?

PTSD may be caused by such events as exposure to war as a combatant or civilian, threatened or actual physical assault from sexual violence, torture, incarceration as a prisoner of war, and natural and manmade disasters. The exposure must be actual direct exposure (not indirect through media). The disorder is more severe and long lasting when the traumatic event is caused directly by other human beings or purposely inflicted pain (e.g., torture or sexual violence).

35. What is meant by intrusive thoughts or events?

One or more intrusive events may be associated with the original emotional trauma. Such events include memories, dreams, and flashbacks.

There may be intense psychological or physiologic reactions to internal or external cues that symbolize the event.

36. What kinds of avoidance behavior are noted?

Stimuli associated with the trauma are avoided. This can include avoiding talking about the event and avoiding people or situations that are reminders.

37. What are negative alterations in cognition and mood associated with PTSD?

Such features can include memory loss, anger guilt, loss of interest in previously enjoyed activities, feeling detached from others, or anhedonia.

38. What kinds of alterations in arousal are noted?

The patient may experience an intense startle response. Other alterations include irritability or insomnia. Patients may also engage in risky or aggressive behaviors.

39. What are special considerations in the ED evaluation of patients with suspected PTSD?

The clinician must make sure to consider thorough clinical evaluation for medical causes of symptoms such as head injury, substance intoxication, or withdrawal.

40. What are treatment consideration in PTSD?

Psychiatric consultation or referral is indicated for patients with PTSD. Therapy initiated soon after a traumatic event may be helpful in minimizing subsequent symptoms and treating the disorder.

WEBSITE

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QUESTIONS

1. What is the best description of primary depression?
   a. Causally unrelated to any other medical or psychiatric illness
   b. Problem that causes the most patient dysfunction
   c. Psychiatric condition caused by serotonin imbalance in certain areas of the central nervous system
   d. Responds better to therapy with a single agent than multiple agents
   The correct answer is *c*.
2. Which of the following is a standard feature of suicide precautions?
   a. Chemical restraint
   b. Discontinue all psychoactive medications
   c. Patient search
   d. Show of force
   The correct answer is *c*.
3. What is an example of avoidance behavior in PTSD?
   a. Avoiding previously enjoyed activities
   b. Flashbacks only at night
   c. Memory loss and confusion
   d. Refusal to talk about certain topics
   The correct answer is *d*.

MANAGEMENT OF THE VIOLENT PATIENT

Kimberly Nordstrom, MD, JD

CHAPTER 97

1. **Is violence a problem in the ED?**
   Yes, the ED is the front door for acute care, and patients often arrive feeling, and sometimes acting, out of control. These patients require a disproportionate amount of staff attention and resources. Violence is a major problem for ED staff. The National Emergency Department Study found that at least 25% of staff did not feel safe at work (reporting they "sometimes," "rarely," or "never" felt safe). An older study looking at teaching hospitals reported 41 of 127 institutions had at least one verbal threat each day, and 23 (of the same 127 institutions) had at least one threat with a weapon each month.

2. **Why does a patient become violent in the first place?**
   When understanding violence, it is important to remember that there is more than one form of violence, in terms of intent. Those with antisocial personality disorder may become violent after they determine that their needs will not be met. In fact, they tend to be quite ingratiating as they manipulate a situation, and then turn aggressive rapidly. More often, violence in the ED is related to the agitated patient. Common causes of agitation in ED include acute intoxication, acute withdrawal with associated delirium, metabolic disorders, trauma, infectious disease, sepsis, cardiovascular disorders, psychiatric disorders, hypoxia, and cerebrovascular disorders.

KEY POINTS: COMMON MEDICAL CONDITIONS THAT MANIFEST AS VIOLENT BEHAVIOR

Agitation from General Medical Condition

Closed head injury or intracranial hemorrhage
Infection causing encephalitis or meningitis
Encephalopathy (particularly from liver or renal failure)
Exposure to environmental toxins
Metabolic derangement (e.g., hyponatremia, hypernatremia, hypocalcemia, hypoglycemia)
Hypoxia
Thyroid disease
Seizure (postictal)
Ingestion or overdose of medications

Agitation from Intoxication/Withdrawal

Alcohol
Recreational drugs (cocaine, ecstasy, ketamine, bath salts, inhalants, methamphetamines)
Medications (opioids, benzodiazepines, barbiturates)

Agitation from Psychiatric Disease

Psychotic disorders
Mania
Agitated/irritable depression
Anxiety disorders
Personality disorders

3. **What can hospitals do to decrease the risk of violence?**
   One way for hospitals to help decrease the risk of violence is to have appropriate safety protocols in place. Hospital grounds should be monitored by roving patrol and/or closed-circuit security cameras. Access to care areas should be controlled. This should include having key entry points guarded by

Abstract

Violence in the ED is all too common, and is a source of disability and burnout for all staff. Violence comes in two basic forms: intentional and semipurposeful. The main way to protect against purposeful violence is to have measures in place to keep weapons out of the ED. For semipurposeful violence, often associated with agitation, prevention begins with identification. If identified early, the patient's behavior may be deescalated through techniques or medications. If not, and the patient becomes frankly violent, physical restraints may become necessary. This chapter will lead you through each of these concepts and help you build your armamentarium with useful tools.

Keywords:

violence, agitation, aggression, deescalation, restraints, emergency medications, debriefing

security. Also, though less popular in some locations, having patients enter through metal detectors could prevent entrance of weapons into the facility.

For violence caused by agitation, the best way for hospitals to decrease risk is to make sure that key personnel are trained in verbal deescalation of agitation. Also, protocols should be in place, so that if a patient were to lose complete control, the nearest staff member has a way to alert other staff of this quickly. This could be in the form of a code word (if other staff are around) or panic button for more isolated rooms. The protocol should also ensure that security officers are quickly summoned. Last, hospital staff should be educated in the proper application of physical restraints.

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 challenge to authority.
- Remove 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?

If there is already a key person in place working on deescalating the patient, the physician should support this and not take over. There should always be one point person when trying to calm a patient. Too many people getting involved and directing the patient may cause the patient to feel even more overwhelmed, leading to increased agitation.

In the event that the physician is the first on the scene or has good experience with calming patients, deescalation techniques should be initiated as soon as possible. The American Association for Emergency Psychiatry has published guidelines around agitation, including use of verbal deescalation and voluntary medications. They supported the use of the 10 Domains of Deescalation as a working construct. As the patient is being slowly calmed through verbal deescalation, it is also a good idea to offer or administer medications. Remember basic principles, such as use of nonconfrontational body language (uncross arms), empathy, and the use of offering simple things to help the patient feel more in control, such as food, water, nicotine replacement, and dimmed lights.

KEY POINTS: DEESCALATING AN AGITATED/VIOLENT PATIENT

10 Domains of Deescalation	Tips
1. Respect personal space.	Stay two arm lengths away from agitated patient.
2. Do not be provocative.	Watch body language, tone, and choice of words.
3. Establish verbal contact.	Only one person interacts with patient.
4. Be concise.	Keep wording simple.
5. Identify wants and feelings.	Ask patient what usually helps when he or she is feeling this way.
6. Listen closely to what the patient is saying.	Acknowledge that you are listening.
7. Agree, or agree to disagree.	Agree, when you can.
8. Lay down the law and set clear limits.	Clearly state unacceptable behaviors (screaming is not OK, but crying is).
9. Offer choices and optimism.	Offer a choice, but only if a choice is feasible. (Would you prefer Haldol or Risperdal?)
10. Debrief the patient and staff.	It is often helpful to talk about tense situations after the fact.

6. What if that doesn't work?

If verbal deescalation and voluntary medications are of little effect, and the patient is becoming more out of control, the room should be cleared of all movable furniture and anything else that could be used as a weapon. If multiple staff, including security, are not already in place, they should be quickly called. As the patient loses control, risk of violence toward self and others greatly increases. Physical restraints may become necessary. Physical restraints should be placed only after enough qualified staff are available to safely immobilize the patient and place restraints. The number of staff needed to place restraints varies depending on a patient's size and physicality but should never be less than five: 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. All four limbs should be restrained, and as the patient calms, he or she can slowly be taken out one or two limbs at a time.

For the disoriented (delirious or demented) patient who may be pulling out intravenous (IV) lines, two-point (arm) restraints may be adequate for safety.

7. What do I need to remember when physically restraining a patient?

First, the team needs to remain calm. When working with an agitated patient, it is very common for the team to become more "pumped up," because their epinephrine levels might be high. This can lead to being more physical with the patient than is necessary. Before placing restraints, each member should know which limb is assigned to him or her. This should be a coordinated event and not haphazard. The patient needs to be immobilized and placed onto a bed or gurney. Restraints need to be snug but not tight. Each person placing the restraint should check that one finger can move easily between the restraint and patient's skin.

Patients can never be restrained in prone position because of the risk of suffocation, but patients can have bad outcomes in other positions as well, necessitating continued monitoring of their physical condition. As a patient calms, the restraints should be reduced and, finally, discontinued.

Staff should be aware of all state laws, organizational guidelines (Joint Commission), and hospital regulations that relate to use of physical restraints. Basic tenants include needing to show that less restrictive measures were used but not helpful, that the patient was educated as to why he or she was placed into restraints, and what behaviors are necessary to no longer need restraints.

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. Less restrictive measures must be tried, because restraining a patient goes against a fundamental right that is afforded by the U.S. Constitution. The courts have held both physicians and hospitals liable for injuries that have occurred when violent or otherwise incapacitated patients escaped hospital grounds or were 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 behavior in the ED. Courts have routinely held that physicians may administer medications to patients without their consent if they would otherwise present an imminent risk of dangerous behavior.

9. What medications are recommended for emergency treatment of agitation?

The three main classes of drugs used to treat agitation are benzodiazepines, first-generation antipsychotics, and second-generation antipsychotics. Ketamine may also be used. All three classes have medications with a parenteral formulation. Ketamine, a dissociative analgesic with hallucinogenic properties, has also gained popularity in the ED and emergency medical services (EMS) setting for severe agitated delirium that maybe life threatening.

- Benzodiazepines: These are useful in agitation, mania, psychosis, alcohol withdrawal, benzodiazepine withdrawal, and sympathomimetic toxidromes such as cocaine or amphetamine toxicity.
- First-generation or 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 the result of their sedating properties. In 2001, the U.S. 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. When using a traditional antipsychotic such as haloperidol, it is advisable to provide protection from possible extrapyramidal symptoms (EPSs) by coadministration of an anticholinergic agent, such as diphenhydramine at a dosage of 50 mg PO, IM, or IV, or benztropine at a dosage of 1 mg PO, IM, or IV.
- Second-generation or 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. See Figure 97-1 for specific dosing.
- Ketamine is usually given IM in the setting of agitated delirium. IM dosages range from 6.5 to 13 mg/kg. Patients should be monitored for severe central nervous system (CNS) depression, respiratory depression, or laryngospasm.

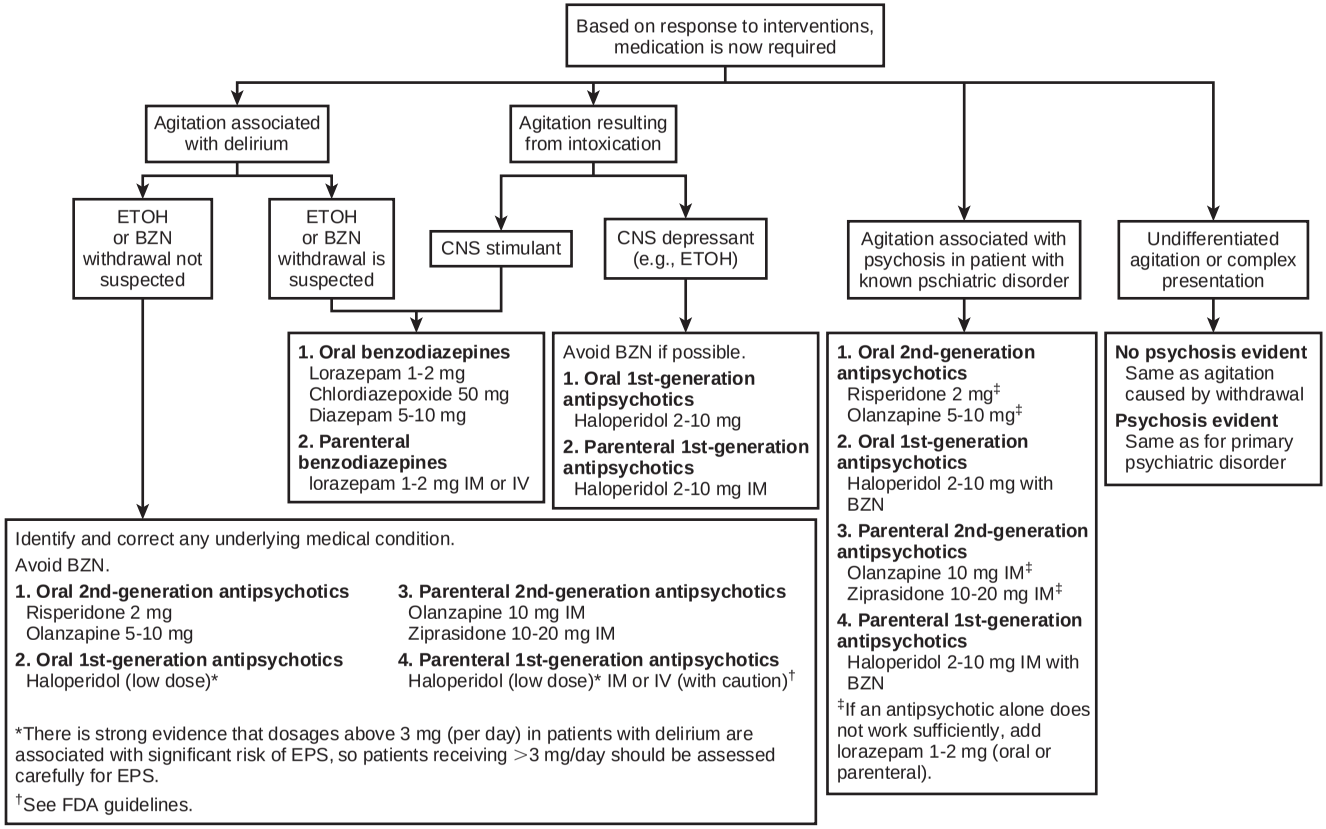

Figure 97-1. Quick reference for dosing and administration of medications for agitation. *BZN,* Benzodiazepine; *CNS,* central nervous system; *EPS,* extrapyramidal symptoms; *ETOH,* ethanol; *FDA,* U.S. Food and Drug Administration; *IM,* intramuscularly; *IV,* intravenously. *(From Wilson MP, Pepper D, Currier GW, et al: The psychopharmacology of agitation: consensus statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup.* West J Emerg Med *13:26–34, 2012. License: http://creativecommons.org/licenses/by-nc/4.0/).*

10. I normally use haloperidol; what if two doses of haloperidol have not calmed the patient?

If you have already given multiple doses of haloperidol, it is now time to add another agent. In most cases this would be a benzodiazepine, such as lorazepam or diazepam.

11. What about the pediatric patient?

The American Academy for Child and Adolescent Psychiatry has practice principles around the use of chemical and physical restraints that closely mirror principals related in this chapter. It is noted that oral medications should be offered before the administration of parental medications. Also, if the patient already takes a medication that is used for agitation, consider giving an extra dose before adding another medication.

Benzodiazepines, especially lorazepam and midazolam, are commonly used for agitation in the pediatric population. One consideration, when using this class, is that benzodiazepines can cause a paradoxic reaction, leading to increased agitation. Medications that are histaminergic, such as diphenhydramine and chlorpromazine, are also commonly used. Chlorpromazine, a low-potency typical antipsychotic, is often given at a dose of 12.5 to 25 mg (depending on the size and treatment history of the patient). This medication can be titrated for more antipsychotic benefit or used at lower dosages, with the primary benefit of calming, with some sedation. Low-dosage haloperidol (1 to 2.5 mg), a high potency typical antipsychotic, can also be considered. If the patient is naive to neuroleptics, high-potency antipsychotics should be used with caution and only at the dosage necessary for your target, because a side effect may be extreme.

12. Summarize the main side effects to watch for with these drugs.

- Benzodiazepines: Caution should be used if the patient is intoxicated on alcohol or under the effect of another CNS depressant, because respiratory depression may occur. Side effects are often dosage dependent, with more severe effects occurring at large dosages. Severe side effects include respiratory depression, somnolence, hypotension, cardiovascular depression, and coma.
- Antipsychotics: High-potency typicals (haloperidol) can cause extrapyramidal symptoms, such as akathisia (extreme restlessness), dystonias (extreme muscle spasm), and dyskinesias (abnormal movements); sedation; hypotension/hypertension; tachycardia; and arrhythmias. QT prolongation can occur, particularly in those predisposed because of a cardiac condition or who have electrolyte disorders, such as hypokalemia or hypocalcemia. Atypical antipsychotics more commonly cause orthostatic hypotension, dizziness, and akathisia. They may also cause EPSs, but are less likely than typical antipsychotics to do so. Neuroleptic malignant syndrome is a rare, but possibly deadly, reaction to antipsychotics. It is characterized by altered mentation, autonomic instability (hypertension, hyperthermia, tachycardia), and neuromuscular changes (rigidity).

KEY POINTS: MAJOR SIDE EFFECTS OF ANTIPSYCHOTICS

1. Akathisias: Inner feeling of restlessness (mimics agitation)
2. Dystonic reactions: Severe muscle spasm (e.g., torticollis)
3. Neuroleptic malignant syndrome (rare): Characterized by a triad of altered mentation, autonomic instability, and neuromuscular changes
4. Anticholinergic effects: Can lead to delirium if using high dosages or multiple agents
5. Hypotension: Consider use of fall precautions
6. Lowered seizure threshold: Especially important if other medications and/or recreational drugs are involved
7. Cardiac arrhythmias and QT prolongation: Consider ECG, especially if patient has cardiac history and hypokalemia or hypocalcemia

13. Give a quick reference on dosing and administration.

See Figure 97-1.

14. How should restrained patients be monitored?

Patients who are physically restrained are to be continuously monitored by trained staff. It is also important to frequently check vital signs, especially pulse oximetry, to prevent or quickly identify medical complications of restraints.

15. Does the ED staff need any treatment?

ED staff, especially nurses, are often targets of aggression. Physical injuries can occur, but more commonly, the damage is psychological. One way to prevent chronic effects of psychological damage is by use of immediate debriefing. Critical incident stress debriefing (CISD) has specific goals to help those who have been the victim of violence. The goals are:

- Allow the person to vent feelings and thoughts related to the event.
- Normalize a person's reaction to the event.
- Connect appropriate resources.
- Plan for reactions during subsequent future events.
- Reenter the workplace.

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Acknowledgment

The editors thank Danielle Raeburn, MD, Katherine M. Bakes, MD, and Douglas Ikelheimer, MD, for their contributions to this chapter in previous editions of this text.

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QUESTIONS

1. Should restrained patients be continuously monitored?
   a. Yes
   b. No
   c. Occasionally
   d. Never
   The correct answer is *a*.
2. Which medications should not be used when sedating a pediatric patient?
   a. Benzodiazepines
   b. Home medication used for agitation
   c. Low-dosage antipsychotics
   d. High-dosage antipsychotics
   The correct answer is *d*.
3. All of the following are common side effects from antipsychotics except:
   a. Dystonic reactions
   b. Akathisias
   c. Anticholinergic effects
   d. Neuroleptic malignant syndrome
   The correct answer is *d*.

INTIMATE PARTNER VIOLENCE

Debra E. Houry, MD, MPH

CHAPTER 98

1. **Is intimate partner violence (IPV) more of a law enforcement issue than it is a health issue?**
   No, research shows that up to one fourth of all women coming to the ED for care have experienced partner violence within the past year. Injuries and illnesses caused by abuse affect their lives more often than diseases such as hypertension, cancer, or diabetes. Survivors of IPV 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. *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.

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 women deserved the abuse, and that women could just leave their situations if they 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.

Abstract

Intimate partner violence (IPV) impacts many women both physically and mentally. In the ED, providers should recognize physical injury patterns and associations with partner violence, and screen women at risk.

Keywords:

domestic violence, intimate partner violence (IPV), injury

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 identify patients that health providers do not personally screen for IPV, because 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 important, 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). 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 head, face, neck
2. Defensive injuries
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 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?

- Have you ever been hurt or injured by a partner or expartner?
- Are there situations in your relationship where you have felt afraid?
- Has your partner ever abused you or your children?
- Do you feel safe in your current relationship?
- Is there a partner from a past relationship who is making you feel unsafe now?

14. What about screening all women for IPV?

The Institute of Medicine has recommended screening and counseling for interpersonal and domestic violence as part of preventive care. One screening tool that has been tested clinically is the Partner Violence Screen. This consists of these three questions:

1. Have you been hurt or injured in the past year by anyone? If so, by whom?
2. Do you feel safe in your current relationship?
3. 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 are 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. Do I have any legal responsibilities?

You might. As of the last article written on this in 2002, 45 states had a law that mandates reporting intentionally inflicted injuries; however, these laws vary greatly as to what injuries must be reported. Each emergency physician must be familiar with the current reporting requirements in his or her state. The most current review of laws can be found at www.acf.hhs.gov/sites/default/files/fysb/state_compendium.pdf.

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 photograph.)
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, and why does she just not 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.

Acknowledgment

Kim M. Feldhaus, MD, was an author on previous editions and developed the initial version.

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QUESTIONS

1. Why do women exposed to IPV return to the batterer?
   a. No money
   b. Need to protect children
   c. Both of the above
   The correct answer is *c*.
2. What injuries should heighten suspicion for IPV?
   a. Injury that does not fit history
   b. Defensive injury
   c. Both of the above
   The correct answer is *c*.
3. What should be done if a woman comes to the ED with suspected IPV?
   a. Send her home.
   b. Treat injuries only.
   c. Assess for injuries and safety.
   The correct answer is *c*.

EMS MEDICAL OVERSIGHT

Marlow Macht, MD, MPH, and Lara D. Rappaport, MD, MPH

KEY POINTS: MEDICAL OVERSIGHT OF EMS SYSTEMS

1. Medical oversight requires ongoing continuing education and quality assurance.
2. Medical oversight can be direct or indirect.
3. Emergency medical services (EMS) has proven benefit for treatment of cardiac arrest, respiratory distress, and traumatic injury.

1. What is medical oversight?

This is the means by which physicians give direction and authority to nonphysicians to provide emergency medical care outside of the hospital and without a physician being present to ensure that the care provided to ill or injured patients by EMS personnel is appropriate. Before organized EMS, ill and injured patients were cared for and transported by personnel who had little more than basic first-aid training. There was essentially no physician input regarding training, scope of practice, or quality of care provided by prehospital personnel. The first standardized EMS curriculum, in 1976, required increased physician involvement in education, skill acquisition, and a requirement to provide medical oversight of prehospital personnel.

2. Why is medical oversight of prehospital personnel and care important?

The importance of medical oversight lies in the concept that nonphysicians with appropriate education and training can provide advanced-level medical care safely and effectively. However, the safety and efficiency are a direct reflection of the quality of medical oversight and the relationship between providers and medical director.

3. How is medical oversight provided?

Medical oversight can be direct, with in-person, radio, or telephone contact regarding an individual patient, or indirect, using protocols and standing orders. Indirect medical control must include ongoing continuing education and quality assurance. Continuing education includes skills verification and knowledge updates. Quality assurance includes monitoring system-wide data and individual real-time or retrospective call review.

4. Is being an EMS medical director an administrative role?

Although the delegated practice of medicine necessarily involves some administrative tasks, it is imperative that the medical director function first and foremost as a physician. EMS agency directors are primarily responsible for budget, human resources, and administrative policies. Medical directors are responsible for ensuring the provision of clinically sound prehospital medicine.

5. Who are the key stakeholders in an EMS system?

The primary stakeholder of an EMS system must be its current and future patients. In common with public health physicians, the EMS medical director has a responsibility to the population as a whole, and must weigh the impact of clinical care decisions for patients currently under care to those who will next call for care.

The other stakeholders in an EMS system are the elected officials who represent the population and fund components of the system: the government and third-party payers, the hospitals and health systems, the response agencies, and the personnel who provide EMS response.

6. In what conditions has EMS been demonstrated to have proven benefit?

EMS is the key determinant in survival from out-of-hospital cardiac arrest; with the key EMS interventions being defibrillation and high-quality cardiopulmonary resuscitation (CPR) (with minimally interrupted compressions and minimizing perishock pause). EMS also assists in

Abstract

Medical oversight of emergency medical services (EMS), through ongoing continuing education and quality assurance, prevents mortality in patients with time-dependent illness and injury.

Keywords:

emergency medical services (EMS), medical oversight, medical direction, prehospital care, paramedics, cardiac arrest, emergency medical technician (EMT)

minimizing death and disability by transporting seriously injured patients to trauma centers, providing bronchodilators to patients with reactive airway disease; providing noninvasive positive pressure ventilation to patients with chronic obstructive pulmonary disease (COPD), exacerbation, or pulmonary edema; and providing a package of care to patients with ST-segment elevation myocardial infarction (STEMI).

7. In what other, difficult-to-study ways might EMS be beneficial?

Some EMS practices are unlikely to be studied in a randomized controlled trial, yet are likely to benefit patients. For example, the provision of intravenous (IV) dextrose to the altered hypoglycemic patient is clearly of benefit but unlikely to be studied.

Other considerations include providing analgesia to the patient with a long bone fracture, assessing death in the out-of-hospital setting, or recognizing nonaccidental trauma. The prehospital provider performs a key role in gathering data from the scene of the call that often will have a significant influence on the ED and hospital course.

8. What are some key controversies in current EMS practice?

The ideal approach to prehospital airway management remains a major question. In cardiac arrest, it is unclear if passive oxygenation, bag-valve-mask ventilation, supraglottic airways, or endotracheal intubation confer a survival advantage. Prehospital endotracheal intubation for trauma or severe respiratory distress has both supporters and detractors. A well-done randomized controlled trial demonstrated similar outcomes when comparing prehospital intubation to bag-mask ventilation for pediatric patients; however, the generalizability may be limited. When endotracheal intubation is performed, a growing body of literature supports video laryngoscopy as the preferred technique.

The use of long back boards for spinal immobilization has long been a standard of care in EMS but does not have evidence of benefit and does have evidence of harm. Alternatives include the use of scoop stretchers, vacuum mattresses, or placement on the gurney. However, rigorous data remain lacking as to which is preferred.

9. What are the common performance benchmarks of an EMS system?

Modern EMS systems should be able to demonstrate timeliness of response and intervention to patients with cardiac arrest, acute coronary syndrome, respiratory distress, and major trauma, at a minimum. Other potential areas of benchmarking include treatment of seizures, management of stroke, and treatment of pain.

Patient-centered outcome measures (for example, neurologically intact survival from cardiac arrest) should be the highest priority. Alternatives include measurement of processes proven to improve outcome (for example, minimizing perishock pause or application of noninvasive positive pressure ventilation for COPD exacerbation).

10. Who are the members of the EMS workforce?

Prehospital medicine in the United States is practiced by emergency medical responders, emergency medical technicians (EMTs), advanced EMTs, and paramedics. In some cases, nurses, nurse practitioners, physician assistants, and physicians also provide prehospital care.

11. What skills can be performed by prehospital providers at different levels of training?

Emergency medical responders may perform basic airway management, including oropharyngeal airways; manually stabilize the cervical spine or extremity fractures; control bleeding; and apply an automated external defibrillator (AED). In addition to the emergency medical rescue (EMR) skills, EMTs may also insert nasopharyngeal airways, assist patients in taking their own prescribed medications, splint, and provide spinal immobilization. Advanced EMTs are permitted to insert supraglottic airways, establish IV access, and provide several key oral, subcutaneous, intramuscular, and IV medications. Paramedics may perform endotracheal intubation and cricothyrotomy, decompress the pleural space, and provide cardioversion, manual defibrillation, and transcutaneous pacing.

Nurses who work in the prehospital setting are primarily involved in critical care and aeromedical transport. Nurse practitioners and physician assistants are relatively rare in the prehospital setting but perform tasks commensurate with their training in a mobile capacity. Physicians who directly provide routine care in the prehospital setting most commonly are involved with critical care transport.

12. How important is physician involvement in education and training for prehospital personnel?

Physician involvement in the development of educational programs and the delivery of training provides an opportunity to interact with and provide guidance to the personnel. Having personal knowledge of the skill level of the prehospital providers develops a level of trust in their ability to assess and respond appropriately to most situations they encounter.

13. What are the different models of EMS systems?

EMS systems are most commonly fire department based, third service (separate from police or fire), private, or hospital based. In the United States, fire department–based systems are most common.

In contrast, in many European countries, ambulances are often staffed by physicians. In a globally more common model, physician extenders staff ambulances.

14. What are the strengths and weaknesses of fire department–based EMS?

Fire department–based systems have the advantage of readiness, the potential to have employees trained both as firefighters and EMS personnel, high job satisfaction, and low attrition. The disadvantages include the cost and fixed location of fire stations and the competing responsibilities of fire suppression and hazardous materials response versus medical care.

15. What are the strengths and weaknesses of third-service EMS?

Third-service systems (so named as a public safety service separate from fire and police) have the advantage of being able to focus exclusively on EMS in hiring, training, and staffing. Because EMS agencies can bill for their services, they can be more financially self-sufficient than fire department–based agencies. The potential disadvantages include the need to integrate response with other public safety agencies, and the potential for duplication of responses.

16. What are the strengths and weaknesses of private sector EMS?

Private sector EMS agencies, similar to third-service agencies, have the advantage of being able to focus exclusively on EMS. In addition, they may be free of municipal hiring restrictions and can bring the benefit of expertise from serving multiple different communities. However, private EMS agencies also have a fiduciary duty to shareholders. This responsibility can make recruitment and retention of EMS personnel more challenging.

17. What are the strengths and weaknesses of hospital-based EMS?

Hospital-based systems bring the resources of a large health care organization to EMS. This can include expertise in quality improvement, purchasing and supply chain management, and information technology. Being tied to a single hospital system can result in a perceived or actual conflict of interest in destination choices, and hospital-based systems also face challenges in integration with other public safety agencies.

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QUESTIONS

1. Which of the following best determines survival from out-of-hospital cardiac arrest?
   a. Advanced airway management
   b. Pharmacologic therapy
   c. Defibrillation and high-quality CPR
   d. Rapid transport
   The correct answer is *c*.
2. Medical oversight includes which of the following?
   a. Development of budgets
   b. Hiring of EMS personnel
   c. Determining staffing levels
   d. Retrospective call review
   The correct answer is *d*.
3. Which is a prehospital therapy with proven benefit based on controlled trials?
   a. Noninvasive positive pressure ventilation for COPD exacerbation
   b. Returning to the hospital using lights and sirens
   c. Administering IV dextrose to hypoglycemic patients
   d. Using a long spine board for transport
   The correct answer is *a*.

CHAPTER 100

DISASTER MANAGEMENT

Elena Garcia, MD, and Christopher B. Colwell, MD

1. Define the term *disaster*.

A disaster is any situation that disrupts normal community function, overwhelms the community's ability to respond to the situation, and threatens the safety or health of the citizens. Simply stated, a disaster is best described as an event where the needs exceed the available resources. It is not defined by the size or nature of the event, but rather by the community's ability to respond to it. It is easy to see how an airplane crash with 400 people injured is a disaster in any community; even the best resources will be quickly overwhelmed by the need. But consider the impact a single vehicle rollover crash with five critical patients might have on the limited resources of a rural community. The area may have only one ambulance, a very small number of prehospital personnel with advanced skills, and no local hospital, which will necessitate that the ambulance and EMS personnel leave their jurisdiction to transport critically ill patients, stripping the community of all of their usual EMS resources. The similarity between these two scenarios is the extent to which the incident outstripped the available resources in the area.

Disaster: Needs > Resources

2. What is the difference between a mass casualty incident (MCI) and a disaster?

An MCI is an incident that produces a large number of casualties, which generally leads to a medical disaster where patient need exceeds locally available resources. A disaster may imbalance the community's ability to ensure safety of its citizens and may cripple the infrastructure of an area.

3. Are all disasters MCIs?

No, not all disasters are MCIs. For instance, a flood can cause significant damage to homes, communication lines, access to food and other services for a community, and displace many from their homes, but it may not cause a significant number of casualties and thus may not be a true MCI. Disasters may be natural (floods, tsunamis, earthquakes, tornadoes), man-made (plane crash, train crash, industrial explosions, fires, chemical spills, radiation leaks), and even terrorist-related (biologic, chemical, explosive, radiologic, or nuclear events).

4. How is an MCI different from a mass gathering?

A mass gathering has been defined as an event of more than 1000 people (sometimes defined as those with more than 25,000 people) gathered at a specific location for a specific period of time for a specific purpose. Importantly, this creates a situation where the large gathering of persons may result in a delayed public safety response to medical emergencies by limiting access to patients as a result of the environment, the location, or mere crowd dynamics. Unlike an MCI, which is typically unanticipated and potentially catastrophic (such as the New York Twin Towers collapse of September 11, 2001, or the Boston marathon bombing in 2013), the medical response for a mass gathering can be planned, coordinated, and executed to offset a potentially disastrous scenario. However, a mass gathering may become an MCI if circumstances threaten the health and safety of the event patrons (e.g., unusually hot climate and inadequate access to water causing hundreds of people to suffer from heat exhaustion and heat stroke).

5. Why is there a need for disaster planning?

The quality of medical care in general is tied directly to the experience of the practitioner. Proficiency of a given medical intervention depends on the frequency of performance of that intervention. In a disaster, people are trying to quickly perform what they do not ordinarily do, often in an unfamiliar environment, and therefore it makes sense to plan and exercise the contingencies regularly. No matter how experienced someone is, the level of care, resources, and framework for resource management undergo major alterations during a disaster. The goal is to provide the greatest amount of good for the greatest number of people; thus there is an altered standard of care.

Abstract

This chapter provides an overview of disaster response and incident command.

Keywords:

disaster, mass casualty incident (MCI); all-hazards approach; hazard vulnerability assessment; incident command system (ICS); National Incident Management System (NIMS); incident command; planning section; logistics section; operations section; finance section; triage; simple triage and rapid transport (START); sort, assess, life-saving interventions, and treat/transport (SALT); critical incident stress management (CISM)

6. Define the all-hazards approach to disaster planning.

To have the right contingencies in place, it is essential to know what the potential hazards are in a given area and the threat that they pose. In an all-hazards approach, a review called a *hazard vulnerability analysis* is done, usually annually, to identify potential disaster etiologies and sites that could happen in a community, such as a chemical plant explosion, a train crash, or an earthquake. Each event is ranked by likelihood and then by the extent of the impact it would have on the ability to provide patient care. Based on this ranking, an agency, hospital, or community can begin to develop emergency plans to deal with the most likely events to ensure that lines of authority are established, appropriate communications occur, and all involved parties understand their roles and responsibilities. A disaster plan ideally should also implement response activities that are as close as possible to normal daily operations.

7. What are the four phases of a disaster response?

1. Activation: Initial notification that the incident, establishment of an incident command structure, and response to the incident with attention to scene safety for first responders
2. Implementation (response): Search and rescue of victims, triage, and initial stabilization and transport of patients
3. Mitigation: Hazards controlled, treatment provided to patients
4. Recovery: Responders return to normal operations, restock supplies, and debrief event; displaced persons sheltered in temporary areas until they can return to their homes

8. What is an incident command system (ICS)?

An ICS is a standardized structure that provides command and control of personnel and resources at a disaster or multiagency scene. There are five key functions accomplished through an ICS:

1. Incident command
2. Planning
3. Operations
4. Logistics and supply
5. Finance

Using an ICS improves the ability of multiple agencies to work together, because they are using common terminology and structure.

9. What is the National Incident Management System (NIMS)?

NIMS, developed by the Department of Homeland Security, standardizes the ICS structure to be used by emergency medical services (EMS) agencies, fire departments, law enforcement, and hospitals in an effort to enhance response coordination at all levels and for all types of incidents.

10. Does ICS have to be used for every incident?

ICS is not required for every disaster or incident, and in a small incident may practically be only one person who performs several functions to manage the response. However, for large-scale incidents involving multiple local, and perhaps even federal, agencies, NIMS mandates the use of a formal ICS (Fig. 100-1).

11. Describe each of the five key functions in ICS?

1. Incident command: Conducts and oversees the overall management of the incident response
2. Planning: Determines what is needed to manage the incident
3. Logistics: Obtains and supplies what is needed to manage the incident
4. Operations: Uses what is needed to manage the incident
5. Finance: Pays for it all

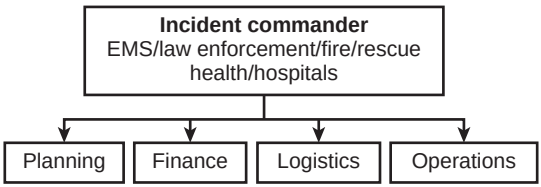

Figure 100-1. Incident command system (ICS) structure.

12. What is the weakest link in a response to an incident?

The biggest problem encountered is communication. This occurs both in the field and at the hospital, and may be the result of different radio frequencies, overwhelmed cell phone towers, or simply unclear hierarchy and uncertain disaster plans.

In addition to internal communication of responders, a public information officer should be assigned to communicate with local media, which can inform the community about potential hazards, evacuation routes, and shelters and food access. Reviews of major events that have occurred have consistently identified communication as an area that represents opportunities for improvement.

13. How does triage occur at a scene?

Triage is a French word that means *to sort.* Napoleon's surgeon is thought to be the first physician to use triage on a battlefield in the 1800s to sort out soldiers who could be treated in a nearby hospital and returned to duty. In an MCI, field triage is expanded from solely evaluating an individual patient to assessing priorities to identify the sickest patients and ensure they are transported and treated first. Although several systems exist for triaging victims of multicasualty incidents, the basic concept generally identifies four groups of patients:

1. Red (immediate): Critical or immediately life-threatening illness or injury (e.g., tension pneumothorax, hypovolemic shock)
2. Yellow (delayed): Serious but not immediately life-threatening illness or injury (e.g., most types of fractures)
3. Green (minor): "Walking wounded" (e.g., anxiety attack after witnessing event)
4. Black (dead/dying, or expectant): Dead or resource-intensive victims (e.g., 100% total body surface area burn).

Triage tags or colored tape is used to clearly indicate the categories of patients to assist in rapid assessment on scene.

14. How is triage applied in a disaster situation?

It should be assumed that situation will overwhelm the available resources until evidence exists to the contrary. Therefore triage will need to occur before definitive treatment or transport until the scene has been assessed and all victims have been identified. In these incidents, the basic philosophy is that the good of the many takes precedence over the good of the few. This is different from almost every other situation health care providers face, because we typically apply all available resources to the the few that need them the most and only turn our attention to others when the sickest have been stabilized to the best of our ability. Such an approach assumes resources that may not be available in a disaster situation. Patients falling into the black triage category will not receive the same aggressive treatment they might under other circumstances in order to apply the limited resources to those most likely to benefit from them.

15. Are there any exceptions to this rule of prioritizing patients in the red category over those in the black category?

Yes, in lightning injuries where a group of people have been affected, priority will go to those in cardiac arrest, because this represents the one situation where a single intervention may immediately change that patient's outcome, whereas those that are not in cardiac arrest from a lightning strike are generally not in need of immediate medical attention.

16. What triage system is most commonly used in the United States?

The triage system currently in most common use is START (**s**imple **t**riage **a**nd **r**apid **t**reatment), which has been widely adopted and is used by organizations such as the Domestic Preparedness branch of the United States Department of Defense. A Centers for Disease Control and Prevention (CDC) working group has also proposed a national triage method referred to as *SALT* (**s**ort, **a**ssess, **l**ife-saving interventions, and **t**reat/transport). Numerous other triage systems exist, such as JumpSTART for pediatrics, MASS (**m**ove, **a**ssess, **s**ort, **s**end) triage, Fire Department of New York (FDNY) modified START triage, and Sacco triage method (STM). There is ongoing research to determine which system is the most accurate. Variables such as age (very young or very old), comorbidities, and type of incident (e.g., chemical exposure) may influence the accuracy of currently accepted triage methods.

17. Tell me more about START.

START, developed by the Newport Beach, California Fire Department and Hoag Hospital in 1983 and revised in 1994, is designed to triage a patient in less than 30 seconds. First, those able to

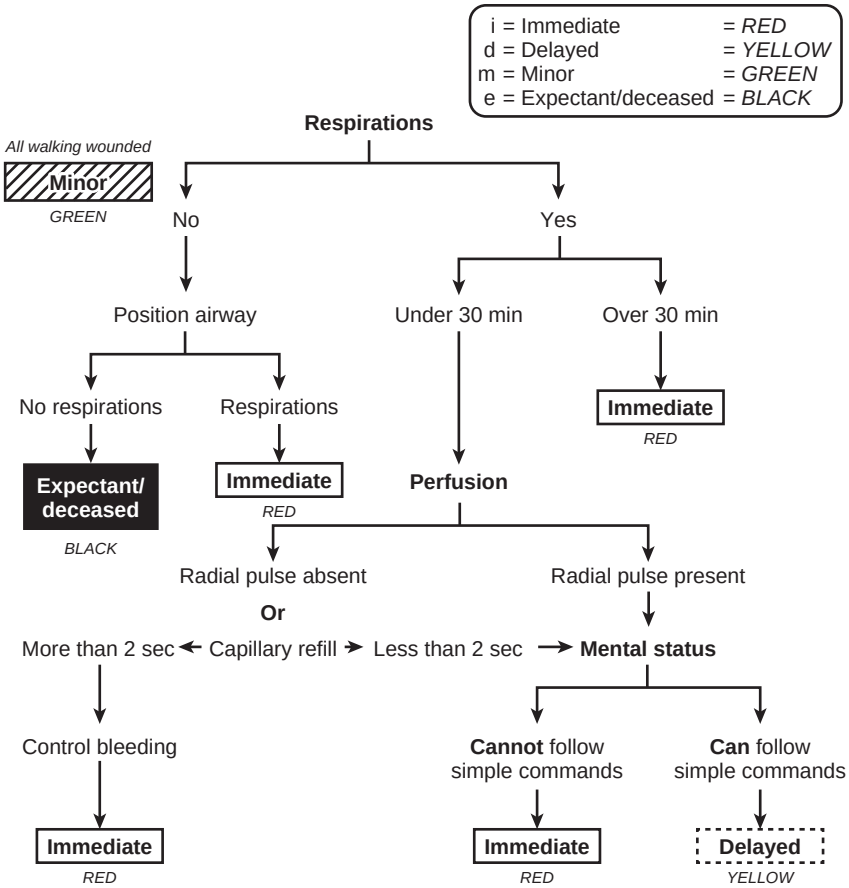

Figure 100-2. START (simple triage and rapid treatment) algorithm.

ambulate are immediately classified as *walking wounded* and sorted to a separate area (green). Priority is then given to the remaining victims on the ground. The triage officer assesses for breathing and spontaneous respirations. If breathing or circulation is abnormal and the patient is alive (has a pulse), a basic airway maneuver is attempted, such as a jaw thrust or the insertion of an oropharyngeal airway (OPA), and the patient is categorized as *red*; if the patient is not breathing and has no pulse, he or she is triaged into the black category. Assuming normal respirations, perfusion is assessed by radial pulse and capillary refill time (if absent radial pulse and capillary refill >2 seconds, patient is triaged as *red*). Finally, mental status is assessed by the patient's ability to follow commands (if no, triaged to red, and if yes, triaged to yellow). An easy mnemonic used to remember this approach is *RPM: 30 to 2, can do,* where *RPM* denotes respirations, perfusion, and mental status, and *30 to 2, can do* indicates respiratory rate less than 30, capillary refill less than 2 seconds, and ability to follow commands (Fig. 100-2). Red patients are prioritized for transport, followed by the yellow patients. Green patients may not actually need a formal evaluation at the hospital, or may be able to be transported en masse (i.e., a school bus). This sort of rapid assessment allows for maximum use of the limited resources that are available at a disaster scene.

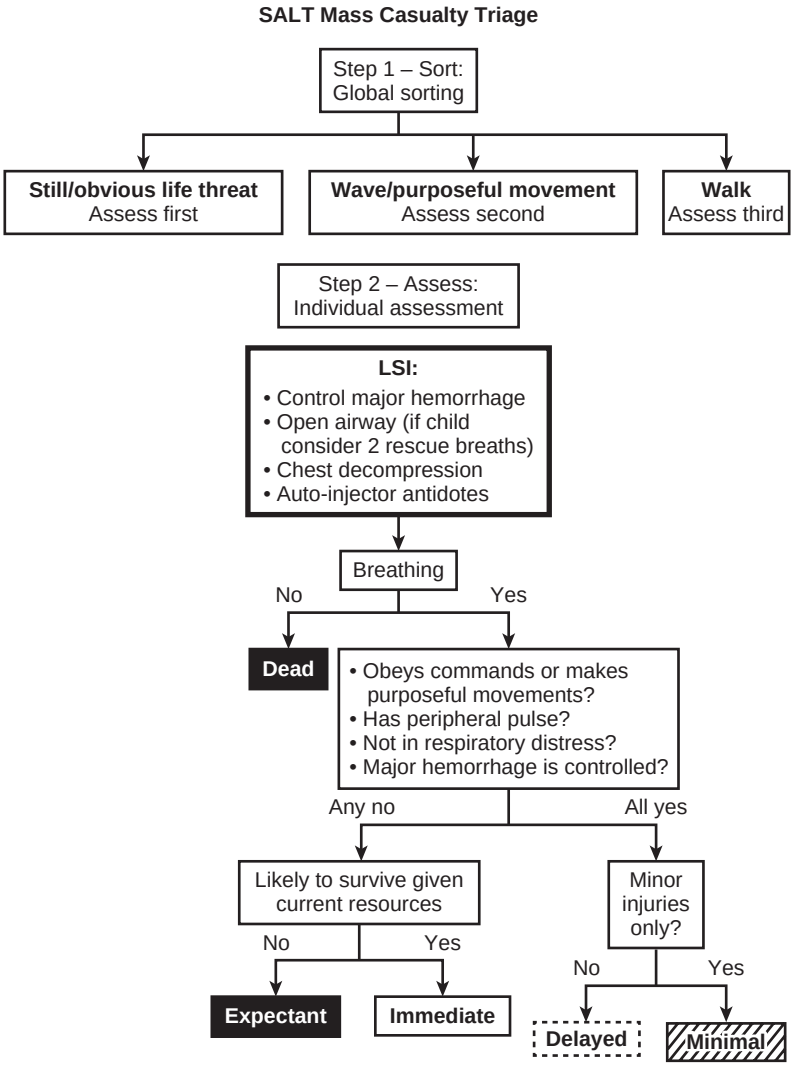

Figure 100-3. SALT (sort, assess, life-saving interventions, and treat/transport) triage algorithm. *LSI,* Life-saving intervention.

18. What is the difference between START and SALT triage systems?

Similar to START, SALT first performs a global sorting by assessing for the ability to ambulate. Those who can walk are assessed last. Next, if victims can follow a simple command (such as "Raise your hand if you can hear me"), they are considered less emergent and assessed second. Priority for assessment is given to those not responding, who are then triaged as either *red, black,* or *gray,* based on their injuries and response to simple life-saving interventions (such as a needle decompression for a tension pneumothorax). An important distinction between the two systems is that SALT triage incorporates a gray color designation for patients who are deemed *expectant,* meaning those who have little likelihood to survive their injuries, even with adequate resources. This also allows for palliative interventions for these patients, so they are not simply left to die (Fig. 100-3).

19. Which one is better, START or SALT?

Retrospective analysis has shown START to be a reliable and easy-to-learn triage system for first responders. Studies comparing the START and SALT triage systems have found START easier to learn and use in a real disaster scenario. Notably, both are prone to overtriaging patients (identifying victims as more sick than they really are), which can be detrimental to the overall system because it may divert limited resources to people who do not truly need them.

20. How does transport occur for victims in an MCI?

On scene, it is important to have open access to ingress and egress routes, so ambulances can quickly and easily transport patients. A coordinated transport plan is essential to ensure that the closest hospital to a disaster does not become overwhelmed and turn into the next disaster scene. It is important logistically to ascertain the capabilities (how many patients they can accommodate, whether they are a trauma center) of all local hospitals in order to distribute patients appropriately and prevent overwhelming any single facility whenever possible. Red category patients in need of immediate care should be given priority for transport and ideally be sent to a trauma center. Patients with minor injuries or nontraumatic complaints can be sent to nontrauma centers for initial stabilization or definitive care.

21. When do patients need to be transported to the hospital emergently (i.e., lights and sirens [L&S])?

The use of warning L&S predates modern EMS systems as a means of improving response to and return from an accident scene. When transporting a patient to a hospital with L&S, there is a well-recognized increased risk of emergency medical vehicle collisions, not only increasing morbidity to those in the ambulance but to innocent bystanders and general public. Studies have shown that L&S transport to a hospital offers minimal benefit even for time-critical interventions, offering on average less than 3 minutes faster arrival time over a nonemergent return, even for transports over longer distances. Additionally, patient outcome has not been clearly shown to benefit, bringing into question the utility of this warning system in most scenarios, particularly a large-scale disaster or MCI.

22. What about spinal immobilization for patients?

Routine spinal immobilization with a cervical collar and backboard for patients with trauma is falling out of favor, because there are not inconsequential risks of increased pain, respiratory compromise, and the development of pressure sores. Current practice guidelines advocate for the selective use of spinal immobilization, based not merely on mechanism of injury but on the presence of spinal pain and tenderness, symptoms of neurologic compromise (weakness or numbness), altered mentation (including intoxication), and significant distracting injuries. Whereas scoop stretchers and similar devices may facilitate extrication from the scene, particularly in a disaster where terrain may be unstable and difficult to safely navigate, the continued use of a backboard after a patient is on a gurney and in an ambulance may not be necessary, especially because spinal precautions can be maintained by simply securing the patient to the bed and minimizing movement. Practically speaking, in an MCI, it may be logistically challenging if not impossible to immobilize every patient on a backboard and with a cervical collar based on mechanism of injury alone. Although local established protocols should guide standard operational use of spinal immobilization, deviation from protocol may be necessary in the event of a disaster.

23. Can doctors and nurses be helpful on scene for an MCI?

Yes, if they have training and expertise in responding to MCIs and are knowledgeable of and experienced in prehospital medicine and the concept of triage.

24. What is critical incident stress management (CISM)?

CISM helps first responders deal with the emotional aftermath of a disaster. Stress is a normal response to a disaster, which can affect an entire community and perhaps an entire country (e.g., Boston marathon bombing of 2013, Aurora movie theater shooting of 2012, the terrorist attacks of September 11, 2001). It is not surprising that those on scene are often particularly affected psychologically, and perhaps physically, as well. Debriefing is an important part of CISM, because it allows all personnel the opportunity to speak openly about their experience in an effort to heal from the event. Debriefing also has the additional benefit of identifying pitfalls of local response and field triage in order to improve future disaster response.

KEY POINTS: DISASTER MANAGEMENT

1. Disasters and MCIs occur whenever the needs of an incident exceed the resources available to respond to it.
2. Communication is the most important component to ensuring an efficient and effective response.
3. An ICS can bring order to chaos for a disaster or MCI.

WEBSITE

For more information about the NIMS, including educational opportunities: www.fema.gov/nims; accessed 3-19-15.

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QUESTIONS

1. Which are the correctly paired triage designations and colors?
   a. Yellow = expectant, red = delayed, green = immediate, black = minor
   b. Red = immediate, yellow = delayed, green = minor, black = expectant
   c. Green = immediate, black = delayed, red = expectant, yellow = minor
   d. Black = expectant, red = delayed, yellow = minor, green = immediate
   The correct answer is *b*.
2. What is the definition of *disaster*?
   a. When the needs exceeds the resources available
   b. A natural event that damages significant property
   c. A federally defined event where national resources are required
   d. An incident leading to multiple victims and casualties
   The correct answer is *a*.
3. What are the components of an ICS?
   a. Commander, captain, sergeant, lieutenant
   b. Finances, medical, public relations, security, engineering
   c. Operations, logistics, finance, command, planning
   d. Response, supplies, finance, transportation, media relations
   The correct answer is *c*.

WEAPONS OF MASS DESTRUCTION

Aaron M. Eberhardt, MD, and Peter T. Pons, MD, FACEP

CHAPTER 101

1. **Why is it important for emergency physicians (EPs) to be familiar with weapons of mass destruction (WMDs)?**
   EPs play an integral role in the planning, preparation, and response to not only unintentional and natural disasters, but also terrorist attacks. Casualties from these types of incidents will largely arrive at an ED, and EPs are increasingly being called 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. Are we ready to respond?**
   Much has been done to address the issue of terrorism prevention since the attacks of September 11, 2001. EPs must continue to be guided by the recognition that the threat of terrorism will never be fully eliminated and that the capability 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. **Do we have hazardous materials (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. NBC attacks will likely occur in population-dense areas that 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 biologic weapons attack does not lend itself to a traditional HAZMAT response at all, because 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 integration and coordination of multiple federal, state, and local agencies. The impact of traditional media, as well as social 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
   - Be highly lethal or toxic
   - Be easy to disperse over large areas
   - Be relatively stable in the environment so that it does not break down too fast
   - Be packaged in such a way that it can withstand the energy transfer that occurs during delivery
   - Be relatively easy to obtain and inexpensive to manufacture

6. **What should EPs do to prepare and protect themselves?**
   EPs 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 important, 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.

Abstract

This chapter reviews the basic principles of managing an incident involving a weapon of mass destruction (WMD). An overview of the chemical, biologic, radiologic, and explosive injuries is provided.

Keywords:

weapon of mass destruction (WMD), anthrax, nerve agents, terrorism, personal protective equipment (PPE), radiation exposure, radiologic dispersal device (RDD), dirty bomb, acute radiation syndrome, bioterrorism, blast injury, blast lung, decontamination

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 HAZMAT 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. It can be combined with an 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: This level of PPE provides minimal skin protection and is used when no respiratory protection is required.

RADIATION

8. What are the basic physics of radiation?

In general terms, radiation is energy that is propagated through space. 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 radioisotopes. If this ionization interacts with a biologically important molecule, such as DNA, the genome may not function appropriately. 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.

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: All or a portion of the body is exposed to penetrating γ radiation from an external source. Significant cellular damage can occur. After 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.
- Incorporation: This is 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.

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 J/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, γ radiation has a QF of 1. 1 Gy of pure γ radiation = 1 Sv 1 Sv = 100 rem

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, 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 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 lack of charge, 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.

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 potentially 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.
- Radiologic 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 results from the explosion, and 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 the environment.
- Nuclear bomb: Although obviously the most potentially devastating attack, this is the least likely method of attack because there are strict security measures for existing stockpiles, and it is difficult to obtain the money and technology needed to manufacture a new weapon.

13. Describe the three acute radiation syndromes (ARSs).

1. 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). It usually occurs after exposure to between 0.7 and 10 Gy (70 to 1000 rads).
2. Gastrointestinal (GI) syndrome: There is irreversible destruction of the GI lining, causing nausea, vomiting, and diarrhea. Survival is extremely unlikely, because death is caused by overwhelming sepsis and electrolyte disturbances. GI syndrome usually occurs after exposure to between 6 and 10 Gy (600 to 1000 rads).

3. 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 usually occurs with a dose greater than approximately 50 Gy (5000 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.

1. 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.
2. Latent period: Resolution of symptoms experienced in the initial stage with the patient appearing relatively well. This stage can last from several hours to approximately 2 weeks.
3. 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 exceeding 8 Gy (800 rads), the primary effects are on the lining of the intestines leading to diarrhea, fever, sepsis, and electrolyte disturbance.
4. Recovery or death: Survival is highly unlikely with doses exceeding 10 Gy (1000 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.

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% or greater of total body surface area being considered a major burn. Treatment of whole-body irradiation becomes more complicated. Numerous adjunctive medications may be useful, including potassium iodide (KI), diethylenetriaminepentaacetate (DTPA), Prussian blue, and filgrastim (Neupogen). Such patients should be considered to be immunocompromised and treated as such. There is an excellent overview of WMDs in general and these treatments in particular on the Centers for Disease Control and Prevention (CDC) website.

18. When should treatment with KI tablets be considered?

After a radiologic or nuclear event, it is possible that radioactive iodine may be released. It may be suspended in the air or contaminate food or water sources, which may be inhaled or ingested. The

Table 101-3. Role of the Absolute Lymphocyte Count

MINIMAL LYMPHOCYTE COUNT WITHIN 48 HOURS OF EXPOSURE	ESTIMATED ABSORBED DOSE (GY)	PROGNOSIS
1000-3000	0-0.5	Likely no injury
1000-1500	1-2	Significant but good prognosis
500-1000	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.

thyroid gland will then absorb this radioactive iodine, which can lead to irreversible destruction of the gland. KI tablets contain stable (nonradioactive) 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://emergency.cdc.gov/radiation/ki.asp.

19. What is the most appropriate course of action for a patient with radiologic exposure and associated major trauma?

Caring for life-threatening trauma always takes precedence over management of the radiologic exposure. The traumatic injuries are an immediate threat to the patient's survival, whereas the radiation exposure is more of a long-term consideration. Decontamination can and should be delayed in those patients requiring immediate surgical intervention until after the resuscitation and all necessary procedures have been accomplished.

CHEMICAL WEAPONS

20. List the characteristics of chemical weapons.

- Volatility is 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). 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 nonpersistent or persistent based on their ability to vaporize in less than or greater than 24 hours, respectively. Persistent chemicals 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 multiplied by the amount of time a patient is exposed. One can go further and look at the LCt_{50}, which is the Ct of a vapor or aerosol that will kill 50% of those exposed to the agent.
- Latency is 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 have been exposed and need to be decontaminated and treated.

21. What are the different classes of chemical weapons?

See Table 101-4.

22. Describe the pathophysiology and clinical symptoms caused by nerve agents.

The pathophysiology of nerve agents should be familiar to EPs, because the chemical effects are very similar to the organophosphate insecticides.

The nerve agents inhibit acetylcholinesterase (AChE) at the postsynaptic nerve receptors. This leads to excessive acetylcholine accumulation and 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.

23. What is the easiest way to remember the effects of nerve agents?

One of the most common ways to remember the toxidrome is by the *SLUDGE* mnemonic:

Salivation
Lacrimation
Urination
Defecation
GI symptoms
Emesis

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)
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) Chlorobenzylidene malononitrile (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)

From Centers for Disease Control and Prevention: *Emergency preparedness and response: chemical emergencies*. Available at www.bt.cdc.gov/chemical; accessed 10-2-14.
GI, Gastrointestinal.

The patient can have life-threatening bronchorrhea and bronchospasm. The CNS symptoms can include seizure, coma, or apnea. Two better mnemonics are *DUMBBELS* and *MTWHF* for the muscarinic and nicotinic effects respectively.

DUMBBELS
Diarrhea
Urination
Miosis/muscle weakness
Bronchorrhea and bradycardia
Emesis
Lacrimation
Salivation and sweating
MTWHF
Mydriasis
Tachycardia
Weakness
Hypertension and hyperglycemia
Fasciculations

24. How deadly are nerve agents?

The dose of VX that will kill half of exposed victims (LD_{50}) is 10 mg (skin exposure) on a 70-kg man. To give 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.

25. 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 (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 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.

Table 101-5. Treatment for Nerve Agent Toxicity

PATIENT (AGE IN YEARS)	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 g) IV slowly	Atropine: 6 mg IM 2-PAM: 15 mg/kg (1 g) IV slowly

From U.S. 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. Available at www.atsdr.cdc.gov/MHMI/mmg166.html; accessed 10-2-14.

2-PAM, Pralidoxime chloride; *IM,* intramuscularly; *IV,* intravenously.

Notes:

1. May repeat atropine (2 mg IM or 1 mg IM for infants) every 5 to 10 minutes until secretions dry and breathing becomes more comfortable.
2. Phentolamine can be used for 2-PAM–induced hypertension (5 mg IV for adults and 1 mg IV for children).
3. Diazepam can be used for seizures.

BIOLOGIC AGENTS

26. What is bioterrorism?

Bioterrorism is an attack characterized by the deliberate release of viruses, bacteria, or biologic toxins to cause illness or death in people, animals, or plants.

27. Have terrorists really used biologic agents?

Biologic weapons have been used in warfare since antiquity. In the fourteenth and fifteenth centuries, warring armies would hurl plague-infected corpses over the walls of cities they were attempting to conquer. Accounts exist of biologic weapons being used in both World War I and World War II. Many terrorist groups have used biologic weapons as well. In 1984, 750 people in Oregon became sick after eating at salad bars in four different restaurants that were 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.

28. Does the manufacturing of biologic agents require a lot of money and sophisticated equipment?

The manufacture and dispersal of a biologic agent is significantly easier to accomplish than a nuclear attack. The previously mentioned incidents with *Salmonella* and anthrax demonstrate how easily this can happen.

29. How are biologic attacks different from exposure to radiation or chemical agents?

A biologic attack creates two significant challenges that are different from a radioactive exposure or chemical attack. First, the development of symptoms is usually delayed. Second, symptoms may initially be very nonspecific and will likely be attributed to a less serious cause. These factors often lead to a significant delay in recognition, and therefore the ability to appropriately respond to a biologic attack. If the agent used is highly communicable, an epidemic could develop very quickly.

30. How does the CDC categorize biologic agents?

The CDC prioritizes biologic 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.

- Category A: 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: 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 the 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: 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

31. What are the general descriptive characteristics of biologic agents?

See Table 101-6.

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 dosage 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 World Health Organization: *Public health response to biological and chemical weapons: WHO guidance* (2004). Available at www.who.int/csr/delibepidemics/biochemguide/en/; accessed 10-2-14.

32. 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 biologic attack, the aerosolized anthrax spores are inhaled, taken up by macrophages in the lung, and transported to mediastinal lymph nodes, where they transform into the vegetative state. Once the bacteria start replicating, they produce toxins that cause edema (edema factor) and hemorrhage and necrosis (lethal factor).

33. 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, flulike syndrome with fever, nausea, vomiting, muscle aches, and fatigue. This can quickly progress to difficulty breathing, respiratory failure, shock, and death. Of 10 patients 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.

34. How should I treat anthrax?

CDC recommendations for inhalational anthrax include ciprofloxacin and doxycycline, plus one or two additional antibiotics if treating meningitis. For cutaneous anthrax, ciprofloxacin and doxycycline also are first-line therapy. Treatment should be continued for 7-14 days. A recent addition to the Strategic National Stockpile is raxibacumab, which is a monoclonal antibody active against the third factor (protective 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 medication.

35. What other sources are available to learn more about biologic weapons?

One of the textbooks of military medicine is entitled *Medical Aspects of Biological Warfare.* This text is updated regularly and is a great source for more information about biologic weapons. This text can be accessed at www.cs.amedd.army,mil/borden/.

36. How should I protect myself when I am caring for patients exposed to biologic weapons?

Universal precautions should be observed at all times when dealing with a biologic event. Table 101-7 lists person-to-person transmissibility and isolation requirements by biologic 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 used.

37. How will I know if a biologic attack has occurred?

Recognizing a biologic 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

Table 101-7. Transmissibility and Isolation Requirements for Biologic 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

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

38. What should I do if I suspect an attack has occurred?

All emergency providers should familiarize themselves with their hospitals' internal disaster plans and reporting processes. Early involvement of local and state public health is important for both diagnostic and epidemiologic follow-up study. 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

39. With all these other highly effective and lethal terrorist weapons, are people really still using explosives?

Absolutely; the widespread use of NBC weapons is significantly limited, because 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 2013, worldwide, more than 22,000 people were killed in terrorist attacks by bombing or combined bombing/arson. At the Boston Marathon bombing in 2013, 3 people were killed and more than 250 injured, with at least 14 requiring amputations. 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.

40. Describe the five blast injury categories after explosions.

1. 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.
2. 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.
3. 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.
4. 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.

5. Quinary: Injuries resulting from additives such as bacteria or radiation (dirty bombs), which may result in a hyperinflammatory response and syndrome.

41. 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.

42. What is blast lung?

Blast lung is 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

43. 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 from 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.

44. 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 patients that will inevitably arrive independently of EMS for evaluation and care. Hospital staff members involved in the decontamination process should use 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. Although a variety of neutralizing solutions, such as dilute bleach solution, have been suggested as decontamination solutions, most are not used in the hospital setting because they require prolonged contact time (15 to 20 minutes) and have the potential for causing additional skin injury.

45. How do I decontaminate a patient who has been exposed to radioactive material?

Once the patient is removed from the radiologically 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:

1. 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 toward mucous membranes. Care must also be taken to not abrade the skin, because radionuclides can be absorbed through skin abrasions. This process will successfully remove approximately 95% of the contamination.
2. 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.

46. How are victims of biologic agent exposure decontaminated?

In most cases, victims of a biologic attack will seek treatment 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: WMDS

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 time in a relatively small geographic area.
4. In the event of a biologic 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, an RDD that involves using a conventional explosive to disperse some radioactive material.

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QUESTIONS

1. Which of the following WMDs is a terrorist most likely to use to create casualties?
   a. Biologic
   b. Chemical
   c. Explosive
   d. Radiologic
   The correct answer is *c*. Explosives remain the cheapest and easiest weapon for a terrorist to obtain and use.
2. After an explosion at a mass gathering, a number of people complain of burning sensation of their eyes, nose, and pharynx. Which class of agents is the most likely source of these symptoms?
   a. Asphyxiants
   b. Nerve agents
   c. Pulmonary toxicants
   d. Vesicants
   The correct answer is *c*. The pulmonary agents, such as chlorine and ammonia, are easily available and present with these symptoms.
3. What is the most appropriate treatment for a patient complaining of sudden onset of blurred vision, tearing, rhinorrhea, and trouble breathing?
   a. Atropine and 2-PAM
   b. Botulinum antitoxin
   c. British anti-lewisite (BAL)
   d. Hydroxocobalamin
   The correct answer is *a*. This symptom complex strongly suggests the likelihood of nerve agent exposure, for which the treatment is atropine and 2-PAM.

CHAPTER 102

TACTICAL MEDICINE

Paul R. Hinchey, MD, MBA, and Andrew Harrell IV, MD

1. What is tactical medicine?

Tactical emergency medical support (TEMS), is the integration of close medical support for law enforcement operations and law enforcement officer activities. TEMS is a growing area of emergency medical services (EMS) and emergency medical practice.

2. What has driven the development of TEMS as an area of practice?

The increasing numbers of active shooter/mass casualty incidents (AS/MCIs) has helped the concept of TEMS grow. The recognition of the need for more robust, forward-located civilian medical response has grown from the body of knowledge gained from military science and medical practice that close, point of wounding (POW) care increases the likelihood of survival for victims of trauma injuries.

3. Provide some specific examples of these incidents.

Events such as the Columbine shooting (1999) and the Boston bombing (2013) show that a forward-leaning response posture for EMS, encompassing TEMS principles, helps reduce morbidity and mortality by using practices and interventions that help treat the most likely injuries in AS/MCI events. The recognition that hemorrhage control and basic airway management principles coupled with early and aggressive treatment of tension pneumothorax, including needle decompression by providers not specializing in advanced life support (ALS) can improve outcomes.

The International Association of Fire Fighters (IAFF), Department of Homeland Security, American College of Surgeons, and Hartford Consensus all have language that incorporates the tenets of TEMS into civilian responses for AS/MCI events.

4. How does practice of this subspecialty fit within emergency medicine?

TEMS care and operational oversight by emergency medicine physicians is a new and growing area of EMS subspecialty medicine within the American Board of Emergency Medicine (ABEM) structure. TEMS is an increasingly appreciated and developing area of focus for fire, EMS, and law enforcement agencies. The increasing sophistication and expanding scope of practice of TEMS providers increases the need for involved emergency medicine and EMS-trained physicians to obtain appropriate levels of medical and operational knowledge based on sound science and research.

5. What are the main entities or recognized bodies providing recommendations on the practice and scope of TEMS medicine?

The Committee on Tactical Combat Casualty Care (CoTCCC) and Committee on Tactical Emergency Casualty Care (C-TECC) are the two main bodies that provide direction for skills and training for TEMS. The lessons learned from the recent years of U.S. military operations worldwide have singularly influenced this area of civilian emergency medicine and EMS practice.

6. What does *SWAT* stand for?

SWAT is an acronym for *special weapons and tactics.*

7. What is the role of SWAT, and why does it create a need for tactical medicine?

SWAT officers are called upon to operate in settings or conditions that are too risky for regular patrol law enforcement personnel. These high-risk conditions and situations create greater risk for injury to themselves and to suspects. Given the increased risk, it is critical to have more immediate access to care. SWAT teams are also commonly called upon to operate in remote areas and on extended deployment, creating a need for careful monitoring and maintenance of team health, readiness, and overall well-being. In the traditional model of prehospital care, providers would typically stage and wait at a safe distance from an event and rely on law enforcement to either bring the injured to care or declare the scene safe for EMS providers to enter. Tactical medicine programs provide special training to the medical care providers on police tactics and care in austere environments that allow them to safely deliver care in these environments.

Abstract

This chapter describes tactical emergency medical support (TEMS) and how it differs from standard emergency medical services (EMS) response.

Keywords:

emergency medical services (EMS), tactical emergency medical support (TEMS), cover versus concealment, care under fire, tactical field care, evacuation care, remote assessment, tourniquet, junctional wounds, QuickClot combat gauze, Celox (chitosan), HemeCon, NuStat, nasopharyngeal airway (NPA), needle decompression, tension pneumothorax

8. In what areas of tactical medicine can emergency physicians have input and participation?

Emergency physicians can participate in TEMS activities from the simple and generally well-understood conventional "medical director" functions for EMS providers supporting law enforcement operations through oversight, protocol development, training, and online medical control functions to much more, hands-on work as TEMS providers working along side law enforcement officers on specialty teams.

9. What are the goals of TEMS?

- Enhance mission completion.
- Assess medical threat.
- Monitor environmental effects.
- Reduce death, injury, and illness among team members, innocent bystanders, or victims and suspects.
- Reduce line of injury, death, and disability.
- Reduce lost work time.
- Provide preventive medicine and maintain team health.
- Coordinate with receiving facilities.
- Decrease liability.
- Preserve forensic evidence and crime scene.

10. What is the difference between cover and concealment?

Concealment prevents direct visualization but does not provide any protection from gunfire or projectiles (e.g., vegetation, wooden doors). Cover provides protection from hostile fire (e.g., concrete walls, engine block of vehicle) The effectiveness of cover is dependent upon the nature of the weapon being used and its ability to penetrate objects.

11. What are the zones of tactical operations, and how are they defined?

- Hot zone: This area is considered subject to direct hostile action.
- Warm zone: There is a potential for exposure hostility and to injury, but this threat is not immediate.
- Cold zone: This zone is outside the area of potential hostility and therefore poses no threat of injury.

12. What is "barricaded care," or remote patient care?

When hostages or casualties cannot be reached by the provider because of exposure to fire, or suspects have barricaded themselves, a tactical provider may still be able to perform an assessment remotely and direct care. Tactical providers should be trained in remote assessment using direct visual assessment techniques (perhaps binoculars) or available communication. Once an assessment has been performed, tactical providers can give direction to casualties or non–health care providers to begin to render basic care to themselves or others.

13. What comprises the tactical/military primary assessment, and how does it differ from the traditional primary assessment?

The tactical primary assessment places greater emphasis on early and aggressive hemorrhage control before initiating assessment of airway, breathing, and circulation (ABCs). This is reflected in the following mnemonic:

XABCDE
E**X**sanguinating hemorrhage
Airway
Breathing
Circulation
Disability
Expose

The military uses a similar model, *MARCH:*
Massive hemorrhage
Airway
Respiratory
Circulation
Head injury

14. What are the priorities in providing care in a tactical environment?

- Security: Tactical providers and/or the officers who accompany them must neutralize or contain any threat to ensure the security of their area or risk becoming casualties themselves.
- Immediate action plan: A well-formed plan takes into account ongoing security needs, as well as rapid assessment of the number of patients, and determines an appropriate location for a casualty collection point.
- Medical assessment and management: Assessment can begin once security is reasonably ensured. It may occur simultaneously with initiation of the immediate action plan.

15. What are the phases of tactical medical care, how are they defined, and what care is provided in each phase?

- Care under fire: The care provided in the hot zone while the victim and provider are still under hostile fire. Care is typically limited to moving the casualty to cover, immediate control of exsanguinating hemorrhage, and evacuation, if possible.
- Tactical field care: Care provided in the warm zone where there is no exposure to direct hostile fire, but the threat still potentially exists. Care in this phase addresses immediate life threats. This is the first opportunity to safely address airway management and breathing issues, such as sucking chest wound or tension pneumothorax. Any major hemorrhage not previously controlled is addressed, including the use of additional tourniquets, hemostatic agents, or pressure dressings. The extent of the management of these conditions depends on the security of the environment, the equipment available, and the timeline to evacuation. If time allows, care may include establishing intravenous (IV) access and volume replacement, providing analgesia, and use of prophylactic antibiotics, where necessary.
- Evacuation care: Care provided in the cold zone where there is no threat of hostility. In a civilian environment, this phase of care is typically provided while en route to definitive care at a level 1 or 2 trauma center. The extent of this care depends on the distance to and availability of definitive care. Care in this phase is similar to routine civilian trauma care.

16. How does hemorrhage control in the tactical or combat environment differ from that of civilian care?

The traditional approach to hemorrhage control begins with application of direct pressure and the use of tourniquets as a last resort. Combat and tactical environments also use direct pressure for compressible wounds but emphasize early use of a tourniquet for life-threatening hemorrhage from extremity wounds. The tactical provider also makes use of hemostatic agents and wound packing seldom used in traditional prehospital or hospital care environments. A growing body of literature supporting these practices is likely to lead to increased use in routine prehospital and ED care.

17. What are desirable features of a tactical/combat tourniquet?

Tourniquets should be constructed of a wide strap (at least 1.5 inches wide) to reduce soft-tissue injury, be self-applicable by the injured using one hand, and use a windlass or ratcheting mechanism to generate adequate pressure for hemorrhage control from large vessels.

18. How can I tell when a tourniquet is appropriately applied?

It should be tight enough to stop hemorrhage and create an extremity that is pale and pulseless.

19. In the tactical environment, what are the advantage(s) and limitations of using a tourniquet over other hemorrhage control techniques?

Most commercial tourniquets facilitate self-application by the wounded, providing immediate hemorrhage control without the need to expose others to hostile fire. Tourniquets allow control of exsanguinating hemorrhage without the need for continuous attention demanded by pressure dressings and direct pressure techniques. They can be applied quickly, allowing providers to address other clinical interventions or patient movement. Obviously, effective use of tourniquets is limited to injuries to the extremities. Junctional injuries or injuries to the torso require other hemorrhage control techniques.

20. What is the maximum time a tourniquet can be left in place without injury or loss of the limb?

The exact duration of tourniquet use with successful limb salvage is unknown. Tourniquets are commonly applied for up to 2 hours during surgery without limb sequelae. In general, the longer a tourniquet is in place, the greater the probability of permanent injury or loss of limb.

21. Where should a tourniquet be placed?

The recommendations for tourniquet application vary. Some authors recommend placement on the proximal extremity. This "high and tight" placement ensures a single compressible vessel, typically close to bone, and facilitates hemorrhage control to the entire extremity. Others advocate for placing the tourniquet just proximal to the wound. There is no published literature describing a clear benefit to either placement. Regardless of preference, a tourniquet should be tightened until the limb is pale and pulseless, and never applied over a joint.

22. What are the side effects of the failure to adequately tighten a tourniquet?

If a tourniquet is not tightened appropriately there is a risk of occluding venous return without cutting off arterial blood flow, contributing to compartment syndrome and necessitating fasciotomy, and risk of permanent sequelae.

23. What are the features of recommended hemostatic agents for use in the tactical environment?

A number of hemostatic agents have been developed and refined, based on the experience of military and tactical providers. Early efforts at hemostatic agents included the use of powdered or granular agents. These were impractical in a tactical environment, caused an exothermic reaction resulting in significant burns, complicated wound care, and were occasionally associated with embolization. Newer recommended hemostatic agents do not cause exothermic reactions and are impregnated into gauze. These allow effective use in a wider variety of environments and facilitate packing of deep wounds.

24. What areas of the body are amenable to wound packing in a tactical environment?

These are primarily the junctional areas, including:

- Buttock
- Pelvis/pelvic girdle
- Axilla
- Extremities
- Neck

25. What are the different types of hemostatic agents currently used in tactical care?

- QuickClot combat gauze: Uses a nonexothermic mineral compound (kaolin) impregnated in gauze that triggers the intrinsic clotting cascade. Large clots should be quickly evacuated, the source of bleeding identified, and the gauze packed into the wound in direct contact with the bleeding source. To maximize effectiveness, the gauze should be applied directly onto the hemorrhaging vessel(s). Combat gauze should be held in place for a minimum of 3 minutes to ensure full effect. Unlike plain gauze, saturated combat gauze should be removed and replaced by new gauze with fresh agent.
- Celox: A polysaccharide (chitosan) derived from shellfish. When it comes in contact with blood, it forms a gel-like substance that aids in clot formation. It was found to be somewhat less effective than combat gauze but has the added benefit of working independently of the clotting cascade. This makes it effective in patients with coagulopathies associated with anticoagulants and hypothermia.
- HemeCon: Also a chitosan derivative and functions in a manner similar to Celox. Like other agents, it is available in a gauze form that facilitates wound packing.
- NuStat: A blend of cellulose and silica-based fibers that are woven into a flexible dressing that enhances platelet activation and blood clotting.

26. What is the basic airway adjunct of choice in the tactical environment?

The nasopharyngeal airway (NPA), when used in conjunction with manual airway maneuvers and patient positioning, addresses the majority of airway concerns in the tactical environment.

27. What is the advanced airway intervention of choice in the tactical or combat environment?

Surgical cricothyrotomy uses a minimum of equipment, can be performed quickly, and provides a definitive airway. It does not require the use of a laryngoscope, suction, or paralytics, and direct placement limits the need for adjuncts to ensure and maintain proper placement.

28. What is the intervention of choice for managing tension pneumothorax in the field?

Use needle decompression. Large-gauge, over-the-needle catheters, such as 10-, 12-, or 14-gauge, should be used to help prevent obstruction and/or kinking. To ensure that the pleural space is reached, longer catheters should be used. A 3.25-inch catheter reached the pleura in 99% of those studied in a military cohort. Placement site includes the traditional second intercostal space in the midclavicular line. However, ballistic vests or body mass may necessitate a lateral approach using the fourth intercostal space in the mid or anterior axillary line.

29. Why is it important to manage a sucking chest wound, and what is the device of choice?

Penetrating trauma to the thorax interferes with normal pulmonary mechanics. Large wounds that are greater than two thirds of the diameter of the trachea can result in air preferentially being drawn into and out of the chest through the defect in the chest wall. These injuries should be managed with an occlusive dressing to help restore pulmonary mechanics. The use of commercial devices that incorporate a valve mechanism or taping three sides of an occlusive material have been described as a means of reducing the risk of tension pneumothorax. There is no published human evidence about the efficacy of these techniques and devices, although several animal studies have been recently published.

30. Why is it essential to manage hypothermia in the tactical environment?

Even mild hypothermia can contribute to coagulopathies in the trauma victim, and the presence of hypothermia has been independently associated with increased mortality. Hypothermia is common in both combat and civilian trauma victims, with as many as two thirds of civilian trauma victims arriving at the hospital with hypothermia. In the tactical environment, treating hypothermia once it has developed is far more difficult than taking steps to prevent it before it occurs. As a result, tactical and combat training programs have placed greater emphasis on initiating hypothermia management as soon as possible after wounding.

31. What is the leading cause of combat death?

Nearly 50% of combat deaths are the result of exsanguinating hemorrhage. Almost 20% of these occur in areas that can be easily controlled, whereas the remainder are large vessel injuries of the thorax. Statistics such as these have lead to the increased emphasis on training and use of tourniquets in the military and civilian tactical environment.

32. What are the most common areas of the body injured during combat?

- Head and neck: 4% to 24%
- Thoracic injuries: 4% to 15%
- Abdominal injuries: 2% to 20%
- Extremity injuries: 50% to 75%

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QUESTIONS

1. What is the largest cause of preventable death in casualties in the TEMS environment?
   a. Extremity hemorrhage amenable to tourniquet application
   b. Penetrating chest or abdominal injuries in need of surgical intervention
   c. Airway obstruction requiring advanced interventions
   d. Blast injuries from improvised explosive device (IED) detonations
   The correct answer is *a*.
2. All of the following are goals of TEMS, except:
   a. Enhance law enforcement mission completion and success.
   b. Reduce incidence of injury and illness among tactical team members, bystanders, and suspects.
   c. Provide for preventive health and maintain readiness for law enforcement teams.
   d. Increase the liability exposure for law enforcement tactical operations.
   The correct answer is *d*.
3. The first step of primary assessment in the tactical environment is:
   a. Airway
   b. Breathing
   c. Circulation
   d. Exsanguinating hemorrhage
   The correct answer is *d*.