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*Tintinalli's*  
**EMERGENCY  
MEDICINE  
MANUAL** 7th Edition



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American College of  
Emergency Physicians®

ADVANCING EMERGENCY CARE 

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# Tintinalli's Emergency Medicine Manual

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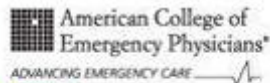
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## Preface

Following the lead of the 7th edition of *Emergency Medicine: A Comprehensive Study Guide*, the 7th edition of *The Manual of Emergency Medicine* has been upgraded more than any prior edition of the book. The reader will find more color images and radiographs including ultrasound and computed tomography scans to aid in diagnosis, more tables that summarize information, and more breadth in its coverage of the practice of emergency medicine than ever before. The *Manual*, and its predecessor the *Companion Handbook*, has been published in English, Spanish, French, Italian, Greek, Turkish, Polish, Portuguese, and Chinese, which reflects the growing number of emergency medicine practitioners worldwide. With each chapter we have tried to reflect the diversity of global disease, with our hope that additional new translations will be made available.

Despite these substantive changes, the original goal of the *Manual* is preserved in this new edition. This manual is written by and for active clinicians who are engaged in the day-to-day practice of emergency medicine. We hope that this handbook will assist practitioners of emergency medicine with the skillful and timely care of their patients in the emergency department. Each chapter remains succinct in its discussion of the Clinical Features, Diagnosis and Differential, and Emergency Department Care and Disposition of each disease entity. In this edition, we have increased coverage of pediatrics with new chapters on Hematologic-Oncologic Emergencies, and Renal Emergencies in children. Additionally we have new chapters on Low Probability Coronary Syndromes, Urinary Retention, Food and Waterborne Diseases, and World Travelers as well as increased discussion of toxicology and trauma. Color photographs and diagnostic images are embedded in their respective chapters for instant recognition of challenging and life threatening disorders.

We would like to express our sincere appreciation to the *Manual of Emergency Medicine* chapter authors for their commitment and work ethic in helping to produce this handbook. All authors are experienced clinicians; we thank them for taking time away from their busy practices to summarize

these topics. We also are indebted to numerous individuals who assisted us with this project; in particular, we would like to thank Anne Sydor, Jennifer Orlando, and Christina Thomas at McGraw-Hill Medical. Finally, without the love, support, and encouragement of our growing families, this book would not have been possible; DMC dedicates this book to Lisa, Jill, Oliva, Paul and Joseph; OJM dedicates this book to Elizabeth, Gabrielle, Natasha, Davis, and Sabrina; RKC dedicates this book to Marc, Matthew, Lissy, and Noah; GDM dedicates this book to Roo, Padre, and Steve; SHT dedicates this book to Caroline, Sarah, Alice, Cathrine; DAH dedicates this book to Nicole, Zachary, and Logan.

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# **SECTION 1 Resuscitative Problems and Techniques**

# CHAPTER 1

## Advanced Airway Support

Robert J. Vissers

Control of the airway is the single most important task for emergency resuscitation.

### INITIAL APPROACH

The initial approach to airway management is simultaneous assessment and management of the adequacy of airway patency (the A of the ABCs) and oxygenation and ventilation (the B of the ABCs).

1. Assess patient's color and respiratory rate; respiratory or cardiac arrest may be an indication for immediate intubation.
2. Open the airway with head tilt–chin lift maneuver (use jaw thrust if C-spine injury is suspected). If needed, bag the patient with the bag-valve-mask device that includes an O<sub>2</sub> reservoir. A good seal depends on proper mask size. This technique may require an oral or nasal airway or two rescuers (one to seal the mask with 2 hands and the other to bag the patient).
3. Provide continuous monitoring of vital signs, oxygen saturation, and end-tidal CO<sub>2</sub> (if possible).
4. Determine the need for invasive airway management techniques. Do not wait for arterial blood gas analyses if the initial assessment indicates the need for invasive airway management. If the patient does not require immediate airway or ventilation control, administer oxygen by facemask to ensure an O<sub>2</sub> saturation of 95%. Do not remove oxygen to draw an arterial blood gas analysis unless deemed safe from the initial assessment.
5. Preoxygenate all patients prior to intubation regardless of saturation. Assess airway difficulty before initiation of advanced airway techniques.

# OROTRACHEAL INTUBATION

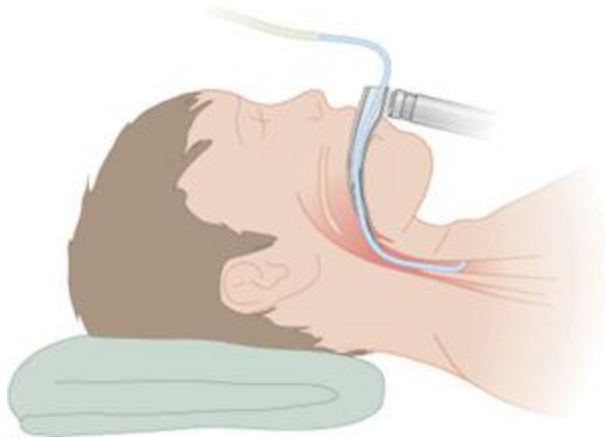
The most common means used to ensure a patent airway, prevent aspiration, and provide oxygenation and ventilation is orotracheal intubation. Rapid sequence intubation (RSI) should be used unless the patient's condition makes it unnecessary (ie, cardiac arrest) or it is contraindicated because of an anticipated difficult airway.

## Emergency Department Care and Disposition

1. Prepare equipment, personnel, and drugs before attempting intubation. Assess airway difficulty and anticipate required airway rescue. Assemble and place suction, bag-valve-mask and rescue devices within easy reach. Personnel should be present at the bedside to pass equipment or bag the patient, if required.
2. Ensure adequate ventilation and oxygenation and monitoring while preparing equipment. Preoxygenate with a non-rebreather oxygen mask at maximal oxygen flow rates or with a bag-valve-mask if the patient is not ventilating adequately.
3. Select blade type and size (usually a No. 3 or 4 curved blade or a No. 2 or 3 straight blade); test the blade light. Select the tube size (usually 7.5 to 8.0 mm in women, 8.0 to 8.5 mm in men) and test the balloon cuff. The use of a flexible stylet is recommended.
4. Position the patient with the head extended and neck flexed, possibly with a rolled towel under the occiput. If C-spine injury is suspected, maintain the head and neck in a neutral position with an assistant performing inline stabilization.
5. With the handle in the operator's left hand, insert the blade to push the tongue to the patient's left and slowly advance to the epiglottis. Suctioning may be required. It is not uncommon to go past the larynx into the esophagus. Gradually withdraw the blade to reveal the epiglottis. If the curved blade is used, slide the tip into the vallecula and lift (indirectly lifting the epiglottis); if a straight blade is used, lift the epiglottis directly.

Lift along the axis of the laryngoscope handle. Avoid levering the blade on the teeth to prevent dental trauma.

6. Once the vocal cords are visualized, gently pass the tube between the cords. Remove the tube, check for tube placement by ventilating and listening for bilateral breath sounds and absence of epigastric sounds. Inflate the cuff.
7. If the cords are not visualized, manipulate the thyroid cartilage using backward, upward, and rightward pressure (the “burp” maneuver) to help bring the cords into view. If unsuccessful, reoxygenation may need to be performed with bag-valve-mask device. Consider changing the blade, the tube size, or the position of the patient before further attempts. Consider using an intubating stylet (bougie, see [Fig. 1-1](#)). Three unsuccessful attempts define a failed airway, and other rescue techniques must be considered.



**FIGURE 1-1.** Depiction of a gum elastic bougie after insertion, before placement of the endotracheal tube. Procedure: The tip is angled to facilitate insertion when the glottis cannot be fully visualized. Insertion into the trachea can be appreciated by feeling the tip moving over the tracheal rings. The ETT is threaded over the introducer into the trachea, and the introducer is then removed.

8. Confirm placement objectively with an end-tidal CO<sub>2</sub> detector, capnography, or in cardiac arrest, an esophageal detection device. Check

tube length; the usual distance (marked on the tube) from corner of the mouth to 2 cm above the carina is 23 cm in men and 21 cm in women.

9. Secure the tube and verify placement with a portable radiograph.

Immediate complications include unrecognized esophageal intubation or mainstem bronchus intubation. Failure to confirm the position immediately can result in hypoxia and neurologic injury. Endobronchial intubation is usually on the right side and is corrected by withdrawing the tube 2 cm and listening for equal breath sounds.

## **RAPID SEQUENCE INTUBATION (RSI) INDUCTION**

RSI is the simultaneous administration of an induction and a neuromuscular blocking agent to facilitate orotracheal intubation. This technique couples sedation with muscular paralysis. Anticipated difficulty in mask ventilation or intubation is a relative contraindication to RSI.

The presence of 2 of the following 5 factors predict possible difficulty with bagging: facial hair, obesity, no teeth, advanced age, or snoring. Multiple external features, such as facial hair, obesity, short neck, short or long chin, and any airway deformity, suggest possible difficulty with intubation. A small oral opening, decreased neck mobility, a poor view of the posterior pharynx also suggest possible problems. If difficulty is anticipated, consider other methods of airway management such as videolaryngoscopy, awake intubation, cricothyrotomy, or an alternative airway device.

1. Prepare equipment, medication, and personnel before initiation of RSI. Check equipment function.
2. Preoxygenate the patient with 100% oxygen.
3. Consider pretreatment in patients with reactive airway disease or increased intracranial pressure. Evidence is mixed on whether pretreatment improves outcomes. **Fentanyl**, 3 micrograms/kilogram may be used in normotensive patients with possible raised intracranial



pressure, cardiac ischemia, or aortic dissection. Lidocaine, 1 milligram/kilogram IV, could be used in patients with possible raised ICP or asthma.

4. An induction agent should be pushed intravenously. **Etomidate**, 0.3 milligrams/kilogram, is an excellent choice in most circumstances. **Propofol**, 0.5 to 1.5 milligram/kilogram or **ketamine** 1 to 2 milligram/kilogram may also be considered. Use caution in hypotensive patients; avoid propofol. **Ketamine** is a good choice for patients with active bronchospasm, and may be the best choice in hypotensive patients.
5. Push a paralytic agent intravenously immediately after the induction dose. **Succinylcholine**, 1.0 to 1.5 milligram/kilogram, is preferred in most cases because of its rapid onset and short duration of action; it should not be used in a patient with a neuromuscular disorder, a denervation injury older than 7 days, or severe burns older than 5 days because hyperkalemia may occur. **Rocuronium**, 1 milligram/kilogram IV, a nondepolarizing agent, is a reasonable alternative.
6. Cricoid pressure may be applied at the discretion of the provider, however, it can be associated with a worsening laryngoscopic view and ability to bag-ventilate.
7. Once muscle relaxation occurs, usually after 35 to 60 sec, intubate the trachea and confirm tube placement using the techniques described above.
8. Be prepared to use bag-mask ventilation if intubation fails and saturations are less than 90%. Three unsuccessful attempts define a failed airway, and other rescue techniques must be considered.

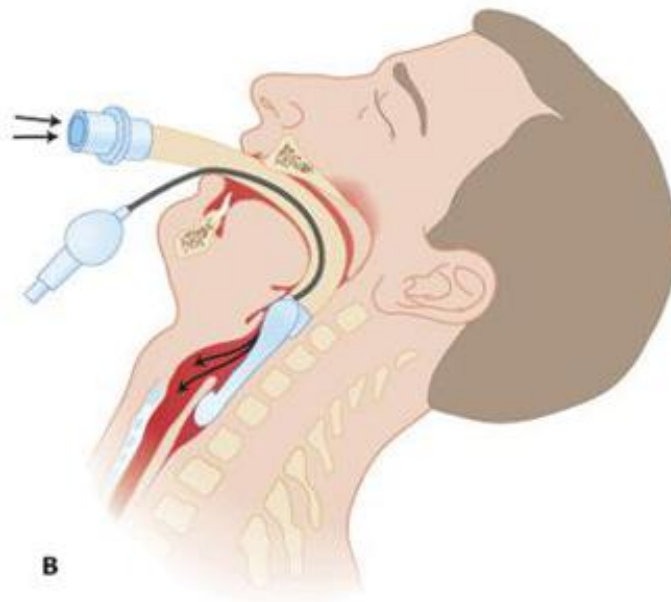
## **ALTERNATIVE AIRWAY DEVICES**

A number of rescue devices are available for management of the difficult airway. Intubating stylets (or gum elastic bougies) are semi-rigid stylets with a coude tip, which can be placed by feel, during laryngoscopy, into the trachea. The tracheal tube is then guided over the intubating stylet into the trachea. This device is useful for anterior cords that cannot be directly visualized.

The laryngeal mask airway (LMA) is an airway device that is placed blindly into the supraglottic space ([Fig. 1-2](#)). A distal ringed balloon is inflated which seals the glottis above the larynx and allows for ventilation. A disadvantage is that the airway is not protected from aspiration and leaks may occur at high ventilatory pressures. The Intubating-LMA allows for the placement of a cuffed endotracheal tube through the device.



**A**



**B**

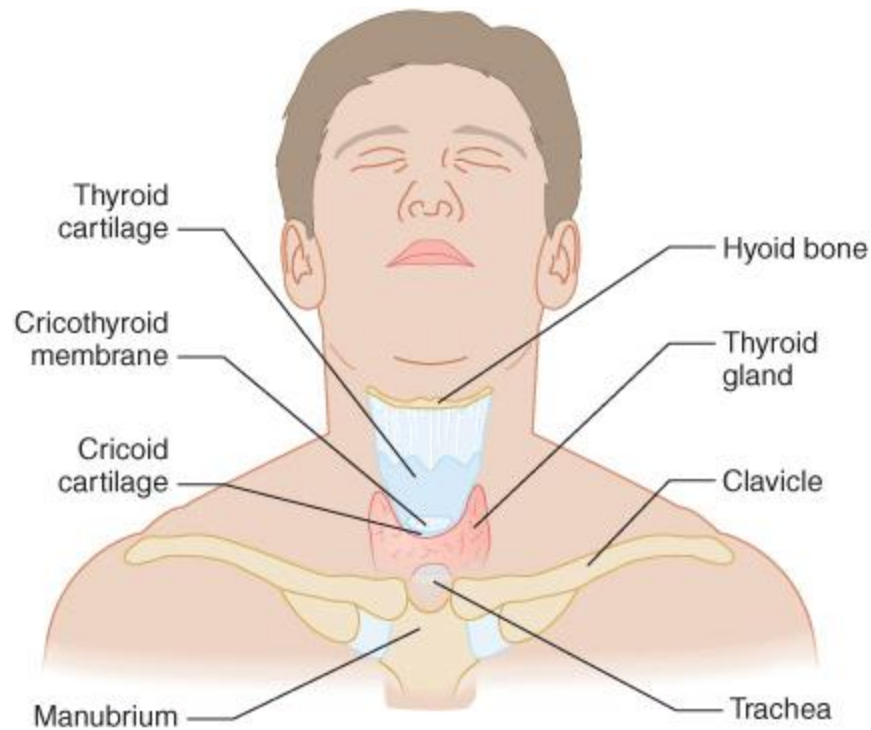
**FIGURE 1-2. A.** Laryngeal mask airway (LMA). **B.** LMA diagram showing placement at the larynx.

Videolaryngoscopy is an excellent option for airway rescue or as a primary intubation technique. Most studies demonstrate improved laryngoscopic views compared to traditional laryngoscopy. These devices can be advantageous in patients with restricted oral opening or cervical spine mobility.

## **Cricothyrotomy**

Cricothyrotomy is performed when intubation, ventilation, and airway rescue has failed. Cricothyrotomy is contraindicated in children younger than 10 to 12 years in whom trans tracheal jet ventilation is the preferred subglottic technique.

1. Use sterile technique. Palpate the cricothyroid membrane and stabilize the larynx ([Fig. 1-3](#)). With a No. 11 scalpel, make a vertical, 3 to 4 cm incision starting at the superior border of the thyroid cartilage. Incise caudally toward the suprasternal notch.



**FIGURE 1-3.** Anatomy of the neck.

2. Repalpate the membrane and make a 1 to 2 cm horizontal incision through the cricothyroid membrane. Keep the blade in place temporarily.
3. Stabilize the trachea by inserting the tracheal hook into the cricothyroid space and retracting the inferior edge of the thyroid cartilage (an assistant should hold the hook after it is placed).
4. Remove the scalpel and insert a dilator to enlarge the opening (LaBorde or Trousseau).
5. Introduce a No. 4 cuffed tracheostomy tube (or the largest tube that will fit). Alternatively, use a small cuffed endotracheal tube (No. 6 or the largest tube that will fit). Inflate the cuff.
6. Check for bilateral breath sounds. After confirmation of tube placement, remove the hook. Secure the tube. The presence of subcutaneous air suggests placement outside the trachea. Placement should be confirmed with an end-tidal CO<sub>2</sub> detector and radiograph.

Manufactured cricothyrotomy kits using a Seldinger technique are also available. Formal tracheostomy is not recommended as an emergency

surgical airway technique due to increased technical difficulty and increased time required.

## **NONINVASIVE POSITIVE PRESSURE VENTILATION**

Noninvasive positive pressure ventilation (NPPV) provides positive pressure airway support using preset volume/pressure of inspiratory air through a face or nasal mask. NPPV has been used as an alternative to endotracheal intubation in patients with ventilatory failure due to COPD, and cardiogenic pulmonary edema. Patients need to be cooperative and without cardiac ischemia, hypotension or dysrhythmia.

Continuous positive airway pressure (CPAP) provides constant positive pressure throughout the respiratory cycle. CPAP pressures are usually between 5 and 15 cm H<sub>2</sub>O and are adjusted to the patients' response to therapy.

Bilevel positive airway pressure (BiPAP) uses different levels of pressure during inspiration and expiration. Initial settings of 8 to 10 cm H<sub>2</sub>O during inspiration and 3 to 4 cm H<sub>2</sub>O during expiration are reasonable and can be titrated up based on clinical response.

Alternative drugs for rapid sequence induction are listed in [Chapter 30](#) of *Emergency Medicine: A Comprehensive Study Guide*, 7th ed. Airway management alternatives to the methods described earlier include blind nasotracheal intubation, digital intubation, transillumination, extraglottic devices, flexible and rigid fiberoptics, retrograde tracheal intubation, and translaryngeal ventilation. These techniques are described in [Chapters 28](#), [30](#), and [31](#) of *Emergency Medicine: A Comprehensive Study Guide*, 7th ed.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 28, "Noninvasive Airway Management," by A. Michael Roman; Chapter 30, "Tracheal Intubation and Mechanical Ventilation," by Robert J. Vissers and Daniel F. Danzl; and Chapter 31, "Surgical Airway Management," by Michael D. Smith.

# CHAPTER 2

## Arrhythmia Management

James K. Takayesu

### SINUS ARRHYTHMIA

Some variation in the sinoatrial (SA) node discharge rate is common; however, if the variation exceeds 0.12 second between the longest and shortest intervals, sinus arrhythmia is present. The electrocardiogram (ECG) characteristics of sinus arrhythmia are (a) normal sinus P waves and PR intervals, (b) 1:1 atrioventricular (AV) conduction, and (c) variation of at least 0.12 second between the shortest and longest P–P interval ([Fig. 2-1](#)). Sinus arrhythmias are affected primarily by respiration and are most commonly found in children and young adults, disappearing with advancing age. Occasional junctional escape beats may be present during very long P–P intervals. No treatment is required.

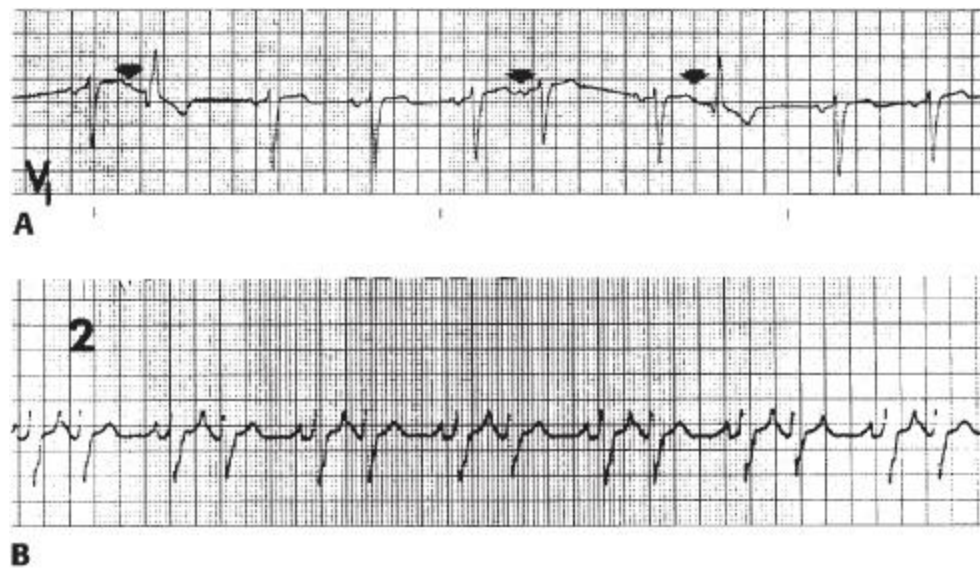


**FIGURE 2-1.** Sinus arrhythmia.

### PREMATURE ATRIAL CONTRACTIONS

Premature atrial contractions (PACs) have the following ECG characteristics: (a) the ectopic P wave appears sooner (premature) than the

next expected sinus beat; (b) the ectopic P wave has a different shape and direction; and (c) the ectopic P wave may or may not be conducted through the AV node (Fig. 2-2). Most PACs are conducted with typical QRS complexes, but some may be conducted aberrantly through the infranodal system, typically with a right bundle branch block pattern. When the PAC occurs during the absolute refractory period, it is not conducted. Since the sinus node is often depolarized and reset, the pause is less than fully compensatory. PACs are associated with stress, fatigue, alcohol use, tobacco, coffee, chronic obstructive pulmonary disease (COPD), digoxin toxicity, coronary artery disease, and may occur after adenosine-converted paroxysmal supraventricular tachycardia (PSVT). PACs are common in all ages, often in the absence of significant heart disease. Patients may complain of palpitations or an intermittent “sinking” or “fluttering” feeling in the chest.



**FIGURE 2-2.** Premature atrial contractions (PACs).

### **Emergency Department Care and Disposition**

1. Discontinue precipitating drugs (alcohol, tobacco, or coffee) or toxins.
2. Treat underlying disorders (stress or fatigue).

3. PACs that produce significant symptoms or initiate sustained tachycardias can be suppressed with agents such as  $\beta$ -adrenergic antagonists (eg, metoprolol 25 to 50 milligrams PO 3 times daily), usually in consultation with a follow-up physician.

## SUPRAVENTRICULAR BRADYARRYTHMIAS

### Sinus Bradycardia

#### *Clinical Features*

Sinus bradycardia occurs when the SA node rate becomes slower than 60 beats/min. The ECG characteristics of sinus bradycardia are (a) normal sinus P waves and PR intervals, (b) 1:1 AV conduction, and (c) atrial rate slower than 60 beats/min. Sinus bradycardia represents a suppression of the sinus node discharge rate, usually in response to 3 categories of stimuli: (a) physiologic (vagal tone), (b) pharmacologic (calcium channel blockers,  $\beta$ -blockers, or digoxin), and (c) pathologic (acute inferior myocardial infarction [MI], increased intracranial pressure, carotid sinus hypersensitivity, hypothyroidism, or sick sinus syndrome).

#### *Emergency Department Care and Disposition*

Sinus bradycardia usually does not require specific treatment unless the heart rate is slower than 50 beats/min and there is evidence of hypoperfusion.

1. **Transcutaneous cardiac pacing** is the only Class I treatment for unstable patients.
  - a. Attach the patient to the monitor leads of the external pacing device.
  - b. When placing transcutaneous pacing pads, place the anterior pad over the left lateral precordium and the posterior pad at the level of the heart in the right infrascapular area. Do not use multifunction pacing defibrillation pads unless the patient is unconscious as they cause a lot of discomfort.



- c. Slowly increase the pacing output from 0 mA to the lowest point where capture is observed, usually at 50 to 100 mA, but may be up to 200 mA. A widened QRS after each pacing spike denotes electrical capture.
  - d. If needed, administer a sedative, such as lorazepam, 1 to 2 milligrams IV, or an opiate, such as morphine, 2 to 4 milligrams IV, for pain control.
2. **Atropine** is a Class IIa treatment for symptomatic bradycardia. The dose is 0.5 milligram IV push, repeated every 3 to 5 min as needed up to a total of 3 milligrams IV. If given via endotracheal tube, increase the dose by 2 to 2.5 times over the IV dose. Slow administration or lower doses may cause paradoxical bradycardia. Atropine may not be effective in cardiac transplant patients since the heart is denervated and has no vagal stimulation
  3. **Epinephrine**, 2 to 10 micrograms/min IV, or **dopamine**, 3 to 10 micrograms/kilogram/min IV, may be used if external pacing is not available.
  4. Internal pacing will be required in the patient with symptomatic recurrent or persistent sinus bradycardia due to sick sinus syndrome.
  5. Isoproterenol, 2 to 10 micrograms/min IV infusion may be effective but carries a risk of increased myocardial oxygen demand and hypotension.

## SUPRVENTRICULAR TACHYARRHYTHMIAS

### Sinus Tachycardia

#### *Clinical Features*

The ECG characteristics of sinus tachycardia are (a) normal sinus P waves and PR intervals and (b) an atrial rate usually between 100 and 160 beats/min. Sinus tachycardia is in response to three categories of stimuli: (a) physiologic (pain or exertion), (b) pharmacologic (sympathomimetics, caffeine, or bronchodilators), or (c) pathologic (fever, hypoxia, anemia, hypovolemia, pulmonary embolism, or hyperthyroidism). In many of these

conditions, the increased heart rate is an effort to increase cardiac output to match increased circulatory needs.

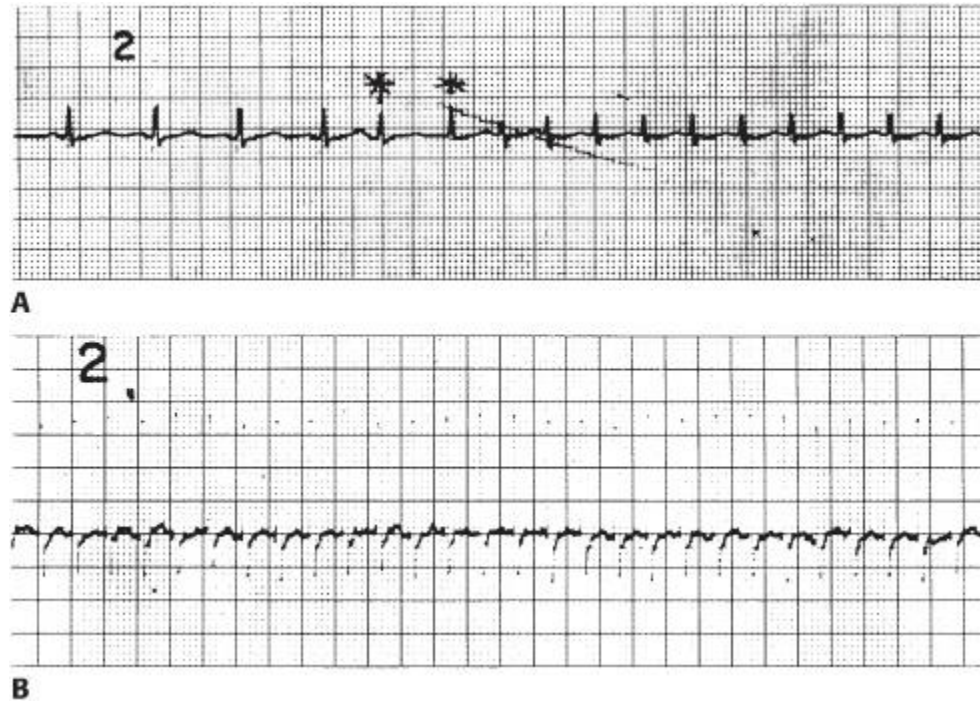
#### *Emergency Department Care and Disposition*

Diagnose and treat the underlying condition.

## **Supraventricular Tachycardia**

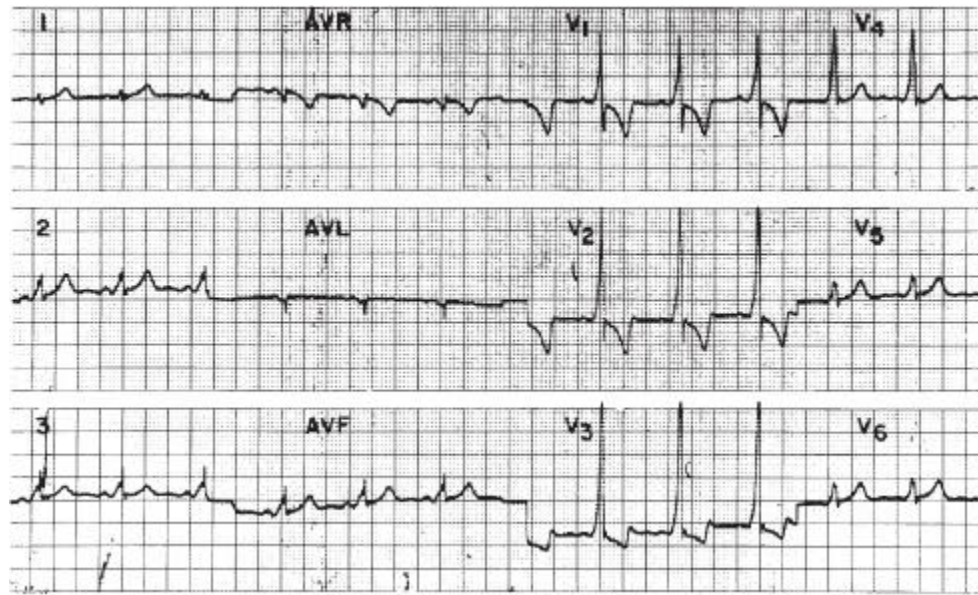
#### *Clinical Features*

Supraventricular tachycardia (SVT) is a regular, rapid rhythm that arises from impulse reentry or an ectopic pacemaker above the bifurcation of the His bundle. The reentrant variety is the most common ([Fig. 2-3](#)). Patients often present with acute, symptomatic episodes termed paroxysmal supraventricular tachycardia (PSVT). Atrioventricular nodal reentrant tachycardia (AVnRT) can occur in a normal heart or in association with rheumatic heart disease, acute pericarditis, MI, mitral valve prolapse, or preexcitation syndromes. In patients with atrioventricular bypass tracts (AVRT), reentry can occur in either direction, usually (80 to 90% of patients) in a direction that goes down the AV node and up the bypass tract producing a narrow QRS complex (orthodromic conduction). In the remaining 10% to 20% of patients, reentry occurs in the reverse direction (antidromic conduction). Ectopic SVT usually originates in the atria, with an atrial rate of 100 to 250 beats/min and may be seen in patients with acute MI, chronic lung disease, pneumonia, alcohol intoxication, or digoxin toxicity.



**FIGURE 2-3.** Reentrant supraventricular tachycardia (SVT). **A.** 2nd (\*) initiates run of PAT. **B.** SVT, rate 286.

There is a high incidence of tachyarrhythmias in patients with preexcitation syndromes including PSVT (40–80%), atrial fibrillation (10–20%), and atrial flutter (about 5%). All forms of preexcitation are caused by accessory tracts that bypass part or all of the normal conducting system, the most common form being Wolff-Parkinson-White (WPW) syndrome ([Fig. 2-4](#)). The ventricles are activated by an impulse from the atria sooner than would be expected if the impulse were transmitted down the normal conducting pathway. This premature activation causes initial fusion beat morphology with slurring of initial QRS complex, causing the pathognomonic delta wave. Among patients with WPW-PSVT, 80% to 90% will conduct in the orthodromic direction and the remaining 10% to 20% will conduct in the antidromic direction. ECG findings of atrial fibrillation or flutter with antidromic conduction down the bypass tract show a wide QRS complex that is irregular with a rate faster than 180 to 200 beats/min (see Atrial fibrillation).



**FIGURE 2-4.** Type A Wolff-Parkinson-White syndrome.

*Emergency Department Care and Disposition*

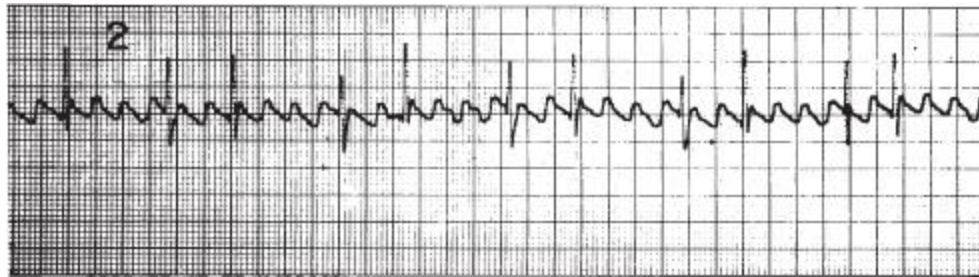
1. Perform synchronized cardioversion in any unstable patient (eg, hypotension, pulmonary edema, or severe chest pain).
2. In stable patients, the first intervention should be vagal maneuvers, including:
  - a. Valsalva maneuver: While in the supine position, ask the patient to strain for at least 10 seconds. The legs may be lifted to increase venous return and augment the reflex.
  - b. Diving reflex: Have the patient immerse the face in cold water or apply a bag of ice water to the face for 6 to 7 seconds. This maneuver is particularly effective in infants.
  - c. Carotid sinus massage: Auscultate to ensure that there is no carotid bruit and massage the carotid sinus against the transverse process of C6 for 10 seconds at a time, first on the side of the nondominant cerebral hemisphere. This should never be done simultaneously on both sides.

3. Administer **adenosine**, 6 milligrams rapid IV bolus, into a large vein followed by a 20 mL normal saline rapid flush. If there is no effect within 2 min, give a second dose of 12 milligrams IV. Most patients experience distressing chest pain, flushing, or anxiety lasting less than 1 min. Ten percent of patients may experience transient atrial fibrillation or flutter after conversion. This is first-line treatment for WPW-associated SVT with a narrow QRS complex (orthodromic conduction) but is ineffective in cases of anterograde conduction over an accessory pathway. Adenosine may induce bronchospasm in asthmatics requiring treatment with bronchodilators.
4. In patients with narrow-complex SVT (orthodromic conduction) and normal cardiac function, cardioversion may also be achieved with the following second-line agents:
  - a. Calcium-channel blockers. **Diltiazem**, 20 milligrams (0.25 milligram/ kilogram) IV over 2 min, or **verapamil**, 0.075 to 0.15 milligram/ kilogram (3 to 10 milligrams) IV over 15 to 60 seconds with a repeat dose in 30 min, if necessary. Verapamil may cause hypotension that can be prevented by pretreatment with calcium chloride or gluconate (500 to 1000 milligrams).
  - b. Beta-blockers. **Esmolol**, 500 micrograms/kilogram IV bolus, **metoprolol**, 5 milligrams IV, or **propranolol**, 0.1 milligram/ kilogram divided in three doses given 2 min apart.
  - c. **Digoxin**, 0.4 to 0.6 milligrams IV.
5. Patients with wide-complex SVT (antidromic conduction across accessory pathway) should be approached as presumed ventricular tachycardia (VT; see Ventricular Tachycardia) unless there is a known history of WPW syndrome. Patients with this type of tachycardia are at risk for rapid ventricular rates and degeneration into VF; therefore, agents that preferentially block the AV node such as  $\beta$ -blockers, calcium channel blockers, and digoxin should not be used. Treat stable patients with **procainamide**, 17 milligrams/kilogram IV over 30 min up to 50 milligrams/kilogram, or until 50% QRS widening is noted (contraindicated in patients with myasthenia gravis since it may increase weakness).

## **Atrial Flutter**

### *Clinical Features*

Atrial flutter is a rhythm that originates from a small area within the atria. ECG characteristics of atrial flutter are (a) a regular atrial rate between 250 and 350 beats/min; (b) “saw tooth” flutter waves directed superiorly and most visible in leads II, III, and aV<sub>F</sub>; and (c) AV block, usually 2:1, but occasionally greater or irregular ([Fig. 2-5](#)). One-to-one conduction may occur if a bypass tract is present. Carotid sinus massage or Valsalva maneuvers are useful techniques to slow the ventricular response by increasing the degree of AV block, which can unmask flutter waves in uncertain cases. Atrial flutter is seen most commonly in patients with ischemic heart disease as well as CHF, acute MI, pulmonary embolus, myocarditis, blunt chest trauma, and digoxin toxicity. Atrial flutter may be a transitional arrhythmia between sinus rhythm and atrial fibrillation. Consider anticoagulation in patients with an unclear time of onset or duration longer than 48 hours before conversion to sinus rhythm due to increased risk of atrial thrombus and embolization.



**FIGURE 2-5.** Atrial flutter.

### *Emergency Department Care*

The treatment is the same as atrial fibrillation and is discussed below.

## **Atrial Fibrillation**

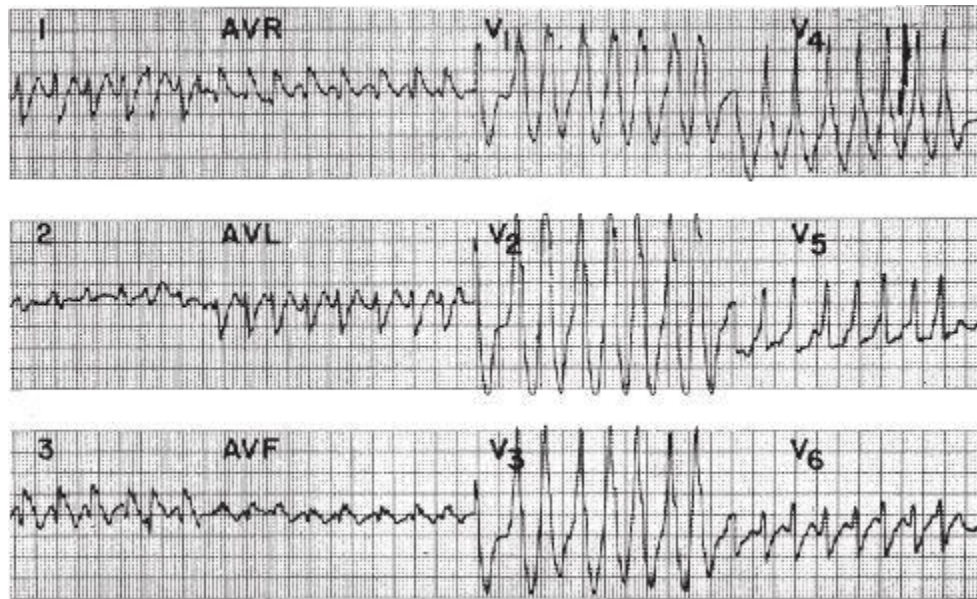
### *Clinical Features*

Atrial fibrillation (Afib) occurs when there are multiple, small areas of atrial myocardium continuously discharging in a disorganized fashion. This results in loss of effective atrial contraction and decreases left ventricular end-diastolic volume, which may precipitate CHF in patients with impaired cardiac function. The ECG characteristics of Afib are (a) fibrillatory waves of atrial activity, best seen in leads  $V_1$ ,  $V_2$ ,  $V_3$ , and  $aV_F$ ; and (b) an irregular ventricular response, usually between 170 to 180 beats/min in patients with a healthy AV node ([Fig. 2-6](#)).



**FIGURE 2-6.** Atrial fibrillation.

Afib may be paroxysmal (lasting for less than 7 days), persistent (lasting for more than 7 days), or chronic (continuous). Afib can be idiopathic (lone Afib) or may be found in association with longstanding hypertension, ischemic heart disease, rheumatic heart disease, alcohol use (“holiday heart”), COPD, and thyrotoxicosis. Patients with LV dysfunction who depend on atrial contraction may suffer acute CHF with Afib onset. Rates of greater than 300 beats/min with a wide QRS complex are concerning for a preexcitation syndrome such as WPW ([Fig 2-7](#)).



**FIGURE 2-7.** Atrial fibrillation in Wolfe-Parkinson-White syndrome.

Patients with Afib who are not anticoagulated have a yearly embolic event rate as high as 5% and a lifetime risk greater than 25%. Conversion from chronic Afib to sinus rhythm carries a 1% to 5% risk of arterial embolism; therefore, anticoagulation for 3 weeks is required before cardioversion in patients with Afib for longer than 48 hours duration and in those patients with an uncertain time of onset who are not on anticoagulation therapy.

*Emergency Department Care and Disposition*

1. Treat unstable patients with synchronized cardioversion (50-100 J).
2. Stable patients with Afib for longer than 48 hours should be anticoagulated with **heparin** (80 units/kilogram IV followed by an infusion of 18 units/kilogram/h IV) before cardioversion. Consider a transesophageal echocardiogram to rule out atrial thrombus before cardioversion.
3. Control rate with diltiazem. Administer 20 milligrams (0.25 milligram/kilogram) IV over 2 min followed by a continuous IV infusion, 5 to 15 milligrams/h, to maintain rate control. Give a second dose of 25



milligrams (0.35 milligram/kilogram) in 15 min if the first dose fails to control rate. Alternative rate control agents for patients with normal cardiac function include **verapamil**, 5 to 10 milligrams IV, **metoprolol**, 5 to 10 milligrams IV, and **digoxin**, 0.4 to 0.6 milligram IV. Treat patients with preexcitation syndromes (eg, WPW) with **procainamide**, 17 milligrams/ kilogram IV, over 30 min up to 50 milligrams/kilogram or until 50% QRS widening is noted. Avoid  $\beta$ -adrenergic or calcium channel blockers (ie, verapamil) due to the risk of causing degeneration to VF.

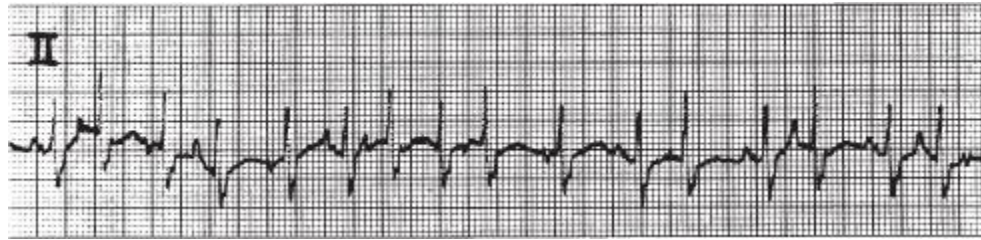
4. In patients with impaired cardiac function (EF <40%), use **amiodarone**, 5 milligrams/kilogram IV over 30 min, followed by 1200 milligrams over 24 hours (contraindicated in patients with iodine or shellfish allergy; increased risk of rhabdomyolysis if co-administered with simvastatin) or **digoxin** 0.4 to 0.6 milligram IV.
5. Patients with Afib for shorter than 48 hours may be chemically or electrically cardioverted in the emergency department. Use amiodarone, ibutilide (see comments for atrial flutter), procainamide, flecainide, or propafenone in patients with normal cardiac function. **Ibutilide** is dosed at 0.01 milligram/kilogram IV up to 1 milligram, infused over 10 min. A second ibutilide dose may be given if there is no response in 20 min. Ibutilide should not be administered to patients with known structural heart disease, hypokalemia, prolonged QTc intervals, hypomagnesemia, or CHF because of the possibility of provoking torsades de pointes. Monitor for 4 to 6 hours after giving ibutilide. Patients with impaired cardiac function may be cardioverted with amiodarone or electrically.

## **Multifocal Atrial Tachycardia**

### *Clinical Features*

Multifocal atrial tachycardia (MAT) is defined as at least 3 different sites of atrial ectopy. The ECG characteristics of MAT are (a) 3 or more differently shaped P waves; (b) changing PP, PR, and RR intervals; and (c) atrial rhythm usually between 100 and 180 beats/min ([Fig. 2-8](#)). Because the rhythm is irregularly irregular, MAT can be confused with atrial flutter or atrial fibrillation (AFib). MAT is found most often in elderly patients with

decompensated COPD, but it also may be found in patients with congestive heart failure (CHF), sepsis, methylxanthine toxicity, or digoxin toxicity.



**FIGURE 2-8.** Multifocal atrial tachycardia (MFAT).

#### *Emergency Department Care and Disposition*

1. Treat the underlying disorder.
2. Specific antiarrhythmic treatment is rarely indicated. Rate control may be achieved with **verapamil** 5 to 10 milligrams IV, or **diltiazem** 10 to 20 milligrams IV in patients with acute COPD or CHF exacerbations.
3. **Magnesium sulfate** 2 grams IV over 60 seconds followed by a constant infusion of 1 to 2 grams/h may decrease ectopy and convert MAT to sinus rhythm in some patients.
4. Replete potassium levels to greater than 4 mEq/L to increase myocardial membrane stability.

## **Junctional Rhythms**

#### *Clinical Features*

In patients with sinus bradycardia, SA node exit block or AV block, junctional escape beats may occur, usually at a rate between 40 and 60 beats/ min, depending on the level of the rescue pacemaker within the conduction system. Junctional escape beats may conduct retrogradely into the atria, but the QRS complex usually will mask any retrograde P wave ([Fig. 2-9](#)). When alternating rhythmically with the SA node, junctional

escape beats may cause bigeminal or trigeminal rhythms. Sustained junctional escape rhythms may be seen with CHF, myocarditis, acute MI (especially inferior MI), hyperkalemia, or digoxin toxicity (“regularized Afib”). If the ventricular rate is too slow, myocardial or cerebral ischemia may develop. In cases of enhanced junctional automaticity, junctional rhythms may be accelerated (60 to 100 beats/min) or tachycardic ( $\geq 100$  beats/min), thus overriding the SA node rate.



**FIGURE 2-9.** Junctional escape rhythm, rate 42.

#### *Emergency Department Care and Disposition*

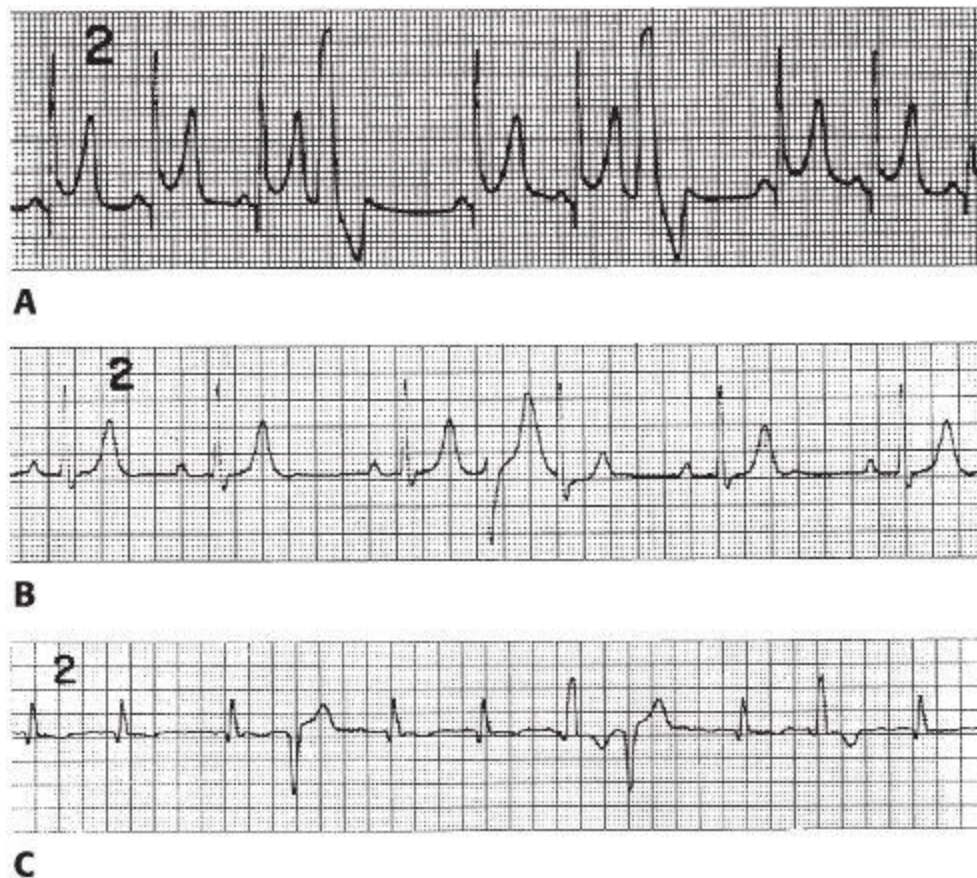
1. Isolated, infrequent junctional escape beats usually do not require specific treatment.
2. If sustained junctional escape rhythms are producing symptoms, treat the underlying cause.
3. In unstable patients, give **atropine** 0.5 milligram IV every 5 min to a total of 2 milligrams. This will accelerate the SA node discharge rate and enhance AV nodal conduction.
4. Use transcutaneous or transvenous pacing in unstable patients not responsive to atropine.
5. Manage patients with digoxin toxicity as discussed for SVT.

## **VENTRICULAR ARRHYTHMIAS**

### **Premature Ventricular Contractions**

#### *Clinical Features*

Premature ventricular contractions (PVCs) are due to impulses originating from single or multiple areas in the ventricles. The ECG characteristics of PVCs are: (a) a premature and wide QRS complex; (b) no preceding P wave; (c) the ST segment and T wave of the PVC are directed opposite the preceding major QRS deflection; (d) most PVCs do not affect the sinus node, so there is usually a fully compensatory post-ectopic pause, or the PVC may be interpolated between 2 sinus beats; (e) many PVCs have a fixed coupling interval (within 0.04 second) from the preceding sinus beat; and (f) many PVCs are conducted into the atria, thus producing a retrograde P wave (Fig. 2-10). If 3 or more PVCs occur in a row, patients are considered to have nonsustained ventricular tachycardia.



**FIGURE 2-10.** Premature ventricular contractions (PVCs). **A.** Unifocal PVC. **B.** Interpolated PVC. **C.** Multifocal PVCs.

PVCs are very common, occurring in most patients with ischemic heart disease and acute MI. Other common causes of PVCs include digoxin toxicity, CHF, hypokalemia, alkalosis, hypoxia, and sympathomimetic drugs. Pooled data and meta-analyses have found no reduction in mortality from suppressive or prophylactic treatment of PVCs. Ventricular parasystole occurs when the ectopic ventricular focus fires frequently enough to compete with the SA node and is associated with cardiac ischemia, electrolyte imbalance, and hypertensive or ischemic heart disease.

#### *Emergency Department Care and Disposition*

1. Stable patients require no treatment.
2. Patients with 3 or more PVCs occur in a row should be managed as VT.
3. For hemodynamically unstable patients with PVCs, consider **lidocaine** 1 to 1.5 milligrams/kilogram IV (up to 3 milligrams/kilogram) unless the patient is allergic to amide anesthetics.

## **Accelerated Idioventricular Rhythm**

#### *Clinical Features*

The ECG characteristics of accelerated idioventricular rhythm (AIVR) are (a) wide and regular QRS complexes; (b) a rate between 40 and 100 beats/min, often close to the preceding sinus rate; (c) mostly runs of short duration (3 to 30 beats/min); and (d) an AIVR often beginning with a fusion beat ([Fig. 2-11](#)). This condition is found most commonly with an acute MI or in the setting of reperfusion after successful thrombolysis.



## FIGURE 2-11. Accelerated idioventricular rhythms (AIVRs).

### *Emergency Department Care and Disposition*

Treatment is not necessary. On occasion, AIVR may be the only functioning pacemaker, and suppression with lidocaine can lead to cardiac asystole.

## Ventricular Tachycardia

### *Clinical Features*

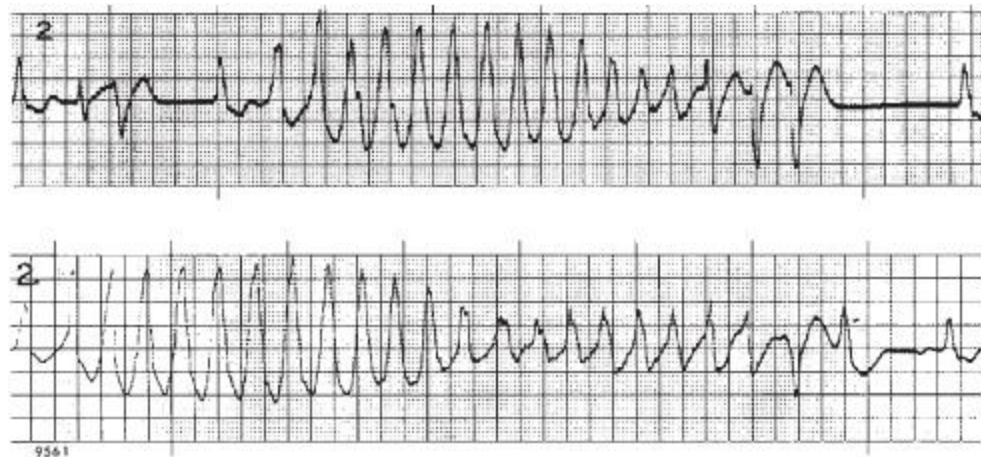
VT is the occurrence of 3 or more successive beats from a ventricular ectopic pacemaker at a rate faster than 100 beats/min. The ECG characteristics of VT are (a) a wide QRS complex, (b) a rate faster than 100 beats/min (most commonly 150 to 200 beats/min), (c) a regular rhythm, although there may be some initial beat-to-beat variation, and (d) a constant QRS axis ([Fig. 2-12](#)). The most common causes of VT are ischemic heart disease and acute MI. Because of this fact, patients presenting with VT should be considered candidates for urgent revascularization.



**FIGURE 2-12.** Ventricular tachycardia.

Other etiologies include hypertrophic cardiomyopathy, mitral valve prolapse, drug toxicity (digoxin, antiarrhythmics, or sympathomimetics), hypoxia, hypokalemia, and hyperkalemia. In general, all wide complex tachycardia should be treated as VT regardless of clinical symptoms or initial vital signs. Adenosine appears to cause little harm in patients with VT; therefore, stable patients with wide complex tachycardia due to

suspected SVT with aberrancy (see previous section) may be treated safely with adenosine when the diagnosis is in doubt. Atypical VT (torsade de pointes, or twisting of the points) occurs when the QRS axis swings from a positive to a negative direction in a single lead at a rate of 200 to 240 beats/min ([Fig. 2-13](#)). Drugs that further prolong repolarization—quinidine, disopyramide, procainamide, phenothiazines, and tricyclic antidepressants—exacerbate this arrhythmia.



**FIGURE 2-13.** Two examples of short runs of atypical ventricular tachycardia showing sinusoidal variation in amplitude and direction of the QRS complexes: “Le torsade de pointes” (twisting of the points). Note that the top example is initiated by a late-occurring PVC (lead II).

#### *Emergency Department Care and Disposition*

1. Defibrillate pulseless VT with unsynchronized cardioversion starting at 100J. Treat unstable patients who are not pulseless with synchronized cardioversion.
2. Treat hemodynamically stable patients with **amiodarone**, 150 milligrams IV over 10 min with repeated boluses every 10 min up to a total of 2 grams. Alternatively, an infusion of 0.5 milligram/min over 18 hours may be given after the initial bolus. Second-line agents include procainamide (in patients without suspected MI or LV dysfunction) and lidocaine.

3. For patients with torsades de pointes: Try overdrive pacing set at 90 to 120 beats/min to terminate torsades de pointes.
4. **Magnesium sulfate** 1 to 2 grams IV over 60 to 90 seconds followed by an infusion of 1 to 2 grams/h can be effective.
5. **Isoproterenol**, 2 to 10 micrograms/min IV infusion, is also used in refractory torsades but carries a risk of increased myocardial oxygen demand.

## **SVT with Aberrancy Versus Ventricular Tachyarrhythmias**

Patients with wide-complex tachycardia should be approached as having VT until proven otherwise. Age over 35 years, a history of MI, CHF, or coronary artery bypass grafting strongly favor VT. ECG signs favoring VT include AV dissociation, fusion beats, precordial lead QRS concordance, and a QRS duration longer than 0.14 second.

## **Ventricular Fibrillation**

### *Clinical Features*

VF is the totally disorganized depolarization and contraction of small areas of ventricular myocardium during which there is no effective ventricular pumping activity. The ECG shows a fine-to-coarse zigzag pattern without discernible P waves or QRS complexes ([Fig. 2-14](#)). VF is seen most commonly in patients with severe ischemic heart disease, with or without an acute MI. It also can be caused by digoxin or quinidine toxicity, hypothermia, chest trauma, hypokalemia, hyperkalemia, or mechanical stimulation (eg, catheter wire). Primary VF occurs suddenly, without preceding hemodynamic deterioration, and usually is due to acute ischemia or peri-infarct scar reentry. Secondary VF occurs after a prolonged period of hemodynamic deterioration due to left ventricular failure or circulatory shock.





**FIGURE 2-14.** Ventricular fibrillation.

*Emergency Department Care and Disposition*

1. Perform immediate electrical defibrillation (unsynchronized) at 200 J (biphasic) and 360 J (monophasic). If VF persists, do 5 cycles of CPR, check pulse, and defibrillate again if no pulse is present. Keep defibrillation pads on the patient and in the same location because, with successive countershocks, transthoracic impedance decreases.
2. If the initial 2 cycles of CPR and defibrillation are unsuccessful, administer antiarrhythmic treatment using **amiodarone** 300 milligrams IV push. **Lidocaine** is second-line and is dosed at 1.5 milligrams/kilogram IV followed by 0.75 milligram/kilogram IV for two more doses. Repeat the CPR-defibrillation cycle.
3. If no pulse is present after the third CPR-defibrillation cycle, give **epinephrine** 1 milligram IV push, or **vasopressin** 40 units IV push (1 time only), followed by a 20-mL normal saline flush and immediate resumption of the CPR-defibrillation cycle.
4. In refractory VF, administer **magnesium sulfate** 1 to 2 grams IV over 60 to 90 seconds followed by an infusion of 1 to 2 grams/h.

## **CONDUCTION DISTURBANCES**

### **Atrioventricular (AV) Block**

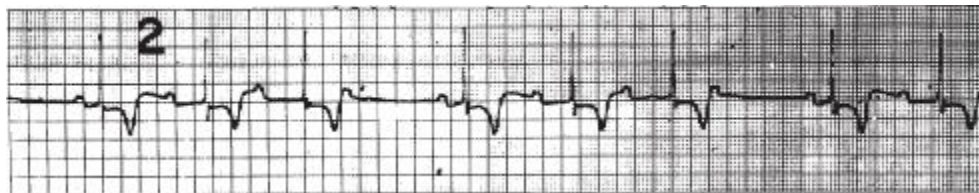
First-degree AV block is characterized by a delay in AV conduction, manifested by a prolonged PR interval ( $> 0.2$  second). It can be found in normal hearts and in association with increased vagal tone, digoxin toxicity,

inferior MI, amyloid, and myocarditis. First-degree AV block needs no treatment. Second-degree AV block is characterized by intermittent AV nodal conduction: some atrial impulses reach the ventricles, whereas others are blocked, thereby causing “grouped beating.” These blocks can be subdivided into nodal blocks which are typically reversible and infranodal blocks which are due to irreversible conduction system disease. Third-degree AV block is characterized by complete interruption in AV conduction with resulting AV dissociation.

## Second-Degree Mobitz I (Wenckebach) AV Block

### *Clinical Features*

Mobitz I AV block is a nodal block causing a progressive prolongation of conduction through the AV node until the atrial impulse is completely blocked. Usually, only one atrial impulse is blocked at a time. After the dropped beat, the AV conduction returns to normal and the cycle usually repeats itself with the same conduction ratio (fixed ratio) or a different conduction ratio (variable ratio). Although the PR intervals progressively lengthen before the dropped beat, the increments by which they lengthen *decrease* with successive beats causing a progressive *shortening* of each successive R–R interval before the dropped beat ([Fig. 2-15](#)). This block is often transient and usually associated with an acute inferior MI, digoxin toxicity, or myocarditis or can be seen after cardiac surgery. Because the blockade occurs at the level of the AV node itself rather than at the infranodal conducting system, this is usually a stable rhythm.



**FIGURE 2-15.** Second-degree Mobitz I (Wenckebach) AV block 4:3 AV conduction.

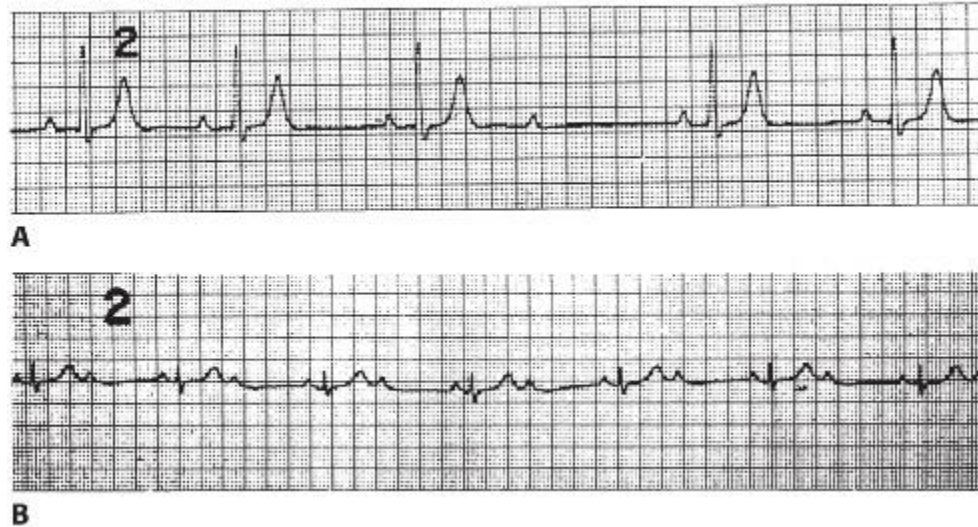
### *Emergency Department Care and Disposition*

1. Specific treatment is not necessary unless slow ventricular rates produce signs of hypoperfusion.
2. In cases associated with acute inferior MI, provide adequate volume resuscitation before initiating further interventions.
3. Administer **atropine** 0.5 milligram IV repeated every 5 min. Titrate to the desired heart rate or until the total dose reaches 2 milligrams.
4. Although rarely needed, transcutaneous pacing may be used.

## **Second-Degree Mobitz II AV Block**

### *Clinical Features*

Mobitz II AV block is typically due to infranodal disease, causing a constant PR interval with intermittent non-conducted atrial beats ([Fig. 2-16](#)). One or more beats may be non-conducted at a single time. This block indicates significant damage or dysfunction of the infranodal conduction system; therefore, the QRS complexes are usually wide coming from the low His-Purkinje bundle or the ventricles. Type II blocks are more dangerous than type I blocks because they are usually permanent and may progress suddenly to complete heart block, especially in the setting of an acute anterior MI, and almost always require permanent cardiac pacemaker placement. When second-degree AV block occurs with a fixed conduction ratio of 2:1, it is not possible to differentiate between a Mobitz type I (Wenckebach) and Mobitz type II block.



**FIGURE 2-16. A.** Second-degree Mobitz II AV block. **B.** Second-degree AV block with 2:1 AV conduction.

#### *Emergency Department Care and Disposition*

1. **Atropine** 0.5 to 1 milligram IV bolus repeated every 5 min as needed up to 2 milligrams total dose is first-line treatment for symptomatic patients. All patients should have transcutaneous pacing pads positioned and ready for use in the case of further deterioration into complete heart block.
2. Initiate transcutaneous cardiac pacing (see section on sinus bradycardia) in patients unresponsive to atropine.
3. If transcutaneous pacing is unsuccessful, initiate transvenous pacing (0.2 to 20 mA at 40 to 140 beats/min via a semi-floating or balloon-tipped pacing catheter).

### **Third-Degree (Complete) AV Block**

#### *Clinical Features*

In third-degree AV block, there is no AV conduction. The ventricles are paced by an escape pacemaker from the AV node or infranodal conduction

system at a rate slower than the atrial rate ([Fig. 2-17](#)). When third-degree AV block occurs at the AV node, a junctional escape pacemaker takes over with a ventricular rate of 40 to 60 beats/min; and, because the rhythm originates from above the bifurcation of the His bundle, the QRS complexes are narrow. Nodal third-degree AV block may develop in up to 8% of acute inferior MIs and it is usually transient, although it may last for several days.



**FIGURE 2-17.** Third-degree AV block.

When third-degree AV block occurs at the infranodal level, the ventricles are driven by a ventricular escape rhythm at a rate slower than 40 beats/min. Third-degree AV block located in the bundle branch or the Purkinje system invariably has an escape rhythm with a wide QRS complex. Like Mobitz type II block, this indicates structural damage to the infranodal conduction system and can be seen in acute anterior MIs. The ventricular escape pacemaker is usually inadequate to maintain cardiac output and is unstable with periods of ventricular asystole.

*Emergency Department Care and Disposition*

1. Perform transcutaneous cardiac pacing in unstable patients until a transvenous pacemaker can be placed.
2. In stable patients, apply transcutaneous pacing pads. Treat the same as second-degree Mobitz II AV block.

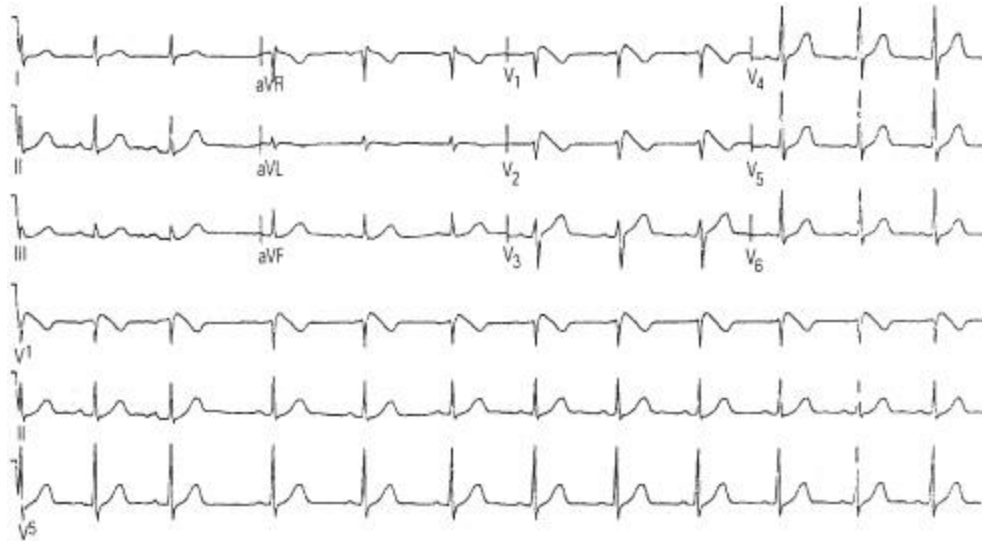
## **FASCICULAR BLOCKS**

Conduction blocks may arise in one or more of the three infranodal conduction pathways. Blockage of either of the left fascicles does not

prolong the QRS duration, but will change the QRS axis. Left anterior fascicular block (LAFB) causes left axis deviation while left posterior fascicular block (LPFB) causes right axis deviation. Right bundle branch block (RBBB) will prolong the QRS duration ( $> 0.12$  sec) and cause a RSR in the early precordial leads (V1-2). Bifascicular block denotes a combination of any two of these fascicles, the most notable of which is left bundle branch block (LAFB + LPFB). Trifascicular block denotes the presence of first degree AV block in the presence of a bifascicular block and is indicative of significant conduction system disease that includes the AV node, thus increasing the risk of Mobitz II or third-degree AV block and the potential need for permanent pacemaker placement.

## **CONDUCTION ABNORMALITIES THAT CAN CAUSE RHYTHM DISTURBANCES**

Brugada syndrome and long-QT syndrome increase the risk of spontaneous VT/VF and require evaluation for implantable cardiac defibrillator placement when diagnosed. Brugada syndrome is a genetic disorder of fast sodium channels causing a RBBB-pattern in the early precordial leads (V1-2) with a pathognomonic J-point elevation and saddle-shaped or sloped ST segment ([Fig. 2-18](#)). Long QT syndrome is characterized by a QT interval  $> 470$  msec in men and  $> 480$  msec in women and may be congenital or acquired, leading to an increased risk of torsades de pointes.



**FIGURE 2-18.** Brugada syndrome.

## **PRETERMINAL RHYTHMS**

### **Pulseless Electrical Activity**

Pulseless electrical activity is the presence of electrical complexes without accompanying mechanical contraction of the heart. Potential mechanical causes should be diagnosed and treated, including severe hypovolemia, cardiac tamponade, tension pneumothorax, massive pulmonary embolus, MI, and toxic ingestions (eg, tricyclic antidepressants, calcium channel blockers,  $\beta$ -blockers). In addition, profound metabolic abnormalities such as acidosis, hypoxia, hypokalemia, hyperkalemia, and hypothermia also should be considered and treated.

After intubation and initiating CPR, administer **epinephrine**, 1 milligram IV/IO (1:10 000 solution) every 3 to 5 min. If giving via endotracheal tube, increase the dose 2 to 2.5 times and follow with several rapid ventilations to disperse the drug. Treatment is guided by rapid identification and treatment of the underlying cause. Use agents with  $\alpha$ -adrenergic activity, such as

norepinephrine and phenylephrine, to improve vascular tone when indicated. Electrical pacing is not effective.

## **Idioventricular Rhythm**

Idioventricular rhythm is a ventricular escape rhythm at slower than 40 beats/min with a QRS wider than 0.16 second. It is associated with infranodal AV block, massive MI, cardiac tamponade, and exsanguinating hemorrhage.

After intubation and initiating CPR, treatment includes identifying contributing mechanical factors (eg, aggressive volume resuscitation) and  $\alpha$ -adrenergic agents.

## **Asystole (Cardiac Standstill)**

Asystole is the complete absence of cardiac electrical activity and carries a grim prognosis. Treatment is the same as that for pulseless electrical activity.

# **CARDIAC PACEMAKERS AND AUTOMATED INTERNAL CARDIAC DEFIBRILLATORS (AICD)**

Pacemakers, AICDs, or combination units may be used in patients with a history of sudden death, heart failure, or cardiomyopathy. Malfunction can occur at any level of the device, including infection or hematoma in the pocket housing the device, lead infection/displacement, failure to pace, failure to sense, overpacing, or inappropriate defibrillation. Most pacemakers will have a magnetic switch which, when triggered by magnet application to the unit, will cause the pacemaker to function in a fixed asynchronous mode.



*Emergency Department Care and Disposition*

1. Evaluation should include an ECG, electrolytes, and chest x-ray to assess lead position and integrity. Arrangements should be made for electrical interrogation of the unit.
2. Patients with pacing failure may require treatment based on their underlying rhythm and associated symptoms.
3. Patients with overpacing may require magnet application to convert the pacemaker to asynchronous mode pacing at a lower rate.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 22, “Cardiac Rhythm Disturbances,” by Joseph S. Piktel; Chapter 23, “Pharmacology of Antiarrhythmics,” by Brad A. Miller and Elizabeth A. Clements; and Chapter 24 “Pharmacology of Vasopressor Agents” by Brad A. Miller and Elizabeth A. Clements.

## **CHAPTER 3**

# **Resuscitation of Children and Neonates**

Marc F. Collin

Children primarily develop cardiac arrest secondary to hypoxia from respiratory arrest or shock syndromes. Because of age and size differences among children, drug dosages, compression and respiratory rates, and equipment sizes differ considerably ([Table3-1](#)).

Item	54-70	70-85	85-95	95-107	107-124	124-138	138-155
Endotracheal tube size (mm)	3.5	4.0	4.5	5.0	5.5	6.0	6.5
Lip-to length (mm)	10.5	12.0	13.5	15.0	16.5	18.0	19.5
Laryngoscope	1 straight	1 straight	2 straight	2 straight or curved	2 straight or curved	2-3 straight or curved	3 straight or curved
Suction catheter	8F	8F-10F	10F	10F	10F	10F	12F
Stylet	6F	6F	6F	6F	14F	14F	14F
Oral airway	Infant/small child	Small child	Child	Child	Child/small adult	Child/adult	Medium adult
Bag-valve mask	Infant	Child	Child	Child	Child	Child/adult	Adult
Oxygen mask	Newborn	Pediatric	Pediatric	Pediatric	Pediatric	Adult	Adult
Vascular access (gauge)							
Catheter	22-24	20-22	18-22	18-22	18-20	18-20	16-20
Butterfly	23-25	23-25	21-23	21-23	21-23	21-22	18-21
Neurologic tube	5F-6F	6F-10F	10F	10F-12F	12F-14F	14F-18F	18F
Urinary catheter	5F-6F	6F-10F	10F	10F-12F	10F-12F	12F	12F
Chest tube	10F-12F	15F-20F	20F-24F	20F-24F	24F-32F	28F-32F	32F-40F
Blood pressure cuff	Newborn/infant	Infant/child	Child	Child	Child	Child/adult	Adult

Directions for use: (1) measure patient length with centimeter tape; (2) using measured length in centimeters, access appropriate equipment column.

**TABLE 3-1** Length-Based Equipment Chart (Length = Centimeters)\*

## **PEDIATRIC RESUSCITATION**

## **CARDIOPULMONARY**

### **Securing the Airway**

The airway in infants and children is smaller, variable in size, and more anterior than that in the adult. The prominent occiput and relatively large tongue and epiglottis may lead to obstruction when the child is in the supine position.

Mild extension of the head in the sniffing position opens the airway. Chin lift or jaw thrust maneuvers may relieve obstruction of the airway related to the tongue. Oral airways are not commonly used in pediatrics but may be useful in the unconscious child who requires continuous jaw thrust or chin lift to maintain airway patency. Oral airways are inserted by direct visualization with a tongue blade.

A bag-valve-mask system is commonly used for ventilation. Minimum volume for ventilation bags for infants and children is 450 mL. The tidal volume necessary to ventilate children is 10 to 15 mL/kilogram. Observation of chest rise and auscultation of breath sounds will ensure adequate ventilation.

Endotracheal intubation usually is performed with a Miller straight blade with a properly sized tube. Resuscitation measuring tapes have been found to be the most accurate for determining tube size. The formula 16 plus age in years divided by 4 calculates approximate tube size. Uncuffed tubes are used in children up to 8 years.

Initiate ventilation at 20 breaths/min for infants, 15 breaths/min for young children, and 10 breaths/min for adolescents unless hyperventilation is required.

### **Rapid Sequence Intubation**

Rapid sequence intubation is the administration of an intravenous (IV) induction agent with a neuromuscular blocking agent to facilitate endotracheal intubation.

1. Prepare equipment, medication, and personnel before initiation of RSI. Check equipment function.
2. Preoxygenate the patient with 100% oxygen.
3. In children, cricoid pressure can occlude the pliable trachea. Release cricoid pressure, if applied, if laryngoscopy and intubation are difficult.
4. Refer to [Table 3-2](#) for specific induction and paralytic agents used in children.

Medication	Dose <sup>†</sup>	Comments
Induction agents		
Etomidate	0.3 milligram/kilogram	Preserves hemodynamic stability; may suppress adrenal axis even in a single dose; short acting, requires anxiolysis or analgesia after intubation
Ketamine	1–2 milligrams/kilogram	Bronchodilator, preserves respiratory drive, cardiovascular stimulant; drug of choice for intubation for asthma
Propofol	1–2 milligrams/kilogram	Rapid push, higher dose in infants, may cause hypotension; short acting, requires ongoing anxiolysis or analgesia after intubation
Paralytics		
Rocuronium	1 milligram/kilogram	Nondepolarizing agent; longer duration than succinylcholine
Succinylcholine	< 10 kilograms: 1.5–2.0 milligrams/kilogram > 10 kilograms: 1.0–1.5 milligrams/kilogram	Shorter duration than rocuronium; better intubating conditions at 60 s; may cause bradycardia in children and hyperkalemic cardiac arrest in children with undiagnosed neuromuscular disease
Sedatives		
Midazolam	0.1 milligram/kilogram	Short-acting sedative
Lorazepam	0.1 milligram/kilogram	Longer-acting sedative
Analgesics		
Fentanyl	1–2 micrograms/kilogram	Short-acting analgesic; preserves hemodynamic stability
Morphine	0.1–0.2 milligram/kilogram	Longer-acting analgesic; may cause histamine release

\*Premedication is no longer routinely recommended in children due to a lack of supporting evidence.

<sup>†</sup>Rapid-sequence intubation medications can be given IO when IV access cannot be obtained.

### **TABLE 3-2** Common Rapid-Sequence Intubation Medications in Children\*

5. Intubate the trachea, confirm proper placement, and secure the tube.
6. **Atropine** 0.02 milligram/kilogram (minimum dose, 0.1 milligram; maximum dose, 1 milligram) may be used for symptomatic reflex bradycardia.

### **Vascular Access**

Airway management is paramount in pediatric arrest and should not be delayed while obtaining vascular access.

Try peripheral veins (antecubital, hand, foot, or scalp) first. Intraosseous access is also a quick, safe, and reliable route for administering fluids and resuscitation medications. The proximal tibia is the most commonly used site. If peripheral IV or IO is unsuccessful, percutaneous access of the femoral vein or saphenous vein cutdown may be attempted.

There are several manual and mechanical devices available for IO insertion. The insertion site is 1 to 3 cm below the anterior tibial tuberosity and in the middle of the anteromedial surface of the tibia. Using sterile technique, insert the device to penetrate the cortex. Then, remove the cannula from the needle, confirm needle placement by aspirating bone marrow or infusing 5 to 10 cc of saline, and secure the device.

Begin fluid resuscitation with rapid infusion of isotonic saline, 20 mL/kilogram IV bolus. Repeat as needed. If shock or hypotension persist after several boluses, consider initiating a pressor.

### **Drugs**

Proper drug dosages in children require knowledge of the patient's weight. Use a length-based system when an exact weight is unavailable.

The rule of 6s may be used to quickly calculate continuous infusions of drugs such as dopamine and dobutamine. The calculation is 6 milligrams times weight in kilogram: fill to 100 mL with 5% dextrose in water. The infusion rate in mL per hour equals the micrograms per kilogram per min

rate (ie, an infusion running at 1 mL/h = 1 microgram/kilogram/min, or 5 mL/h = 5 micrograms/kilogram/min).

**Epinephrine** is indicated in pulseless arrest and in hypoxia-induced bradycardia unresponsive to oxygenation and ventilation. The initial dose is 0.01 milligram/kilogram (0.1 mL/kilogram of 1:10,000 solution) IV/IO or 0.1 milligram/kilogram (0.1 mL/kilogram) of 1:1,000 solution by endotracheal route. Subsequent doses may be administered every 3 to 5 min as needed.

Consider sodium bicarbonate if ventilation, epinephrine, and chest compressions fail to correct acidosis.

Calcium may be useful in treating hyperkalemia, hypocalcemia, and calcium-channel blocker overdose. Calcium may be given as **calcium chloride**, 20 milligrams/kilogram (0.2 milligram/kilogram of 10% solution), or **calcium gluconate**, 60 to 100 milligrams/kilogram (0.6-1 milligram/kilogram of 10% solution), via IV or IO route.

## Dysrhythmias

Dysrhythmias in infants and children are most often the result of respiratory insufficiency or hypoxia. Careful attention to oxygenation and ventilation, along with correction of hypoxia, acidosis, and fluid balance, are the cornerstones of dysrhythmia management in children.

[Tables 3-3](#), [3-4](#), and [3-5](#) summarize electrical and drug therapies of unstable cardiac rhythms in children.

1. If bradycardia causing cardiorespiratory compromise: a. Provide oxygen and/or ventilation as necessary. b. If heart rate remains bradycardic (<60) with poor perfusion, begin CPR.
2. If symptomatic bradycardia persists, administer <b>epinephrine 0.01 milligram/kilogram (0.1 mL/kilogram of 1:10,000 concentration)</b> IV/IO and repeat every 3 to 5 min as necessary. <b>ET epinephrine 0.1 milligram/kilogram (0.1 mL of 1:1000 concentration)</b> may be used if IV access is unavailable. If bradycardia is due to increased vagal tone or primary AV conduction block, give <b>atropine: 0.02 milligram/kilogram (minimum dose, 0.1 milligram; maximum dose, 1 milligram);</b> may repeat.
3. Consider <b>emergency pacing</b> in patients with complete heart block or sinus node dysfunction unresponsive to oxygenation, ventilation, chest compressions, and medications.
4. Identify and treat the underlying cause.

**TABLE 3-3** Treatment of Pediatric Bradycardia

<p>1. Initiate BLS; CPR; administer oxygen, attach monitor/defibrillator if available. If after any of the steps below the rhythm becomes stable, begin post-resuscitation efforts.</p>
<p>2. <b>If rhythm is shockable, ie, ventricular fibrillation (VF) or ventricular tachycardia (VT):</b></p> <p>a. Defibrillate once w/ <b>2 J/kilogram</b>; may use pediatric AED if &gt;1 y/o (1-8 year olds); Check rhythm.</p> <p>b. If unstable cardiac rhythm persists, resume CPR for 5 cycles. Interrupt only to administer <b>4 J/kilogram</b> (may use pediatric AED) for VF/VT. Give <b>epinephrine 0.01 milligram/kilogram (0.1 mL of 1:10,000 concentration)</b> IV/IO and repeat every 3-5 min as necessary. <b>ET epinephrine</b> 0.1 milligram/kilogram (0.1 mL of 1:1000 concentration) may be used if IV access is unavailable. Check rhythm.</p> <p>c. If unstable cardiac rhythm persists, resume CPR for 5 cycles. Interrupt only to administer <b>4 J/kilogram</b> (may use pediatric AED) for VF/VT. Consider <b>amiodarone</b> 5 milligrams/kilogram IV/IO, <b>lidocaine</b> 1 milligram/kilogram IV/IO, <b>magnesium</b> 25-50 milligrams/kilogram IV/IO. Treat reversible causes. Check rhythm.</p> <p>d. Repeat steps b and c until stable rhythm or decision to discontinue resuscitation.</p>
<p>3. <b>If rhythm is not shockable, ie asystole:</b></p> <p>a. Resume CPR. Give <b>epinephrine 0.01 milligram/kilogram (0.1 mL of 1:10,000 concentration)</b> IV/IO and repeat every 3-5 min as necessary. <b>ET epinephrine</b> 0.1 milligram/kilogram (0.1 mL of 1:1000 concentration) may be used if IV access is unavailable. Check rhythm.</p> <p>b. If asystole persists, repeat step above until a treatable rhythm develops or decision to discontinue resuscitation.</p>

**TABLE 3-4** Treatment of Pediatric Pulseless Arrest



Provide oxygen and ventilation as necessary. Attach monitor/defibrillator if available; perform ECG and evaluate QRS duration.
<b>NARROW COMPLEX QRS (&lt;0.09 sec)</b>
<b>Sinus tachycardia:</b>
<ul style="list-style-type: none"> <li>a. Identify and treat underlying cause.</li> <li>b. ECG reveals normal P waves, variable R-R intervals, constant P-R intervals; infant rate usually &lt;220 bpm; child rate usually &lt;180 bpm)</li> </ul>
<b>Supraventricular tachycardia:</b>
<ul style="list-style-type: none"> <li>a. Try <b>vagal maneuvers first</b>; if unsuccessful, administer <b>adenosine</b>, 0.1 milligram/kilogram (maximum, 6 milligrams) by rapid IV push; if unsuccessful, double dose to 0.2 milligram/kilogram (maximum, 12 milligrams).</li> <li>b. Another option is <b>synchronized cardioversion</b>, 0.5-1 J/kilogram. If unsuccessful, double to 1-2 J/kilogram. Sedate patient, if possible.</li> <li>c. EKG reveals absent/abnormal P waves, HR not variable (infant rate usually &gt;220 bpm; child rate usually &gt; 180 bpm)</li> </ul>
<b>WIDE COMPLEX QRS(≥0.09 sec)</b>
<b>Ventricular tachycardia:</b>
<ul style="list-style-type: none"> <li>1. Perform <b>synchronized cardioversion</b>, 0.5-1 J/kilogram. Sedate if possible. Consider giving <b>adenosine</b> if electrical cardioversion is not delayed.</li> <li>2. Other options include <b>amiodarone</b>, 5 milligrams/kilogram IV over 20-60 min or <b>procainamide</b>, 15 milligrams/kilogram IV over 30-60 min.</li> <li>3. Expert consultation is advised.</li> </ul>

**TABLE 3-5** Treatment of Pediatric Tachycardia With Poor Perfusion

The most common rhythm seen in pediatric arrest is bradycardia leading to asystole. Oxygenation and ventilation will often correct this problem. Epinephrine may be useful if the child is unresponsive to this respiratory intervention.

The next most common dysrhythmia in children is narrow complex supraventricular tachycardia (SVT), with rates between 250 and 350 beats per min. On EKG, p waves are either absent or abnormal. It may be difficult to distinguish between a fast sinus tachycardia and SVT. The presence of normal p waves is strongly suggestive of sinus tachycardia rather than SVT. Young infants may have sinus tachycardia with rates faster than 200 beats/min. Patients with sinus tachycardia may have a history of fever, dehydration, or shock, while SVT is usually associated with a vague, nonspecific history.

## **Defibrillation and Cardioversion**

Ventricular fibrillation and ventricular tachycardia are rare in children. When present, immediate defibrillation at 2 J/kilogram is recommended followed by 1 to 2 min of CPR (5 cycles of 15:2 compressions and ventilations) to restore coronary perfusion and improve oxygen delivery to the myocardium before additional attempts at defibrillation. If the first defibrillation attempt is unsuccessful, double the energy to 4 J/kilogram for each subsequent attempt.

Synchronized cardioversion, 0.5 J/kilogram, is used to treat other unstable tachydysrhythmias. Double the energy level to 1 J/kilogram, if the first attempt is unsuccessful.

Use the largest pads or paddles that still allow contact of the entire paddle with the chest wall. When using paddles, electrode cream or paste is used to prevent burns. One paddle is placed on the right of the sternum at the second intercostal space, and the other is placed at the left midclavicular line at the level of the xiphoid.

## **NEONATAL RESUSCITATION**

1. The first step in neonatal resuscitation is to maintain body temperature. Dry the infant and place on a radiant warmer. If available, place the limbs and torso of very-low-birth-weight newborns (<1500 grams) in specially developed polyethylene bags to help maintain normothermia.
2. Reserve immediate suctioning for babies who have obvious obstruction to spontaneous breathing or who require positive-pressure ventilation. If needed, suction the mouth and then the nose with a bulb syringe or mechanical suction device. Routine deep suctioning is not recommended as it may result in vagally induced bradycardia.
3. Aspiration of meconium-stained amniotic fluid may result in both morbidity and mortality. If the infant is vigorous after delivery, do not suction the mouth, nares, or airway. If the infant is depressed after delivery, perform direct tracheal suctioning using direct laryngoscopic visualization and an endotracheal tube fitted with a meconium-suctioning adapter. If attempted intubation is prolonged and unsuccessful, abandon

further attempts and begin bag-mask ventilation if the heart rate is <100 beats/min.

4. Assess heart rate, respiratory effort, color, and activity quickly over the next 5 to 10 seconds. If the infant is apneic or the heart rate is slow (<100 beats/min), administer positive-pressure ventilation using bag-mask ventilation. For term babies, begin resuscitation with room air rather than 100% oxygen. Use pulse oximetry to guide the use of supplementary oxygen.

If the baby is bradycardic (HR<60 beats/min) after 90 seconds of resuscitation with room air or with blended oxygen, increase the oxygen concentration to 100% until heart rate is normalized. Provide assisted ventilation at rates of 40 to 60 breaths/min. Begin with an inflation pressure of about 20 cm H<sub>2</sub>O. Pressures as high as 30 to 40 cm H<sub>2</sub>O may be required initially in some term infants if chest rise is inadequate and the baby is not responding. However, high inflation pressures may result in pneumothorax, especially in premature infants.

5. If no improvement is noted after 30 seconds of bag-mask ventilation or the infant's condition deteriorates further, perform endotracheal intubation and ventilation. CO<sub>2</sub> detectors aid in determining whether the baby is properly intubated but color change is dependent on adequate circulation. Proper tube placement can also be determined by noting good chest rise, visualization of vapor steam in the endotracheal tube, and auscultation.
6. If the heart rate is still slower than 60 beats/min after intubation and assisted ventilation for 30 seconds, begin cardiac compressions at 90 chest compressions and 30 breaths each min (3:1 ratio). The '2 thumb-encircling hands' technique is preferred to the '2-finger' technique when performing chest compressions.
7. If there is still no improvement in heart rate, initiate drug therapy. Vascular access may be obtained peripherally or via the umbilical vein. The most expedient procedure in the neonate is to place a catheter in the umbilical vein and advance to 10 to 12 cm or until free flow of blood is seen in the catheter.
8. Push **epinephrine**, 0.01 to 0.03 milligram/kilogram of 1:10,000 solution IV, which equals 0.1 to 0.3 mL/kilogram, if the heart rate is still slower than 60 beats/min despite adequate ventilation and oxygenation. Repeat

every 3 to 5 min if necessary. IV administration is preferred to intratracheal administration.

9. Initiate volume expansion with normal saline (or Type O/Rh negative blood, if available), 10 to 20 mL/kilogram, if hypovolemia is suspected on the basis of pallor, slow capillary refill time, weak pulses, and/or inadequate response to other measures.
10. **Sodium bicarbonate** during neonatal resuscitation remains controversial and is generally not administered during initial resuscitation. A dose of 1 mEq/kilogram of a 4.2% solution (0.5 mEq/mL) IV may be given if there is a significant metabolic acidosis; this therapy should be guided by blood gas values.
11. Naloxone is not recommended in the resuscitation for newborns with respiratory depression.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 14, “Resuscitation of Neonates,” by Marc F. Collin; Chapter 15, “Resuscitation of Children,” by William E. Hauda II; and Chapter 29, “Pediatric Airway Management,” by Robert J. Vissers.

## **CHAPTER 4**

# **Fluids, Electrolytes, and Acid-Base Disorders**

Mary A. Wittler

## **FLUIDS**

When altered, fluids and electrolytes should be corrected in the following order: (a) volume; (b) pH; (c) potassium, calcium, and magnesium; and (d) sodium and chloride. Reestablishment of tissue perfusion often equilibrates the fluid-electrolyte and acid-base balances. Because the osmolarity of normal saline (NS) matches that of serum, it is an excellent fluid for volume replacement. Hypotonic fluids such as 5% dextrose in water (D5W) should never be used to replace volume. Lactated Ringer solution is commonly used for surgical patients or trauma patients; however, only NS can be given in the same line with blood components. D5½NS, with or without potassium, is given as a maintenance fluid. The more concentrated dextrose solutions, D10W or D20W, are used for patients with compromised ability to mobilize glucose stores, such as patients with hepatic failure, or as part of total parental nutrition solutions.

## **CLINICAL ASSESSMENT OF VOLUME STATUS**

Volume loss and dehydration can be inferred by the patient history. Historical features include: vomiting, diarrhea, fever, adverse working conditions, decreased fluid intake, chronic disease, altered level of consciousness, and reduced urine output. Tachycardia and hypotension are late signs of dehydration. On physical examination, one may find dry mucosa, shrunken tongue (excellent indicator), and decreased skin turgor. In infants and children, sunken fontanelles, decreased capillary refill, lack of

tears, and decreased wet diapers are typical signs and symptoms of dehydration. Lethargy and coma are more ominous signs and may indicate a significant comorbid condition. Laboratory values are not reliable indicators of fluid status. Plasma and urine osmolarity are perhaps the most reliable measures of dehydration. Blood urea nitrogen (BUN), creatinine, hematocrit, and other chemistries are insensitive.

Volume overload is a purely clinical diagnosis and presents with edema (central or peripheral), respiratory distress (pulmonary edema), and jugular venous distention (in congestive heart failure). The significant risk factors for volume overload are renal, cardiovascular, and liver diseases. Blood pressure does not necessarily correlate with volume status alone; patients with volume overload can present with hypotension or hypertension.

## **MAINTENANCE FLUIDS**

- Adult: D5½NS at 75 to 125 mL/h + 20 mEq/L potassium chloride for an average adult (approximately 70 kilograms).
- Children: D5½NS or D10½NS, 100 mL/kilogram/d for the first 10 kilograms of body weight, 50 mL/kilogram/d for the second 10 kilograms, and 20 mL/kilogram/d for every kilograms thereafter. (See [Chapter 81](#) for further discussion of pediatric fluid management.)

## **ELECTROLYTE DISORDERS**

If the clinical picture and the laboratory data conflict, repeat the lab test prior to initiating therapy. Correcting a single abnormality may not be the only intervention needed because most electrolytes exist in equilibrium with others. Abnormalities should be corrected at the same rate they develop; however, slower correction is usually safe unless the condition warrants rapid or early intervention (eg, hypoglycemia or hyperkalemia). Evaluation of electrolyte disorders frequently requires a comparison of the measured and calculated osmolarities (number of particles per liter of solution). To calculate osmolarity, measured serum values in mEq/L are used:

osmolarity (mOsm/L) =  $2 [\text{Na}^+] + (\text{glucose}/18) + (\text{BUN}/2.8) + (\text{ETOH}/4.6)$

## **Hyponatremia ( $[\text{Na}^+] < 135 \text{ mEq/L}$ )**

### *Clinical Features*

The clinical manifestations of hyponatremia occur when the  $[\text{Na}^+]$  drops below 120 mEq/L; they include nausea, weakness, headache, agitation, hallucinations, cramps, confusion, lethargy, and seizures.

### *Diagnosis and Differential*

Evaluate volume status and measured and calculated serum osmolarities. True hyponatremia presents with reduced osmolarity and is further differentiated based on volume status and urine  $[\text{Na}^+]$ . This state results from primary water gain,  $[\text{Na}^+]$  loss greater than that of water, or alteration in the distribution of water. Factitious hyponatremia (false low measurement of the serum sodium) is due to hyperglycemia, hyperproteinemia, hyperlipidemia, and other osmotically active solutes and is associated with a normal to high osmolarity. The syndrome of inappropriate antidiuretic hormone, characterized by hyponatremia, inappropriately elevated urine osmolality despite low serum osmolarity, elevated urine sodium, and clinical euvolemia, is a diagnosis of exclusion. Causes of hyponatremia are listed in [Table 4-1](#).

<p>Hypotonic (true) hyponatremia (<math>P_{osm} &lt; 275</math>)</p> <p>Hypovolemic hyponatremia</p> <ul style="list-style-type: none"> <li>Extrarenal losses (urinary <math>[Na^+] &lt; 20</math> mEq/L) <ul style="list-style-type: none"> <li>Volume replacement with hypotonic fluids</li> <li>Sweating, vomiting, diarrhea, fistula</li> <li>Third-space sequestration (burns, peritonitis, pancreatitis)</li> </ul> </li> <li>Renal losses (urinary <math>[Na^+] &gt; 20</math> mEq/L) <ul style="list-style-type: none"> <li>Diuretic use</li> <li>Aldosterone deficiency</li> <li>Salt-wasting nephropathies; renal tubular acidosis</li> <li>Osmotic diuresis (mannitol, hyperglycemia, hyperuricemia)</li> </ul> </li> </ul> <p>Euvolemic hyponatremia (urinary <math>[Na^+]</math> usually <math>&gt; 20</math> mEq/L)</p> <ul style="list-style-type: none"> <li>Inappropriate ADH secretion (CNS, lung, or carcinoma disease)</li> <li>Physical and emotional stress or pain</li> <li>Myxedema, Addison disease, Sheehan syndrome</li> <li>Drugs, water intoxication</li> </ul> <p>Hypervolemic hyponatremia</p> <ul style="list-style-type: none"> <li>Urinary <math>[Na^+] &gt; 20</math> mEq/L <ul style="list-style-type: none"> <li>Renal failure (inability to excrete free water)</li> </ul> </li> <li>Urinary <math>[Na^+] &lt; 20</math> mEq/L <ul style="list-style-type: none"> <li>Cirrhosis</li> <li>Congestive heart failure</li> <li>Nephrotic syndrome</li> </ul> </li> </ul> <p>Isotonic (pseudo) hyponatremia (<math>P_{osm} 275-295</math>)</p> <ul style="list-style-type: none"> <li>Hyperproteinemia, hyperlipidemia</li> </ul> <p>Hypertonic hyponatremia (<math>P_{osm} &gt; 295</math>)</p> <ul style="list-style-type: none"> <li>Hyperglycemia, mannitol excess and glycerol use</li> </ul>
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Key: ADH = antidiuretic hormone, CNS = central nervous system.

**TABLE 4-1** Causes of Hyponatremia

***Emergency Department Care and Disposition***

1. Correct existing volume or perfusion deficits with NS.
2. In euvolemic or hypervolemic patients, restrict fluids (500 to 1500 mL of water daily)
3. In severe hyponatremia ( $[Na^+] < 120$  mEq/L) that has developed rapidly with central nervous system (CNS) changes such as coma or seizures, give **hypertonic saline**, 3% NS (513 mEq/L), at 25 to 100 mL/h. The  $[Na^+]$  should not be corrected faster than 0.5 mEq/L/h in chronic hyponatremia or 1.0 mEq/L/h in acute hyponatremia. The  $[Na^+]$  correction should not exceed 12 mEq/L/day.



4. The sodium dose can be calculated as follows: weight (kilograms)  $\times$  0.6  $\times$  (desired  $[\text{Na}^+]$  – measured  $[\text{Na}^+]$ )=sodium deficit (mEq).
5. Complications of rapid correction include congestive heart failure (CHF) and central pontine myelinolysis.

## **Hypernatremia ( $[\text{Na}^+] > 150 \text{ mEq/L}$ )**

### *Clinical Features*

An osmolarity increase of 2% stimulates thirst to prevent hypernatremia. Symptoms of hypernatremia are usually noticeable at a serum osmolarity  $> 350$  or  $[\text{Na}^+] > 158 \text{ mEq/L}$ . Initial symptoms include irritability, tremulousness, and ataxia. Lethargy, coma, and seizures may be seen with osmolarities above 400. Morbidity and mortality are highest in infants and the elderly who may be unable to respond to increased thirst.

### *Diagnosis and Differential*

Hypernatremia is most commonly caused by a decrease in total body water due to decreased intake or excessive loss. It is less often due to an increase in total body  $[\text{Na}^+]$ . Common causes are GI loss, hyperpyrexia, and excessive sweating. An important etiology of hypernatremia is diabetes insipidus (DI), which results in the loss of hypotonic urine. Central DI (no antidiuretic hormone secreted) results from CNS disease, surgery, or trauma. Nephrogenic DI (unresponsive to antidiuretic hormone) results from congenital disease, drugs, hypercalcemia, hypokalemia, or renal disease. The causes of hypernatremia are listed in [Table 4-2](#).

Loss of water
Reduced water intake
Defective thirst drive
Unconsciousness
Inability to drink water
Lack of access to water
Water loss in excess of sodium
Vomiting, diarrhea
Sweating, fever
Diabetes insipidus
Drugs including lithium, phenytoin
Dialysis
Osmotic diuresis, renal concentrating defects
Thyrotoxicosis
Severe burns
Gain of sodium
Increased intake
Increased salt use, salt pills
Hypertonic saline ingestion or infusion
Sodium bicarbonate administration
Mineralocorticoid or glucocorticoid excess
Primary aldosteronism
Cushing syndrome

**TABLE 4-2** Causes of Hypernatremia

*Emergency Department Care and Disposition*

1. Correct existing volume or perfusion deficits with NS or Lactated Ringer solution. Free water deficits are corrected with  $\frac{1}{2}$ NS. Avoid lowering the  $[\text{Na}^+]$  more than 10 mEq/L/day.
2. Each liter of water deficit causes the  $[\text{Na}^+]$  to increase 3 to 5 mEq/L. Use the formula to calculate the free water deficit: water deficit (L) =  $(\text{measured } [\text{Na}^+]/\text{desired } [\text{Na}^+]) - 1$ .
3. If no urine output is observed after NS or lactated Ringer solution rehydration, rapidly switch to  $\frac{1}{2}$  NS: unload the body of the extra sodium by using a diuretic (eg, **furosemide** 20 to 40 milligrams IV).
4. Central diabetes insipidus DI is treated with **desmopressin** with careful monitoring of electrolytes, urine osmolarity, and specific gravity. Consult a specialist.
5. In children with a serum sodium level higher than 180 mEq/L, consider peritoneal dialysis using high-glucose, low- $[\text{Na}^+]$  dialysate in

consultation with a pediatric nephrologist (see [Chapter 81](#) for further discussion).

## **Hypokalemia ( $[K^+] < 3.5$ mEq/L)**

### *Clinical Features*

The signs and symptoms of hypokalemia usually occur at levels below 2.5 mEq/L and affect the following body systems: the CNS (weakness, cramps, hyporeflexia, paresthesias), gastrointestinal system (ileus), cardiovascular system (dysrhythmias, worsening of digoxin toxicity, hypotension or hypertension, U waves, ST-segment depression, and prolonged QT interval), and renal system (metabolic alkalosis and increased ammonia production); glucose intolerance also can develop.

### *Diagnosis and Differential*

Causes can be grouped by decreased  $[K^+]$  intake, increased  $[K^+]$  excretion, or transcellular shift. The most common cause is the use of loop diuretics. [Table 4-3](#) lists the causes.

Shift into the cell
Alkalosis and sodium bicarbonate
β-Adrenergics
Administration of insulin and glucose
Hypokalemic periodic paralysis
Reduced intake
Increased loss
Renal loss
Primary & secondary hyperaldosteronism, Bartter syndrome
Diuretics, osmotic diuresis, postobstructive diuresis
Renal tubular acidosis
Renal artery stenosis
Miscellaneous
Licorice use
Use of chewing tobacco
Hypercalcemia
Liddle syndrome
Magnesium deficiency
Acute leukemia
Drugs and toxins (PCN, lithium, L-dopa, theophylline)
GI loss (vomiting, diarrhea, fistulas), malabsorption

Key: GI = gastrointestinal, PCN = penicillin.

**TABLE 4-3** Causes of Hypokalemia

*Emergency Department Care and Disposition*

1. A 20 mEq/dose [K<sup>+</sup>] will raise the [K<sup>+</sup>] by 0.25 mEq/L.
2. In stable patients, oral replacement is preferred (safe and rapid); a 20 to 40 mEq [K<sup>+</sup>] dose is used.
3. In unstable patients, IV **Potassium chloride** (KCl in doses of 10 to 20 mEq/h may be given). Add no more than 40 mEq of KCl to each liter of IV fluid. Infusion rates should not exceed 40 mEq/hr. Doses greater than 20 mEq/h should be given through a central line. Patients should be monitored continuously for dysrhythmias.

**Hyperkalemia ([K<sup>+</sup>] > 5.5 mEq/L)**

*Clinical Features*

The most concerning and serious manifestations of hyperkalemia are the cardiac effects. At levels of 6.5 to 7.5 mEq/L, the electrocardiogram (ECG) shows peaked T waves (precordial leads) and prolonged PR and short QT intervals. At levels of 7.5 to 8.0 mEq/L, the QRS widens and the P wave flattens. At levels above 8 mEq/L, a sine-wave pattern, ventricular fibrillation, and heart blocks occur. Neuromuscular symptoms include weakness and paralysis. Gastrointestinal symptoms include vomiting, colic, and diarrhea.

### *Diagnosis and Differential*

Beware of pseudohyperkalemia, which is caused by hemolysis after blood draws. Renal failure with oliguria is the most common cause of true hyperkalemia. Appropriate tests for management include an ECG, electrolytes, calcium, magnesium, arterial blood gases (check for acidosis), urine analysis, and a digoxin level in appropriate patients. Causes of hyperkalemia are listed in [Table 4-4](#).

Factitious
Laboratory error
Hemolysis and leukocytosis
Increased plasma [K <sup>+</sup> ] load
Exogenous: diet, salt substitutes, [K <sup>+</sup> ] containing medications
Endogenous: hemolysis, GI bleeding, catabolic states, crush injury
Decreased [K <sup>+</sup> ] excretion
Renal failure
Impaired renin-aldosterone axis
Addison disease
Primary hypoaldosteronism
Other (heparin, ACE inhibitors, prostaglandin inhibitors)
Tubular potassium secretory defect
Sickle cell disease
Systemic lupus erythematosus
Postrenal transplantation
Obstructive uropathy
Potassium sparing diuretics
Abnormal potassium distribution
Insulin deficiency
Hypertonicity (hyperglycemia)
β-Adrenergic blockers
Exercise
Drugs: succinylcholine, β-agonists, digitalis intoxication
Acidosis

Key: GI = gastrointestinal.

**TABLE 4-4** Causes of Hyperkalemia

***Emergency Department Care and Disposition***

1. Symptomatic patients are treated in a stepwise approach: stabilize the cardiac membrane with CaCl<sub>2</sub> or Ca-gluconate; shift [K<sup>+</sup>] into the cell using glucose and insulin and/or bicarbonate and/or albuterol; enhance [K<sup>+</sup>] excretion by using sodium polystyrene sulfonate (Kayexalate), diuretics, or dialysis in severe cases.
2. For levels over 7.0 mEq/L or if there are any ECG changes, give: IV **calcium chloride (10%)** 5 to 10 mL or IV **calcium gluconate (10%)** 10 to 20 mL. In children, give calcium gluconate (10%) 0.5 mL/kilogram.
3. The presence of digoxin toxicity with hyperkalemia is an indication for digoxin immune Fab therapy (see [Chapter 108](#)). Avoid using calcium.
4. In acidotic patients, consider giving 50 to 100 mEq of **sodium bicarbonate slow IV**. In children 1 to 2 mEq/kilogram is given slow IV.

5. Give 50 mL (25 grams) of **D50W** with 10 to 20 units regular **insulin** IV push (5 to 10 units in dialysis patients). In children, give 0.5 to 1 gram/kilogram of glucose as D10W plus insulin 0.1 units/kilogram.
6. Diuresis is maintained with **furosemide** 20 to 40 milligrams IV.
7. **Kayexalate** (PO or rectal [PR]) 1 gram binds 1 mEq  $[K^+]$ . Administer Kayexalate 15 to 30g PO with sorbitol or 30 to 50 grams PR with sorbitol. Sorbitol is used because Kayexalate is constipating. Kayexalate can exacerbate CHF. In children, give Kayexalate 1gram/kilogram PO or PR.
8. In patients with acute renal failure, consult a nephrologist for emergent dialysis.
9. **Albuterol** 5 to 10 milligrams by nebulization may also be used to lower  $[K^+]$ .
10. Kayexalate and insulin/glucose therapies last several hours; all other therapies (except hemodialysis) have transient effects. Frequent monitoring of  $[K^+]$  should occur.

## **Hypocalcemia ( $[Ca^{2+}] < 8.5$ mEq/L or ionized level $< 2.0$ mEq/L)**

### *Clinical Features*

The signs and symptoms of hypocalcemia are usually seen with ionized  $[Ca^{2+}]$  levels below 1.5 mEq/L. Symptoms include paresthesias, increased deep tendon reflexes (DTRs), cramps, weakness, confusion, and seizures. Patients also may demonstrate the Chvostek sign (twitch of the corner of mouth on tapping with finger over cranial nerve VII at the zygoma) or the Trousseau sign (more reliable; carpal spasm when the blood pressure cuff is left inflated at a pressure above the systolic blood pressure for longer than 3 min). Alkalosis decreases the ionized  $[Ca^{2+}]$  fraction (physiologically active form) without changing the total calcium level. Low  $[Ca^{2+}]$  decreases myocardial contractility, so patients may present with CHF or prolonged QT intervals on the ECG.

### *Diagnosis and Differential*

Causes include: shock, sepsis, fat embolism, renal failure, pancreatitis, drugs (usually cimetidine), hypoparathyroidism, hyperphosphatemia, vitamin D deficiency, hypomagnesemia, and fluoride poisoning.

*Emergency Department Care and Disposition*

1. If asymptomatic, use **calcium gluconate** tablets 1 to 4 grams/d PO divided every 6 hours with or without **vitamin D** (calcitriol 0.2 micrograms two times a day). Milk is not a good substitute.
2. In symptomatic patients or those with severe hypocalcemia, give **calcium gluconate**, or **calcium chloride**, 10 mL 10% solution IV slowly over 10 min.
3. Replace magnesium in conjunction with  $[Ca^{2+}]$ .

**Hypercalcemia ( $[Ca^2]$  > 10.5 mEq/L or ionized  $[Ca^{2+}]$  > 2.7 mEq/L)**

Several factors affect the serum calcium level: parathyroid hormone increases calcium and decreases phosphate; calcitonin and vitamin D metabolites decrease calcium. Decreased  $[H^+]$  causes a decrease in ionized  $[Ca^{2+}]$ . A decrease in albumin causes a decrease in  $[Ca^{2+}]$  but not in the ionized portion.

*Clinical Features*

Clinical signs and symptoms develop at levels above 12 milligrams/dL. Patients often have profound volume depletion; concomitant electrolyte abnormalities are frequent. A mnemonic to aid recall of common hypercalcemia symptoms is *stones* (renal calculi), *bones* (osteolysis), *psychic moans* (lethargy, weakness, fatigue, and confusion), and *abdominal groans* (abdominal pain, constipation, polyuria, and polydipsia). ECG changes include depressed ST segments, widened T waves, shortened QT intervals, and heart blocks.



### *Diagnosis and Differential*

Most cases of hypercalcemia are due to hyperparathyroidism or malignancy. A mnemonic to aid recall of the common causes is *PAM P. SCHMIDT*: parathyroid hormone, Addison disease, multiple myeloma, Paget disease, sarcoidosis, cancer, hyperthyroidism, milk-alkali syndrome, immobilization, excess vitamin D, and thiazides.

### *Emergency Department Care and Disposition*

1. Initiate treatment in patients with severe symptoms,  $[Ca^{2+}]$  above 14 milligrams/ dL, or significant dehydration. Restore fluid deficits, enhance calcium elimination, and decrease osteoclastic activity.
2. Correct fluid deficits with NS; several liters may be required. Correct concomitant electrolyte abnormalities cautiously.
3. Loop diuretics inhibit the renal resorption of  $[Ca^{2+}]$ , but worsen dehydration and other electrolyte abnormalities. They are no longer recommended for malignancy-related hypercalcemia. In isolated cases, furosemide (10 to 40 milligrams IV) may be administered after fluid deficits are corrected, with careful attention to avoid dehydration. Thiazide diuretics should not be used.
4. Drugs that inhibit osteoclastic activity include the bisphosphonates, calcitonin, and glucocorticoids. Recommendations for initiating therapy in the ED are lacking; consultation with a specialist is advised.

## **Hypomagnesemia**

### *Clinical Findings*

$[Mg^{2+}]$ ,  $[K^+]$ , and  $[PO_4^-]$  move together intra- and extracellularly. Hypomagnesemia presents with CNS symptoms (depression, vertigo, ataxia, seizures, increased DTR, or tetany) or cardiac symptoms (arrhythmias, prolonged PR, QRS and QT, or worsening of digitalis effects). Also seen are anemia, hypotension, hypothermia, and dysphagia.

### *Diagnosis and Differential*

In adults, the most common cause is alcoholism, followed by poor nutrition, cirrhosis, pancreatitis, correction of diabetic ketoacidosis (DKA), excessive gastrointestinal losses, and renal wasting (especially diurectic use). Severe  $[\text{Mg}^{2+}]$  depletion can occur before significant laboratory changes are seen.

### *Emergency Department Care and Disposition*

1. Correct volume deficits and other electrolyte abnormalities. Oral  $[\text{Mg}^{2+}]$  replacement is sufficient for most patients.
2. In patients with severe symptoms and normal renal function, administer 2 grams **magnesium sulfate IV** over an hour, followed by 6 grams over the first 24 hours. Continuous cardiac monitoring and frequent DTR checks are recommended.

## **Hypermagnesemia**

### *Clinical Findings*

Signs and symptoms manifest progressively: nausea and somnolence occur first, followed by muscle weakness and loss of DTRs. Respiratory depression, hypotension, heart block, and cardiac arrest occur at progressively higher magnesium levels.

### *Diagnosis and Differential*

Hypermagnesemia is rare. Common causes are renal failure with concomitant ingestion of  $[\text{Mg}^{2+}]$ -containing preparations (antacids) and lithium ingestion. Serum levels are diagnostic. Hyperkalemia, hypercalcemia, and hyperphosphatemia often present concurrently.

### *Emergency Department Care and Disposition*

1. In many patients, stopping  $[\text{Mg}^{2+}]$  intake is sufficient. More aggressive therapy includes rehydration with NS.
2. In severely symptomatic patients, give 5 mL (10% solution) of **calcium chloride** IV to antagonize the effects of magnesium.

## ACID-BASE PROBLEMS

### Initial Assessment

#### *Clinical Features*

Several conditions should alert the clinician to possible acid-base disorders: history of renal, endocrine, or psychiatric disorders (drug ingestion); or signs of acute disease: tachypnea, cyanosis, Kussmaul respiration, respiratory failure, shock, changes in mental status, vomiting, diarrhea, or other acute fluid losses.

Acidosis is due to gain of acid or loss of alkali; causes may be metabolic (fall in serum  $[\text{HCO}_3^-]$ ) or respiratory (rise in  $\text{PCO}_2$ ). Alkalosis is due to loss of acid or addition of base and is metabolic (rise in serum  $[\text{HCO}_3^-]$ ) or respiratory (fall in  $\text{PCO}_2$ ). The lungs and kidneys primarily maintain the acid–base regulation. Metabolic disorders prompt an immediate compensatory change in ventilation, thereby venting  $\text{CO}_2$  in cases of metabolic acidosis or retaining it in cases of metabolic alkalosis. The effect of the kidneys in response to metabolic disorders is to excrete the hydrogen ion (with chloride) and recuperate  $[\text{HCO}_3^-]$ , a process that requires hours to days. The compensatory mechanisms of the lungs and kidneys will return the pH toward, but not to, normal.

#### *Diagnosis and Differential*

Diagnosis and differential must begin with defining the nature of the acid-base disorder (with the stepwise approach below) and then determining the

most likely etiology from the differential listings in each section that follows. In a mixed disorder, the pH,  $\text{PCO}_2$ , and  $[\text{HCO}_3^-]$  may be normal, and the only clue to a metabolic acidosis is a widened anion gap (AG, see step 4 below).

## Stepwise Method of Acid-Base Clinical Problem Solving

Use the patient's pre-illness values as a baseline if available; otherwise, a pH of 7.4,  $[\text{HCO}_3^-]$  of 24 mEq/L, and  $\text{PCO}_2$  of 40 mm Hg can be considered normal.

1. Examine the pH for acidemia (pH < 7.4) or alkalemia (pH > 7.4).
2. Establish the primary mechanism by evaluating the  $[\text{HCO}_3^-]$  and  $\text{PCO}_2$ .
  - Metabolic acidosis: pH < 7.4 and  $[\text{HCO}_3^-] < 24$  meq/L
  - Metabolic alkalosis: pH > 7.4 and  $[\text{HCO}_3^-] > 24$  meq/L
  - Respiratory acidosis: pH < 7.4 and  $\text{PCO}_2 > 40$  mm Hg
  - Respiratory alkalosis: pH > 7.4 and  $\text{PCO}_2 < 40$  mm Hg
3. Calculate the AG:  $\text{AG: } [\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-]) = \text{approximately } 10 \text{ to } 12 \text{ mEq/L}$   
approximately 10 to 12 mEq/L is normal.
  - If the AG is increased compared with the known previous value or greater than 15, then an anion gap metabolic acidosis is present.
  - If the AG is unchanged and a metabolic acidosis is present (low  $[\text{HCO}_3^-]$ ), then a normal anion gap (or hyperchloremic) acidosis is present.
4. For anion gap metabolic acidosis, evaluate for a concomitant hidden metabolic process: each 1 mEq/L decrease in  $[\text{HCO}_3^-]$  results in a 1 mEq/L increase in AG. Compare the  $\Delta$  Gap (=present gap - 12) to the  $\Delta$   $[\text{HCO}_3^-]$  (= 24 - present  $[\text{HCO}_3^-]$ ).
  - $\Delta$  Gap =  $\Delta$   $[\text{HCO}_3^-]$ : pure anion gap metabolic acidosis
  - $\Delta$  Gap >  $\Delta$   $[\text{HCO}_3^-]$ : concomitant metabolic alkalosis is likely present
  - $\Delta$  Gap <  $\Delta$   $[\text{HCO}_3^-]$ : concomitant non-AG acidosis is likely present
5. Estimate the compensatory response for the primary process. If the compensatory response is not as expected, then the compensatory

mechanism requires more time for complete mobilization or a secondary acid-base disturbance exists.

- Metabolic acidosis: expected  $PCO_2 = (1.5 \times [HCO_3^-] + 8) \pm 2$ . A simpler observation is the  $PCO_2$  decreases by 1 mm Hg for every 1 mEq/dL decrease in  $[HCO_3^-]$ . This process takes 12 to 24 hrs.
- Metabolic alkalosis: expected  $PCO_2 = 0.9 [HCO_3^-] + 16$ .
- For either of above formulas, if:
  - Current  $PCO_2 =$  expected  $PCO_2$  : normal respiratory compensation
  - Current  $PCO_2 <$  expected  $PCO_2$  : possible concomitant respiratory alkalosis
  - Current  $PCO_2 >$  expected  $PCO_2$  : possible concomitant respiratory acidosis
  - Respiratory acidosis: clinically judge whether the process is acute (<72 hrs) or chronic (> 72 hrs). The  $[HCO_3^-]$  increases 1mEq/L (acute) or 4 mEq/L (chronic) for every 10 mm Hg increase in  $PCO_2$ .
  - Respiratory alkalosis: clinically judge whether the process is acute (72 hrs) or chronic (> 72 hrs). The  $[HCO_3^-]$  decreases 2mEq/L (acute) or 5 mEq/L (chronic) for every 10 mm Hg decrease in  $PCO_2$ .
  - For either of above formulas, if:
    - Current  $[HCO_3^-] =$  expected  $[HCO_3^-]$  : normal metabolic compensation.
    - Current  $[HCO_3^-] <$  expected  $[HCO_3^-]$ : possible concomitant metabolic acidosis.
    - Current  $[HCO_3^-] >$  expected  $[HCO_3^-]$ : possible concomitant metabolic alkalosis.

6. See the sections below for determining the etiology and management.

## Metabolic Acidosis

Metabolic acidosis should be divided into an increased and normal AG acidosis. The term *anion gap* is misleading because the serum has no gap between total positive and negative ions; however, the unmeasured anions exceed the unmeasured cations.

### *Clinical Features*

No matter the etiology, acidosis can cause nausea and vomiting, abdominal pain, change in sensorium, and tachypnea, sometimes a Kussmaul respiratory pattern. Acidosis causes many negative physiologic effects that result in hypoxia. Patients may present with nonspecific complaints or shock.

### *Diagnosis and Differential*

Causes of metabolic acidosis can be divided into 2 main groups: (a) those associated with increased production of organic acids ([Table 4-5](#)); and (b) those associated with a loss of  $[\text{HCO}_3^-]$ , failure to excrete  $[\text{H}^+]$ , or addition of  $[\text{H}^+]$  ([Table 4-6](#)).

Lactic acidosis
Type A: Decrease in tissue oxygenation
Type B: Normal tissue oxygenation
Renal failure (acute or chronic)
Ketoacidosis
Diabetes
Alcoholism
Prolonged starvation (mild acidosis)
High-fat diet (mild acidosis)
Ingestion of toxic substances
Elevated osmolar gap
Methanol
Ethylene glycol
Normal osmolar gap
Salicylate
Paraldehyde
Cyanide

**TABLE 4-5** Causes of High Anion-Gap Metabolic Acidosis

With a Tendency to Hyperkalemia	With a Tendency to Hypokalemia
Subsiding DKA	Renal tubular acidosis–type I (classical distal acidosis)
Early uremic acidosis	Renal tubular acidosis–type II (proximal acidosis)
Early obstructive uropathy	Acetazolamide
Renal tubular acidosis–type IV	Acute diarrhea with losses of $\text{HCO}_3^-$ and $\text{K}^+$
Hypoaldosteronism (Addison disease)	Ureterosigmoidostomy with increased resorption of $\text{H}^+$ and $\text{Cl}^-$ and losses of $\text{HCO}_3^-$ and $\text{K}^+$
Infusion or ingestion of $\text{HCl}$ , $\text{NH}_4\text{Cl}$ , lysine-HCl, or arginine-HCl	Obstruction of artificial ileal bladder
Potassium-sparing diurectics	Dilution acidosis

Key: DKA = diabetic ketoacidosis.

**TABLE 4-6** Causes of Normal Anion Gap Metabolic Acidosis

Causes of anion gap metabolic acidosis include renal failure, lactic acidosis, ketoacidosis, and toxins. A mnemonic to aid the recall of the causes of increased AG metabolic acidosis is: *A MUD PILES*: alcohol, methanol, uremia, DKA, paraldehyde, iron and isoniazid, lactic acidosis, ethylene glycol, salicylates, and starvation. Caution should be used when applying the *A MUD PILES* mnemonic because the presence of alcohol in the patient’s blood does not rule out a more serious cause of acidosis. Iron and isoniazid exert their effects on the AG due to lactic acidosis. Causes of normal anion gap acidosis include GI or renal loss of  $[\text{HCO}_3^-]$ . A mnemonic that can aid the recall of normal AG metabolic acidosis is *USED CARP*: ureterostomy, small bowel fistulas, extra chloride, diarrhea, carbonic anhydrase inhibitors, adrenal insufficiency, renal tubular acidosis, and pancreatic fistula.

***Emergency Department Care and Disposition***

1. Address the underlying cause to restore tissue perfusion and oxygenation.
2. Administer fluids, oxygen, and ventilation as needed.
3. For specific etiologies, consult the appropriate chapters in this handbook for further guidance.

Indications for bicarbonate therapy are listed in [Table 4-7](#). Give 0.5 mEq/kilogram **bicarbonate** for each mEq/L desired rise in  $[\text{HCO}_3^-]$ . The goal is to restore adequate buffer capacity ( $[\text{HCO}_3^-] > 8$  mEq/dL) or achieve clinical improvement in shock or dysrhythmias. Seventy-five mEq

of **sodium bicarbonate** in 500 mL D5W produces a nearly isotonic solution for infusion.

Indication	Rationale
Severe hypobicarbonatemia (< 4 mEq/L)	Insufficient buffer concentrations may lead to extreme increases in acidemia with small increases in acidosis
Severe acidemia (pH < 7.20) with signs of shock or myocardial irritability that is not rapidly responsive to support measures	Therapy for the underlying cause of acidosis depends on adequate organ perfusion
Severe hyperchloremic acidemia*	Lost bicarbonate must be regenerated by kidneys and liver, which may require days

\*No specific definition by pH exists. The presence of serious hemodynamic insufficiency despite supportive care should guide the use of bicarbonate therapy for this indication.

**TABLE 4-7** Indications for Bicarbonate Therapy in Metabolic Acidosis

## Metabolic Alkalosis

Metabolic alkalosis is classified as  $[Cl^-]$  sensitive or  $[Cl^-]$  insensitive. The two most common causes of metabolic alkalosis are excessive diuresis (with loss of potassium, hydrogen ion, and chloride) and excessive loss of gastric secretions (with loss of hydrogen ion and chloride).

### *Clinical Features*

Symptoms of the underlying disorder (usually fluid loss) dominate the clinical presentation, but general symptoms of metabolic alkalosis include muscular irritability, tachydysrhythmia, and impaired oxygen delivery. In most cases, there is also an associated hypokalemia and hypochloremia.

Patients with  $[Cl^-]$  sensitive causes present with hypovolemia secondary to vomiting, diarrhea, or diuretic therapy. Patient with  $[Cl^-]$  insensitive causes present with normo- to hyper-volemia associated with excess mineralocorticoid activity (renin-secreting tumors, adrenal hyperplasia, hyperaldosteronism, Cushing syndrome).

### *Emergency Department Care and Disposition*



1. Administer NS to treat dehydration.
2. Monitor electrolytes.

## **Respiratory Acidosis**

### *Clinical Features*

Respiratory acidosis secondary to hypoventilation may be life threatening. The clinical picture usually is dominated by the underlying disorder. Typically, respiratory acidosis depresses the mental function, which may progressively slow the respiratory rate. Patients may be confused, somnolent, and, eventually, unconscious. Pulse oximetry may be misleading, making arterial blood gases essential for the diagnosis.

The differential diagnosis includes drug overdose, CNS disease, chest wall disease, pleural or lung disease, and trauma.

### *Emergency Department Care and Disposition*

1. The treatment is to improve ventilation. Depressed mental status is an indication for intubation. An exception to this is opiate intoxication where rapid administration of naloxone may improve ventilation.
2. Treat the underlying disorder.

## **Respiratory Alkalosis**

### *Clinical Features*

Hyperventilation syndrome is a problematic diagnosis for the emergency physician because many life-threatening disorders present with tachypnea and anxiety: asthma, pulmonary embolism, diabetic ketoacidosis, and others. Symptoms of respiratory alkalosis often are dominated by the primary disorder promoting the hyperventilation. Symptoms of

hyperventilation include dizziness, carpal-pedal spasm, and, frequently, a chest pain described as tightness.

The diagnosis of hyperventilation due to anxiety is a diagnosis of exclusion. Arterial blood gases can be used to rule out acidosis and hypoxia. Causes of respiratory alkalosis include CNS tumor or stroke, infection or fever, hypoxia, lung disease, hyperthyroidism, toxins (eg, sympathomimetics or aspirin), liver disease, pregnancy, and anemia.

*Emergency Department Care and Disposition*

1. Treat the underlying cause.
2. Rule out life threatening causes of hyperventilation before diagnosing anxiety. Anxiolytics may be helpful, such as **lorazepam** 1 to 2 milligrams IV or PO.
3. Rebreathing into a paper bag can cause hypoxia; it is not recommended.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 19, “Acid-Base Disorders,” by David D. Nicolaou and Gabor D. Kelen; Chapter 20, “Blood Gases,” by Todd L. Slesinger; and Chapter 21, “Fluids and Electrolytes,” by Gabor D. Kelen and Edbert Hsu.

## **CHAPTER 5**

# **Therapeutic Approach to the Hypotensive Patient**

John E. Gough

## **THERAPEUTIC APPROACH TO THE HYPOTENSIVE PATIENT**

Shock is circulatory insufficiency that creates an imbalance between tissue oxygen supply (delivery) and oxygen demand (consumption). Such tissue hypoperfusion is associated with decreased venous oxygen content and metabolic acidosis (lactic acidosis). Shock is classified into four categories based on etiology: (a) hypovolemic, (b) cardiogenic, (c) distributive (eg, neurogenic and anaphylactic), and (d) obstructive.

### **CLINICAL FEATURES**

Factors that influence the clinical presentation of a patient in shock include the etiology, duration, and severity of the shock state and the underlying medical status of the patient. Often the precipitating cause of shock may be readily apparent (eg, acute myocardial infarction, trauma, gastrointestinal [GI] bleeding, or anaphylaxis). It is not uncommon for the patient to present with nonspecific symptoms (eg, generalized weakness, lethargy, or altered mental status). A targeted history of the presenting symptoms and previously existing conditions (eg, cardiovascular disease, GI bleeding, adrenal insufficiency, or diabetes) will aid in identifying the cause and guide the initial treatment of shock. Drug use (prescribed and nonprescribed) is an essential element of the initial history. Medication use may be the cause or a contributing factor to the evolution of shock. For example, diuretics can lead to volume depletion and cardiovascular medications (eg,  $\beta$ -blockers) can depress the pumping action of the heart.

The possibility of drug toxicity and anaphylactic reactions to medications also should be considered.

Assessment of vital signs is a routine part of the physical examination; however, no single vital sign or value is diagnostic in the evaluation of the presence or absence of shock. The patient's temperature may be elevated or subnormal. The presence of hyperthermia or hypothermia may be a result of endogenous factors (eg, infections or hypometabolic states) or exogenous causes (eg, environmental exposures). The heart rate is typically elevated; however, bradycardia may be present with many conditions, such as excellent baseline physiologic status (young athletes), intraabdominal hemorrhage (secondary to vagal stimulation), cardiovascular medication use (eg,  $\beta$ -blockers and digoxin), hypoglycemia, and preexisting cardiovascular disease.

The respiratory rate is frequently elevated early in shock. Increased minute ventilation, increased dead space, bronchospasm, and hypocapnia may be seen. As shock progresses, hypoventilation, respiratory failure, and respiratory distress syndrome may occur.

Shock is usually, but not always, associated with systemic arterial hypotension, with a systolic blood pressure (BP) below 90 mm Hg. The insensitivity of blood pressure to detect global tissue hypoperfusion has been repeatedly confirmed. Thus, shock may occur with a normal blood pressure, and hypotension may occur without shock. Early in shock, the systolic and diastolic BPs may initially be normal or elevated in response to a compensatory mechanism such as tachycardia and vasoconstriction. As the body's compensatory mechanisms fail, BP typically falls. Postural changes in BP, commonly seen with hypovolemic states, will precede overt hypotension. The pulse pressure, the difference between systolic and diastolic BP measurements, may be a more sensitive indicator. The pulse pressure usually rises early in shock and then decreases before a change in the systolic BP is seen.

In addition to these vital sign abnormalities, other cardiovascular manifestations may include neck vein distention or flattening and cardiac dysrhythmias. A third heart sound (S3) may be auscultated in high-output states. Decreased coronary perfusion pressures can lead to myocardial ischemia, decreased ventricular compliance, increased left ventricular diastolic pressures, and pulmonary edema.

Decreased cerebral perfusion leads to mental status changes such as weakness, restlessness, confusion, disorientation, delirium, syncope, and coma. Patients with longstanding hypertension may exhibit these changes without severe hypotension. Cutaneous manifestations may include pallor, pale or dusky skin, sweating, bruising, petechiae, cyanosis (may not be evident if the hemoglobin level is less than 5 grams/dL), altered temperature, and delayed capillary refill.

GI manifestations resulting from low flow states may include ileus, GI bleeding, pancreatitis, acalculous cholecystitis, and mesenteric ischemia. To conserve water and sodium, levels of aldosterone and antidiuretic hormone are increased. This results in a reduced glomerular filtration rate, redistribution of blood flow from the renal cortex to the renal medulla, and oliguria. In sepsis, a paradoxical polyuria may occur and be mistaken for adequate hydration.

Early in shock a common metabolic abnormality is a respiratory alkalosis. As the shock state continues and compensatory mechanisms begin to fail, anaerobic metabolism occurs, leading to the formation of lactic acid and resulting in a metabolic acidosis. Other metabolic abnormalities that may be seen are hyperglycemia, hypoglycemia, and hyperkalemia.

## **DIAGNOSIS AND DIFFERENTIAL**

The clinical presentation and presumed etiology of shock will dictate the diagnostic studies, monitoring modalities, and interventions used. The approach to each patient must be individualized; however, frequently performed laboratory studies include complete blood count; platelet count; electrolytes, blood urea nitrogen, and creatinine determinations; prothrombin and partial thromboplastin times; and urinalysis. Other tests commonly used are arterial blood gas, lactic acid, fibrinogen, fibrin split products, D-dimer, and cortisol determinations; hepatic function panel; cerebrospinal fluid studies; and cultures of potential sources of infection. A pregnancy test should be performed on all females of childbearing potential. No single laboratory value is sensitive or specific for shock. Other common

diagnostic tests include radiographs (chest and abdominal), electrocardiographs, computed tomography scans (chest, head, abdomen, and pelvis), and echocardiograms. Beside US may also help determine the etiology of shock. The following are helpful in this assessment: subcostal cardiac view, inferior vena cava view, parasternal long-axis cardiac view, apical four-chamber cardiac view, right upper quadrant abdominal view, pelvic view, and abdominal aorta view.

Continuous monitoring of vital signs should be instituted in all patients. Additionally, modalities such as pulse oximetry, end-tidal CO<sub>2</sub>, central venous pressure, central venous O<sub>2</sub> saturation, cardiac output, and calculation of systemic vascular resistance and systemic oxygen delivery may be indicated.

A search to determine the etiology of the shock must be undertaken. Lack of response to appropriate stabilization measures should cause the clinician to evaluate the patient for a more occult cause. First, the physician must be certain that the basic steps of resuscitation have been carried out appropriately. Consider whether or not the patient has been adequately volume resuscitated. Early use of vasopressors may elevate the central venous pressure and mask the presence of continued hypovolemia. Ensure that all equipment is connected and functioning appropriately. Carefully expose and examine the patient for occult wounds. Consider less commonly seen diagnoses, such as cardiac tamponade, tension pneumothorax, adrenal insufficiency, toxic or allergic reactions, and occult bleeding (eg, rupture ectopic pregnancy, or occult intraabdominal or pelvic bleeding) in the patient who is not responding as expected.

Please refer to the other chapters in this book regarding the evaluation of the specific forms of shock.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The goal of the interventions is to restore adequate tissue perfusion in concert with the identification and treatment of the underlying etiology.

1. Aggressive airway control, best obtained through endotracheal intubation, is indicated. Remember that associated interventions such as medications (ie, sedatives can exacerbate hypotension) and positive pressure ventilation may reduce preload and cardiac output and may contribute to hemodynamic collapse.
2. All patients should receive supplemental high-flow oxygen. If mechanical ventilation is used, neuromuscular blocking agents should be used to decrease lactic acidosis from muscle fatigue and increased oxygen consumption. Arterial oxygen saturation should be restored to > 93% and ventilation controlled to maintain a PaCO<sub>2</sub> of 35 to 40 mm Hg.
3. Circulatory hemodynamic stabilization begins with IV access through large-bore peripheral venous lines. Central venous access aids in assessing volume status (preload) and monitoring ScvO<sub>2</sub>. US guidance has proven helpful with these procedures. Central venous access is the preferred route for the long-term administration of vasopressor therapy. The Trendelenburg position does not improve cardiopulmonary performance compared with the supine position, and it may worsen pulmonary gas exchange and predispose to aspiration. Passive leg raising above the level of the heart with the patient supine can be effective. Early surgical consultation is indicated for internal bleeding. Most external hemorrhage can be controlled by direct compression. Rarely will clamping or tying off of vessels be needed.
4. The type, amount, and rate of fluid replacement remain areas of controversy. There is no difference in survival comparing crystalloid with colloid resuscitation. Crystalloid solutions continue to be recommended because of the increased cost of colloid agents. Most use isotonic crystalloid intravenous fluids (0.9% NaCl, Ringer lactate) in the initial resuscitation phase. Due to the increased cost, lack of proven benefit, and potential for disease transmission (with FFP), the routine use of colloids (5% albumin, purified protein fraction, fresh-frozen plasma [FFP], and synthetic colloid solutions [hydroxyethyl starch or dextran 70]) is questionable. Standard therapy in the hemodynamically unstable patient is 20 to 40 mL/kilogram given rapidly (over 10 to 20 min). Because only about 30% of infused isotonic crystalloids remain in the intravascular space, it is recommended to infuse approximately 3 times the estimated

blood loss in acute hemorrhagic shock. However, the benefits of early and aggressive fluid replacement in these trauma patients remain unproven as do the benefits of permissive hypotension.

5. Blood remains the ideal resuscitative fluid. When possible, use fully cross-matched PRBCs. If the clinical situation dictates more rapid intervention, type-specific, type O (rhesus negative to be given to females of childbearing years) may be used. The decision to use platelets or FFP should be based on clinical evidence of impaired hemostasis and frequent monitoring of coagulation parameters. Platelets are generally given if there is ongoing hemorrhage and the platelet count is  $50\,000/\text{mm}^3$  or lower; administer 6 units initially. FFP is indicated if the prothrombin time is prolonged beyond 1.5 seconds; administer 2 units initially. Trauma patients requiring transfusion of multiple units of packed RBCs should receive FFP and platelets early in ratios that approach 1:1:1 in order to address the accompanying coagulopathy that will likely be present. The use of fresh whole blood has also been advocated and may be the most effective approach for such patients. The potential need for FFP and platelet transfusions should be considered early and reassessed frequently in an effort to detect and limit the adverse effects of trauma-induced coagulopathy.
6. Vasopressors are used after appropriate volume resuscitation has occurred and there is persistent hypotension. Possible choices include: **dobutamine 2.0 to 20.0 micrograms/kilogram/min, dopamine 5.0 to 20.0 micrograms/ kilogram/min, and norepinephrine 0.5 to 30.0 microgram/min.**
7. The goal of resuscitation is to maximize survival and minimize morbidity using objective hemodynamic and physiologic values to guide therapy. A goal-directed approach of urine output  $> 0.5\text{ mL/kilogram/h}$ , CVP 8 to 12 mm Hg, MAP 65 to 90 mm Hg, and  $\text{ScvO}_2 > 70\%$  during ED resuscitation of septic shock significantly decreases mortality.
8. Acidosis should be treated with adequate ventilation and fluid resuscitation. Sodium bicarbonate (1 mEq/kilogram) use is controversial. Use only in the setting of severe acidosis refractory to above-mentioned methods. Correct only to arterial pH 7.25.



9. Early surgical or medical consultation for admission or transfer is indicated.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 25, “Approach to the Patient in Shock,” by Ronny M. Otero, H. Bryant Nguyen, Emanuel P. Rivers; and Chapter 26, “Fluid and Blood Resuscitation,” by José G. Cabañas, James E. Manning and Charles B. Carins.

## **CHAPTER 6**

# **Anaphylaxis, Acute Allergic Reactions, and Angioedema**

Alix L. Mitchell

Allergic reactions range from localized urticaria to life-threatening anaphylaxis. Anaphylaxis refers to the most severe form of immediate hypersensitivity reaction and encompasses both IgE-mediated reactions and anaphylactoid reactions, which do not require a previous sensitizing exposure.

## **CLINICAL FEATURES**

Anaphylaxis may occur within seconds or be delayed over an hour after an exposure; more rapid reactions are associated with higher mortality. Common exposures are foods, medications, insect stings, and allergen immunotherapy injections. Many cases are idiopathic. Criteria for anaphylaxis describe an acute progression of organ system involvement that may lead to cardiovascular collapse. Organ system involvement can include dermatologic (pruritus, flushing, urticaria, erythema multiforme, angioedema), respiratory tract (dyspnea, wheezing, cough, stridor, rhinorrhea), cardiovascular (dysrhythmias, collapse, arrest), gastrointestinal (cramping, vomiting, diarrhea), genitourinary (urgency, cramping), and eye (pruritus, tearing, redness). A biphasic mediator release can occur in up to 20% of cases causing recurrence of symptoms 4 to 8 hours after the initial exposure. Patients on  $\beta$ -blockers are susceptible to an exaggerated allergic response and may be refractory to first line treatment.

## **DIAGNOSIS AND DIFFERENTIAL**

Anaphylaxis is a clinical diagnosis. History may confirm exposure to a possible allergen, such as a new drug, food, or sting. There is no specific test to verify the diagnosis in real time; anaphylaxis should be considered in any rapidly progressing multi-system illness. Workup should be directed at ruling out other diagnoses while stabilizing the patient. The differential depends on the organ systems involved and may include myocardial ischemia, gastroenteritis, asthma, carcinoid, epiglottitis, hereditary angioedema, and vasovagal reactions.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Resuscitation must begin with airway, breathing, and circulation. Patients with confirmed or suspected anaphylaxis should be placed on a cardiac monitor with pulse oximetry, and intravenous access should be obtained.

1. Administer oxygen as indicated by oximetry. Angioedema or respiratory distress should prompt early consideration for intubation. Preparations should be made for “rescue” transtracheal jet insufflation or cricothyroidotomy.
2. Limit further exposure. This may be as simple as stopping an intravenous drug or removing a stinger. First aid measures, ice, and elevation may be helpful for local symptoms.
3. First line therapy for anaphylaxis is **epinephrine**. In patients without cardiovascular collapse, administer 0.3 to 0.5 milligram (0.3 to 0.5 mL of 1:1000; pediatric dose, 0.01 milligram/kilogram to a maximum of 0.5 milligram) intramuscularly in the thigh. The dose may be repeated every 5 min as needed. Patients who are refractory to IM dosing or in significant shock should receive intravenous epinephrine. A bolus of 100 micrograms of 1:100 000 dilution (place 0.1 mL of 1:1000 in 10 mL normal saline) can be given over 5 to 10 min followed by an infusion of 1 to 4 micrograms/min, with close observation for chest pain or arrhythmias.

4. Hypotensive patients require aggressive fluid resuscitation with **normal saline** 1 to 2 L (pediatric dose, 10 to 20 mL/kilogram).
5. Steroids should be used in all cases of anaphylaxis to control persistent or delayed reactions. Severe cases can be treated with **methylprednisolone** 125 milligrams IV (pediatric dose, 2 milligrams/kilogram). Mild allergic reactions can be treated with oral **prednisone** 60 milligrams (pediatric dose, 2 milligrams/kilogram).
6. Every patient with severe allergic symptoms requires antihistamines. **Diphenhydramine** can be given 50 milligrams IV (pediatric dose, 1 milligram/kilogram). In addition, an H<sub>2</sub> blocker such as **ranitidine** 50 milligrams IV (pediatric dose, 0.5 milligram/kilogram) may be helpful.
7. Bronchospasm can be treated with nebulized  $\beta$ -agonists such as **albuterol** 2.5 milligrams. If refractory, consider an inhaled anticholinergic, **ipratropium bromide** 250 micrograms, and intravenous **magnesium** 2 grams (25 to 50 milligrams/kilogram in children) over 20 to 30 min.
8. For patients on  $\beta$ -blockers with hypotension refractory to epinephrine and fluids, use **glucagon** 1 milligram IV every 5 min. An infusion of 5 to 15 micrograms/min should be started once blood pressure improves.
9. Angiotensin-converting enzyme inhibitors are a common trigger for nonallergic angioedema. Airway compromise can develop rapidly. Treatment is supportive. Epinephrine, steroids, and antihistamines are often given although benefit has not been proven.
10. Patients with hereditary angioedema do not respond to treatment for anaphylaxis and should be treated with C1 esterase inhibitor replacement. Treatment with fresh frozen plasma has been reported as an alternative when C1 esterase inhibitor replacement is not available.
11. Unstable or refractory patients merit admission to the intensive care unit. Patients with moderate to severe symptoms should be admitted for observation. Patient with mild allergic reactions should be observed in the ED and may be sent home if symptoms are stable or improving. Stable patients who received epinephrine are generally felt to be safe for discharge after 4 hours without symptoms. Consider observing patients with a history of severe reactions and patients on  $\beta$ -blockers for a longer period.

12. Discharge patients on an antihistamine and a short course of prednisone. Counsel all patients about the need to return to the ED in the event of late recurrence of symptoms and about avoiding future exposures to the allergen, if known. All patients who have experienced severe allergic reactions should have and know how to use an epinephrine autoinjector. Consider Medic-Alert bracelets and referral to an allergist in these patients.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 27, “Anaphylaxis, Acute Allergic Reactions, and Angioedema,” by Brian H. Rowe and Theodore J. Gaeta.

## **SECTION 2 Analgesia, Anesthesia, and Sedation**

## **CHAPTER 7**

# **Acute Pain Management and Procedural Sedation**

**Boris Garber**

Acute pain is present in 50% to 60% of all emergency department (ED) patients. Procedural sedation and analgesia often is needed for painful interventions or diagnostic studies.

## **CLINICAL FEATURES**

Responses to pain vary and may include increased heart rate, blood pressure, respiratory rate, and behavioral changes. Because subjective impressions may be inaccurate, pain is often assessed with objective scales. Pain relief is a dynamic process and reassessment is mandatory.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Pharmacologic and nonpharmacologic interventions may be helpful for treating anxiety and pain in the ED. Nonpharmacologic interventions include the application of heat or cold, immobilization and elevation of injured extremities, explanation and reassurance, music, biofeedback, guided imagery, and distraction methods, such as feeding sucrose solution to infants. Discussing a painful intervention with a patient immediately before the procedure may decrease the anxiety created by anticipation. When pharmacologic intervention is needed, the selection of agent should be guided by the need for sedation or analgesia, the route of delivery, and the desired duration of effects.

### **Acute Pain Control**

Nonopiate Analgesics, such as **acetaminophen**, 650 to 1000 milligrams (15 milligrams/kilogram PO or PR in children) or nonsteroidal antiinflammatory drugs such as **ibuprofen**, 400 to 800 milligrams PO (10 milligrams/kilogram PO in children) can be used to treat mild to moderate pain. Parenteral NSAIDs are no more effective than oral medications. Adverse effects of NSAIDS include gastrointestinal irritation, renal dysfunction, platelet dysfunction, and impaired coagulation. Aspirin should be avoided in children because of an association with Reye syndrome.

Opiates, such as **morphine**, 0.1 milligram/kilogram IV (0.1 to 0.3 milligram/kilogram in children), **fentanyl**, 1.5 micrograms/kilogram IV (1 to 2 micrograms/kilogram in children), and **hydromorphone**, 0.0125 milligram/kilogram IV (0.015 to 0.020 milligram/kilogram in children) are the agents of choice for moderate to severe pain. Additional doses are given every few minutes at half the original dose until pain is controlled. Side effects of opiates include respiratory depression, nausea and vomiting, confusion, pruritus, and urinary retention. Oral opioids, such as **oxycodone**, 5 to 10 milligrams PO (0.1 milligram/kilogram/dose in children) or **hydrocodone** (5 to 10 milligrams PO (0.1 milligram/kilogram/dose) may be tried for pain relief if procedural sedation and analgesia will not be used.

## **Procedural Sedation and Analgesia (PSA)**

The indications for PSA include painful procedures, such as abscess drainage, wound management, tube thoracostomy, orthopedic manipulation, cardioversion, and diagnostic studies. Analgesia is relief from the perception of pain. Minimal sedation is a drug-induced state characterized by normal responses to voice and normal cardiac and ventilatory functions. Moderate sedation and analgesia (conscious sedation) are characterized by responsiveness to voice or light tactile stimulation with normal cardiac and ventilatory functions. Deep sedation and analgesia are characterized by responsiveness to repeated or painful stimulation, potentially inadequate ventilation, and potential loss of protective reflexes. Dissociative sedation is a type of moderate sedation.

### *Preparation*



The risk of aspiration from recent oral intake increases with the depth of sedation. This risk must be balanced with the urgency of the procedure. The complication rate of PSA depends strongly on depth of sedation and patient's physiological reserve as determined by chronic or acute illness. Patients with significantly limited physiologic reserve, those with severe systemic disease, at the extremes of age, and those with predicted difficult airway ([Chapter 1](#)) may be best served with anesthesia consultation.

When PSA is performed, necessary equipment includes a continuous cardiac monitor and pulse oximetry, oxygen, suction, and immediate availability of appropriate-size resuscitation equipment. The patient should be under constant observation by a provider trained in airway management. Informed consent should be obtained. Blood pressure, heart rate, respiratory rate, and level of consciousness should be monitored. Some advocate routine use of capnography to monitor ventilation in sedated patients. The analgesic or sedative agents chosen should be individualized to the patient and the planned procedure. The agents used for PSA often have a narrow therapeutic index. Therefore, the nondissociative agents should be administered in small, incremental intravenous doses, with adequate time between doses to determine peak effect. All patients undergoing PSA should be reassessed continuously. Patients experiencing transient respiratory depression can usually be managed by bag-mask-valve ventilation.

### *Sedation Management*

[Table 7-1](#) describes selected sedation agents for procedural and sedation and analgesia.

Medication	Recommended Dosage	Route of Administration	Onset	Duration	Use
Nitrous oxide	50:50 mixture with oxygen	Inhalational	2-3 min	15-20 min	Minimal sedation
Midazolam	0.05-0.1 milligram/kilogram	IV	1-3 min	1 h	Minimal or moderate sedation
	May repeat 0.05 milligram/kilogram every 2 min until adequately sedated				
	0.1 milligram/kilogram	IM	15-30 min	1-2 h	Minimal sedation
	Children: 0.1 milligram 0.5 milligram/kilogram 0.2 milligram/kilogram	IM PO/PR Intranasally			
Fentanyl	1-3 micrograms/kilogram, can be titrated up to 5 micrograms/kilogram Children: 1-2 microgram/kilogram	IV	<1 min	30-60 min	Minimal sedation
Fentanyl and midazolam	1-2 micrograms/kilogram fentanyl plus midazolam 0.05 milligram/kilogram to 0.1 milligram/kilogram, as needed, up to two times	IV	1-2 min	1 h	Moderate and deep sedation
Methohexital	1 milligram/kilogram	IV	1 min	10 min	Moderate or deep sedation
Pentobarbital	2 milligrams/kilogram to 2.5 milligrams/kilogram followed by 1.25 milligrams/kilogram, as needed, up to two times	IV rate should be < 50 milligrams/min	30-60 s	15+ min	Minimal and moderate sedation Used frequently for radiological procedures
Ketamine	1 milligram/kilogram	IV	1-3 min	10-20 min	Dissociative sedation
	2-5 milligrams/kilogram	IM	5-20 min	30-60 min	Dissociative sedation
	Up to 4 milligrams/kilogram in children				

Ketamine and midazolam	Ketamine as above plus midazolam, 0.05 to 0.1 milligram/kilogram	IV	1-3 min	30-60 min	Dissociative sedation
Etomidate	0.15 milligram/kilogram, followed by 0.1 milligram/kilogram every 2 min, if needed Children: 0.1 milligram/kilogram-0.5 milligram/kilogram	IV	30-60 s	5-10 min	Moderate, deep sedation Associated with amnesia
Propofol	1 milligram/kilogram, followed by 0.5 milligram/kilogram every 3 min, if needed Children: 1-2 milligrams/kilogram	IV	1-2 min	5-10 min	Moderate and deep sedation
Propofol and ketamine	Propofol as above, ketamine 0.5 milligrams/kilogram-0.5 milligram/kilogram Use higher end dose in children	IV	1 min	Propofol- minutes, ketamine 15-45 min	Moderate and deep sedation

Weight based medication doses are the same in adults and children unless otherwise noted.

**TABLE 7-1** Sedation Agents for Procedural Sedation and Analgesia

Fentanyl is the opiate of choice for most brief PSA procedures because of its rapid onset of action. Fentanyl is less likely to cause hypotension than are other opiates. Chest wall rigidity unresponsive to naloxone may occur at higher doses (5 to 15 micrograms/kilogram) or when rapidly administered potentially necessitating neuromuscular blockade and mechanical ventilation. A small dose of **naloxone (0.1 to 0.2 milligram)** may be used to reverse respiratory depression without blocking subsequent analgesia if needed.

Midazolam is commonly used as a sole agent for minimal sedation. Respiratory depression and hypotension may develop. Flumazenil quickly reverses sedation and respiratory depression due to benzodiazepines. Routine use to reverse sedation is not recommended.

Methohexital is an ultrashort acting barbiturate. The most common adverse effect is respiratory depression. Methohexital, which has been used PR in children, may precipitate seizures and should not be used in patients with a seizure disorder. Pentobarbital is an excellent choice for neuroimaging procedures in children.

**Ketamine** is a dissociative analgesic with sedative and amnestic properties that causes minimal respiratory depression. Ketamine may be administered IV, IM, PO, or PR. Ketamine may cause increased intracranial and intraocular pressure, hypersalivation, bronchorrhea, laryngospasm, and a hallucinatory emergence reaction in older children and adults. **Midazolam** (0.01 milligram/kilogram IM or IV or 0.1 milligram/kilogram PO) may attenuate the emergence reaction, but it may cause respiratory depression and delayed ketamine metabolism. Ketamine is contraindicated in children 3 months and younger and in those with airway abnormalities, a history of congestive heart failure, acute closed head or eye injury, altered mental status or psychosis, CNS mass, poorly controlled seizure disorder, active URI, or glaucoma.

**Etomidate**, is a sedative agent with minimal cardiovascular depression. Side effects include nausea and vomiting, myoclonus, and temporary adrenal insufficiency. Respiratory and CNS depressions may occur, especially when administered with opiates or benzodiazepines.

**Propofol** is an anesthetic agent with antiemetic properties administered by intravenous infusion. The most common side effect is respiratory depression and apnea. Side effects include dose-related cardiovascular depression with decreases in systolic blood pressure of 25% to 40%. Hypovolemia should be corrected before propofol administration. Adjunct analgesic is mandatory for painful procedures. Propofol use is contraindicated in patients who are allergic to eggs or soy products.

## **Children**

Children of all ages feel pain, even neonates. Anxiety issues, pain control, and need for sedation must be addressed. Anxiety may be a significant barrier to a successful procedure performance, especially when patient's cooperation is needed. Parents can provide significant anxiety relief and should be allowed to stay with children. Age appropriate distraction techniques should also be employed. Benzodiazepines, such as midazolam, provide effective pharmacologic anxiety relief when needed. Procedural sedation and analgesia should be used when performing painful procedures or when procedures require the patient to be still. Common medications used for pediatric procedural sedation are listed in [Table 7-1](#).

### *Disposition*

Patients are eligible for discharge only when fully recovered. When discharged, the patient must be accompanied by an adult and should not drive or operate machinery for 24 hours. Because many of the agents used for PSA produce anterograde amnesia, discharge instructions must be given to responsible accompanying adults.

## **Local and Regional Anesthesia**

Local and regional anesthetics are essential tools for ED pain management. Agents can be administered topically, by infiltration directly into the area to be anesthetized or into the area of the peripheral nerves supplying the area to be anesthetized, and IV. This discussion focuses on topical and infiltrative anesthesia.

The toxicity of local anesthetics (LAs) is related to the total dose and the rate of plasma concentration increase and is increased in the setting of hypoxia, hypercarbia, and acidosis. The rate of plasma concentration increase is dependent on the vascularity of the site being infiltrated. Therefore, the maximum dose of LAs that can be administered for intercostal block is one-tenth the subcutaneous dose. Toxic effects include confusion, seizures, coma, myocardial depression, and dysrhythmias. Allergic reactions to LAs are uncommon and usually due to a preservative. If an allergy is suspected, the best approach is to use a preservative-free

agent from the other class of LAs. Alternatively, diphenhydramine or benzyl alcohol may be used as an LA in the setting of a true allergy to conventional LAs.

LAs often cause pain during administration. Slow injection through a 27 or 30 gauge needle, injecting through the wound margin, using warm solution, and using buffered (with bicarbonate) solution decrease injection pain.

Epinephrine (1:100 000) is often added to LAs before administration. Addition of epinephrine increases the duration of anesthesia, provides wound hemostasis, and slows systemic absorption. Epinephrine causes vasoconstriction and therefore is avoided in an end-arterial field such as the digits, pinna, nose, and penis in patients with vascular disease.

Lidocaine, which is the most commonly used LA in the ED, has a 2 to 5 min onset of effect and a 1 to 2 hour duration of effect. The maximum dose of infiltrative **lidocaine** is 4.5 milligrams/kilogram without or 7 milligrams/kilogram with epinephrine. Lidocaine is buffered to decrease the pain of injection by adding 1 mL NaHCO<sub>3</sub> to 9 mL lidocaine. Bupivacaine, which has an onset of effect of 3 to 7 min and duration of effect of 90 min to 6 hours, is preferred for prolonged procedures. The maximum dose of infiltrative **bupivacaine** is 2 milligrams/kilogram without or 3 milligrams/kilogram with epinephrine. Buffer bupivacaine with 1 mL NaHCO<sub>3</sub> to 29 mL bupivacaine.

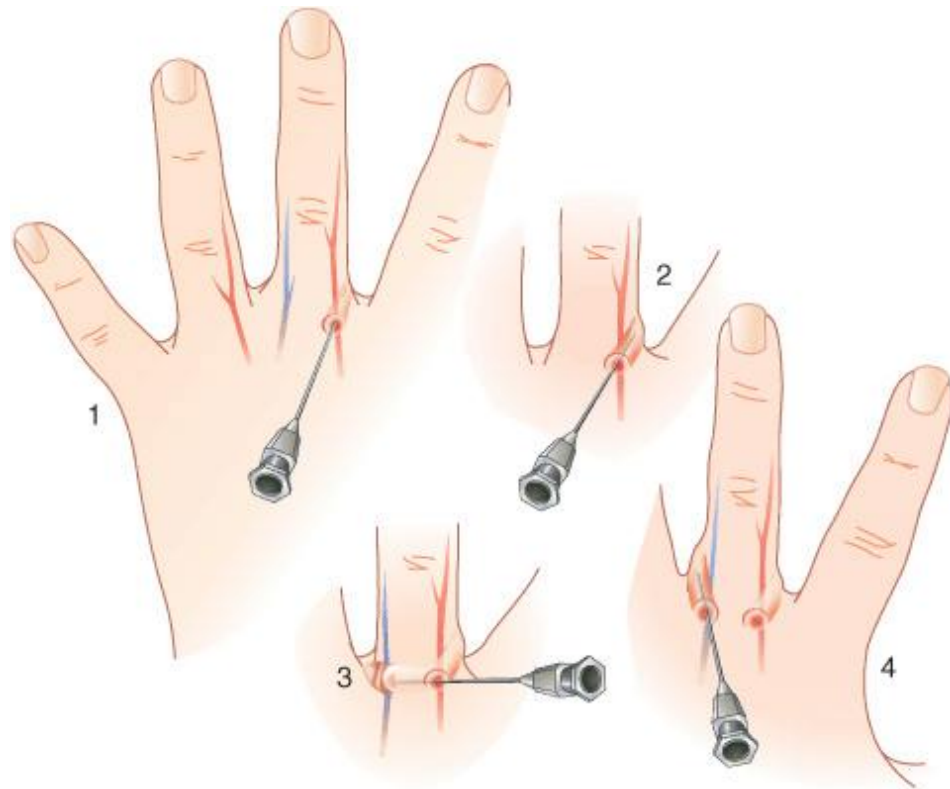
### *Regional blocks*

Regional anesthesia is a technique that infiltrates local anesthetic agents adjacent to peripheral nerves (“nerve blocks”) and is typically used for complicated lacerations, fractures, and dislocations. Distortion of the site is avoided. US guidance can be used. Care must be taken not to inject the anesthetic solution directly into the nerve.

### *Digital Blocks*

Finger and toe blocks are advantageous because less anesthetic is needed, better anesthesia is obtained, and tissues are not distorted. The onset of

anesthesia is delayed when compared with that of LA. Assess and document neurovascular status before the procedure. Lidocaine and bupivacaine are the most commonly used agents and depend on the time needed to perform the procedure. Epinephrine is generally avoided. Complications include nerve injury and intravascular injection leading to systemic toxicity. Always aspirate before injecting to avoid inadvertent intravascular injection of LA.



**FIGURE 7-1.** Needle positions for digital nerve block.

The procedure for digital blocks involves sterile preparation of the skin, followed by the introduction of a 27 gauge or smaller needle into the skin (a skin wheal may be raised before deeper injection) and into one side of the extensor tendon of the affected finger just proximal to the web. After aspiration, approximately 1 mL LA is injected into the tissue on the dorsal surface of the extensor tendon. The needle is advanced toward the palm until its tip is seen beneath the volar skin at the base of the finger just distal to the web. After aspiration, 1 mL LA is injected. Before removing the needle, redirect it across the opposite side of the finger and inject approximately 1

mL across the dorsal digital nerve. Five minutes later, repeat the procedure on the opposite side of the finger ([Fig. 7-1](#)). An alternate method is to inject a 27 gauge needle into the web space between the affected and an adjacent finger while directing the needle to the metacarpal joint of the affected finger. After aspiration, inject 1 to 2 mL into the area of the digital nerve. Before removal of the needle, advance the needle first dorsally and then volarly, and inject 1 mL LA; repeat on the opposite side. Toes can be blocked in similar fashion. Great toes also can be blocked with a modified collar block. A 27 gauge needle is introduced to the dorsolateral aspect of the base of the toe until it blanches the plantar skin. As the needle is withdrawn, 1.5 mL LA is injected. Before the needle is removed, it is passed under the skin on the dorsal aspect of the toe, and 1.5 mL LA is injected as the needle is withdrawn. The needle is reintroduced through the anesthetized skin on the dorsomedial aspect of the toe and advanced until the plantar skin is blanched; as the needle is withdrawn, 1.5 mL LA is injected.

#### ***Local Anesthetic Infiltration***

LAs can provide anesthesia at a site by infiltrating directly into the site or by infiltrating around the peripheral nerves supplying the site. The most common use of LA is infiltration for wound repair or invasive painful procedures. When repairing wounds, LA can be infiltrated into the wound margins or as a “field block” surrounding the wound. When infiltrating intact skin, raising a wheal may cause less pain on subsequent infiltration. LA also can be used in orthopedic procedures, such as fracture and joint reduction, by directly injecting the LA into the affected joint or fracture hematoma.

For some wounds, LA infiltration around the peripheral nerves is advantageous due to decreased total LA required and decreased pain at the site of injection. This is most commonly used for procedures involving the hand, digits, or foot. Before a regional block, assess and document neurovascular status. During administration, the syringe plunger must be drawn back to avoid intravascular injection of LA. Onset of effect of anesthesia with peripheral nerve blocks often is delayed (up to 15 min).

#### ***Topical Anesthetics***



Topical anesthetics can eliminate the need for LA infiltration, are applied painlessly, do not distort wound edges, and may provide hemostasis. Common preparations include lidocaine epinephrine tetracaine (LET), lidocaine prilocaine (EMLA), and various preparations of lidocaine. LET is applied by placing a LET-saturated cotton ball or gauze pad onto the wound for a minimum of 20 to 30 min. LET should not be used on mucous membranes or in end-artery fields.

Topical lidocaine is marketed in a solution, cream, jelly, or ointment. Viscous lidocaine can be used for the temporary relief of inflamed mucous membranes. Lidocaine jelly can be used to facilitate the insertion of urinary catheters, nasogastric tubes, and fiberoptic scopes. As with infiltrative use of lidocaine, care must be taken not to exceed maximal doses.

EMLA is a cream composed of lidocaine and prilocaine used on intact skin to relieve the pain associated with venipuncture, arterial puncture, port access, and other superficial skin procedures. It has a 45 to 60 min onset of effect and a 60 min duration upon withdrawal. Because prilocaine may cause methemoglobinemia, EMLA should be used with caution in infants younger than 3 months and avoided in patients predisposed to methemoglobinemia.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 38, “Acute Pain Management in Adults,” by James Ducharme and Chapter 39, “Pain Management in Infants and Children,” by William M. Lennarz; and Chapter 40, “Local and Regional Anesthesia,” by Douglas C. Dillon and Michael A. Gibbs; and Chapter 41, “Procedural Sedation and Analgesia,” by James R. Miner.

## **CHAPTER 8**

# **Management of Patients With Chronic Pain**

**David M. Cline**

*Chronic pain* is defined as a painful condition that lasts longer than 3 months. It also can be defined as pain that persists beyond the reasonable time for an injury to heal or a month beyond the usual course of an acute disease. Complete eradication of pain is not a reasonable endpoint in most cases. Rather, the goal of therapy is pain reduction and a return to functional status.

## **CLINICAL FEATURES**

Signs and symptoms of chronic pain syndromes are summarized in [Table 8-1](#). Most of these syndromes will be familiar to emergency physicians.

Disorder	Pain symptoms	Signs
Myofascial headache	Constant dull pain, occasionally shooting pain	Trigger points on scalp, muscle tenderness, and tension
Transformed migraine	Initially migraine-like, becomes constant, dull, nausea, vomiting	Muscle tenderness and tension, normal neurologic examination
Fibromyalgia	Diffuse muscular pain, stiffness, fatigue, sleep disturbance	Diffuse muscle tenderness, > 11 trigger points
Myofascial back pain syndrome	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle
Chronic back pain	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	No trigger points, poor ROM in involved muscle
Sciatica (Neurogenic back pain)	Constant or intermittent, burning or aching, shooting or electric shock-like, may follow dermatome; leg pain > back pain	Possible muscle atrophy in area of pain, possible reflex changes
Complex regional pain types I and II	Burning persistent pain, allodynia, associated with immobilization/ disuse (type I) or peripheral nerve injury (type II)	Early: edema, warmth, local sweating; Late: the early signs alternate with cold, pale, cyanosis, eventually atrophic changes
Postherpetic neuralgia	Allodynia, shooting, lancinating pain	Sensory changes in the involved dermatome
Painful diabetic neuropathy	Symmetric numbness and burning or stabbing pain in lower extremities; allodynia may occur	Sensory loss in lower extremities
Phantom limb pain	Variable: aching, cramping, burning, squeezing or tearing sensation	May have peri-incisional sensory loss

Key: ROM = range of motion, RSD = reflex sympathetic dystrophy.

**TABLE 8-1** Signs and Symptoms of Selected Chronic Pain Syndromes

Complex regional pain type I, also known as *reflex sympathetic dystrophy*, and complex regional pain type II, also known as *causalgia*, may be seen in the emergency department (ED) 2 weeks or more after an acute injury. These disorders should be suspected when a patient presents with classic symptoms: allodynia (pain provoked with gentle touch of the skin) and a persistent burning or shooting pain. Associated signs early in the course of the disease include edema, warmth, and localized sweating.

## DIAGNOSIS AND DIFFERENTIAL

The most important task of the emergency physician is to distinguish chronic pain from acute pain that heralds a life- or limb-threatening condition. A complete history and physical examination should confirm the chronic condition or point to the need for further evaluation when unexpected signs or symptoms are elicited.

Rarely is a provisional diagnosis of a chronic pain condition made for the first time in the ED. The exception is a form of post nerve injury pain, complex regional pain. The sharp pain from acute injuries, including fractures, rarely continues beyond 2 weeks' duration. Pain in an injured body part beyond this period should alert the clinician to the possibility of nerve injury.

Definitive diagnostic testing of chronic pain conditions is difficult, requires expert opinion, and, often, expensive procedures such as magnetic resonance imaging, computed tomography, or thermography. Therefore, referral to the primary source of care and eventual specialist referral are warranted to confirm the diagnosis.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. There are two essential points that affect the use of opioids in the ED: (*a*) opioids should be used only in chronic pain if they enhance function at home and at work, and (*b*) a single practitioner should be the sole prescriber of opioids or be aware of their administration by others. A previous narcotic addiction is a relative contraindication to the use of opioids in chronic pain.
2. The evidence based management of chronic pain conditions is listed in [Table 8-2](#). The need for longstanding treatment of chronic pain conditions limits the safety of the nonsteroidal anti-inflammatory drugs. Doses of drugs are as follows. **Nortriptyline**, starting at 25 milligrams/day PO, or **amitriptyline**, starting at 25 milligrams/day PO. **Gabapentin** is started with an initial dose of 300 milligrams daily and is increased up to a maximum of 1200 milligrams 3 times daily according to response.

**Pregabalin**, start 50 milligrams three times daily. **Cyclobenzaprine**, begin with 10 milligrams up to 3 times daily. **Tramadol**, start 50 to 100 milligrams every 4 to 6 hours as needed. **Duloxetine**, start 30 milligrams PO daily.

Disorder	Primary ED treatment	Secondary treatment
Myofascial headache	Phenothiazines IV (acute only)	Cyclic antidepressants
Transformed migraine	Cyclic antidepressants	Stop prior medications
Fibromyalgia	Cyclobenzaprine, tramadol	Amitriptyline, pregabalin
Chronic back or neck pain	Cyclic antidepressants	NSAIDs
Myofascial back pain syndrome	NSAIDs, stay active	Cyclic antidepressants
Sciatica, (neurogenic back pain)	Acute: tapered prednisolone or prednisone	NSAIDs, muscle relaxants
Complex regional pain types I and II	Acute: Prednisone taper	Calcitonin (type I), by specialist.
Postherpetic neuralgia	Tricyclic antidepressants Gabapentin	Tramadol, opioids
Painful diabetic neuropathy	Tricyclic antidepressants Gabapentin	Pregabalin, Tramadol, Duloxetine
Phantom limb pain	Gabapentin	Tramadol, opioids

Key: ED = emergency department, NSAIDs = nonsteroidal anti-inflammatory drugs, RSD = reflex sympathetic dystrophy.

**TABLE 8-2** Management of Selected Chronic Pain Syndromes

- Referral to the appropriate specialist is one of the most productive means to aid in the care of chronic pain patients who present to the ED. Possible outcomes of referral to pain specialists include optimization of medical therapy, trigger point injections, dedicated exercise programs, physical therapy, epidural steroid injections, or nerve blocks as indicated.

## MANAGEMENT OF PATIENTS WITH DRUG-SEEKING BEHAVIOR

The spectrum of drug-seeking patients includes those who have chronic pain and have been advised to avoid taking narcotics, drug addicts who are trying to supplement their habit, and “hustlers” who are obtaining prescription drugs to sell on the street.

# CLINICAL FEATURES

Because of the spectrum of drug-seeking patients, the history given may be factual or fraudulent. Drug seekers may be demanding, intimidating, or flattering. In one ED study, the most common complaints of patients seeking drugs were (in decreasing order) back pain, headache, extremity pain, and dental pain. Many fraudulent techniques are used, including “lost” prescriptions, carrying abnormal x-rays or doctor’s note explaining need for opioids, “impending” surgery, fictitious hematuria with a complaint of kidney stones, self-mutilation, and fictitious injury.

# DIAGNOSIS AND DIFFERENTIAL

The diagnosis of drug-seeking behavior may not be possible in the ED. The medical record can provide a wealth of information regarding the patient, including documentation proving that the patient is supplying false information. Most states now have controlled drug databases that can be queried. Behaviors predictive of drug seeking are listed in [Table 8-3](#). The predictive behaviors are illegal in many states and form a solid basis to refuse narcotics to the patient.

Behaviors predictive of drug-seeking behavior
Sells prescription drugs*
Forges/alters prescriptions*
Fictitious illness, requests opioids*
Uses aliases to receive opioids*
Conceals multiple ED visits for opioids*
Conceals multiple physicians prescribing opioids*
Current illicit drug addiction

\*Behaviors in this category are unlawful in many states.

**TABLE 8-3** Characteristics of Drug Seeking Behavior

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

The treatment of drug-seeking behavior is to refuse the controlled substance, consider the need for alternative medication or treatment, and consider referral for drug counseling.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 42, “Adults with Chronic Pain,” by David M. Cline.

## **SECTION 3 Emergency Wound Management**



## **CHAPTER 9**

# **Evaluating and Preparing Wounds**

**Timothy Reeder**

## **CLINICAL FEATURES**

Traumatic wounds are regularly encountered in the emergency department. It is important to document important historical information such as the mechanism, timing, and location of injury, and the degree of contamination. Associated symptoms of pain, swelling, paresthesias, and loss of function should be identified. Ascertain factors that affect wound healing, such as the patient's age, location of injury, medications, chronic medical conditions (eg, diabetes, chronic renal failure, or immunosuppression), and previous scar formation (keloid). Patients with the sensation of a foreign body are much more likely to have retained a foreign body. Patient characteristics of handedness, occupation, tetanus status, and allergies (eg, to analgesics, anesthetics, antibiotics, or latex) should be documented. When caring for wounds, the ultimate goal is to restore the physical integrity and function of the injured tissue without infection.

When treating a wound, consider the time, mechanism of injury, and its location because these factors play a role in the potential for infection. Shear, compressive, or tensile forces cause acute traumatic wounds. Shear forces are produced by sharp objects with relatively low energy, resulting in a wound with a straight edge and little contamination that can be expected to heal with a good result. Wounds caused by compression forces crush the skin against underlying bone. These high-energy forces produce stellate lacerations. Tension forces produce flap-type lacerations. These wounds typically have surrounding devitalized tissue and result in a wound much more susceptible to infection than those caused by shear forces.

Assessment of a wound's potential for infection is vital. Predictive factors for infection include location, depth, characteristics, contamination,

and patient age. The risk of infection relates to the interaction of bacterial contamination, time to wound closure, and blood supply. The density of bacteria is quite low over the trunk and proximal arms and legs. Moist areas such as the axilla, perineum, and exposed hands and feet have a higher degree of colonization.

Wounds of the oral cavity are heavily contaminated with facultative and anaerobic organisms. Wounds sustained from contaminated objects or environments and animal and human bites have an increased infection risk. Wounds contaminated with feces have a high risk of infection despite determined therapy. Over the first 24 hours, the longer the time from injury to wound closure, the greater the risk of infection. Wounds in highly vascular areas such as the face and scalp are less likely to become infected.

## **DIAGNOSIS AND DIFFERENTIAL**

Wound examination is greatly facilitated by a cooperative patient, good positioning, optimal lighting, and little or no bleeding. Universal precautions should be used during evaluation. A thorough and compulsory examination will minimize the risk of missed foreign bodies, tendon, and nerve injuries, a common cause of litigation.

Documentation of a wound should include the location, size, shape, margins, and depth. Pay particular attention to sensory, motor, tendon, vascular compromise, and injuries to specialized structures. Blood pressure differences between injured and noninjured extremities will help identify significant arterial injuries. Careful palpation and inspection of the wound and surrounding area may show the presence of a foreign body or bony injury. Consider injecting joints with overlying wounds to identify violation of the joint space.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Proper wound preparation is the most important step for adequate evaluation of the wound, to restore the integrity and function of the injured tissue, prevent infection, and maximize cosmetic results.

## **STERILE TECHNIQUE**

Full sterile technique has not been shown to reduce infection after repair. Clean, nonsterile gloves and attention to cleanliness may be used to improve efficiency and cost savings.

## **ANESTHESIA**

1. Control pain with local or regional anesthesia before any wound manipulation (see [Chapter 7](#)). This will enable better preparation and evaluation of the wound and a more relaxed, cooperative patient.
2. Perform a careful neurovascular examination of the involved and distal area before anesthesia. Two-point discrimination (<6mm) will help identify digital nerve injury.

## **HEMOSTASIS**

1. Control of bleeding is necessary for proper wound evaluation and treatment.
2. Direct pressure is the preferred method and is usually effective.
3. Epinephrine containing local anesthetics are safe for digital nerve blocks, nose, and ears except in patients with underlying small vessel disease.
4. Ligation of minor vessels in the extremity may be necessary and can be achieved by applying an absorbable suture material after isolating and clamping the involved vessel.
5. Other means of hemostasis can be used, such as applying pressure with gelatin, cellulose, or collagen sponges placed directly into the wound (eg,

Gelfoam<sup>®</sup>, Oxycel<sup>®</sup>, Actifoam<sup>®</sup>).

6. Cautery, with a bipolar device for vessels <2 mm and battery powered device for capillaries, can be used to control extensive bleeding.
7. Finger tourniquets or pressure cuff proximal to the injury and inflated above the systolic blood pressure will control bleeding. Their duration should be minimized.

## **FOREIGN BODY AND HAIR REMOVAL**

1. Visual inspect the full depth and course of all wounds for foreign bodies (see [Chapter 14](#)).
2. Remove hair, which can act as a foreign body, by clipping 1 to 2 mm above the skin with scissors. Shaving may damage the hair follicles, allowing bacterial invasion, and can increase the infection rate by 10 fold. Ointment can be applied to the hair roots to keep hair away from wound edges.
3. Do not remove hair from the eyebrows due to the potential for abnormal or lack of regrowth.
4. Most foreign bodies and glass shards  $\geq 2\text{mm}$  will be detected by routine radiographs. Foreign bodies with densities similar to those of soft tissue may require the use of computed tomography, magnetic resonance imaging, or ultrasound (see [Chapter 14](#)).

## **IRRIGATION**

1. Wound irrigation reduces the risk of infection.
2. Use low pressure irrigation (0.5 psi) with a slow, gentle wash for uncontaminated wounds and loose tissues.
3. Use high pressure irrigation ( $\geq 7$  psi) with an 18 gauge catheter and syringe for contaminated wounds.
4. Irrigate contaminated wounds until all visible debris is removed.
5. Consider anesthesia before irrigation.

6. Wound soaking is not effective in cleaning contaminated wounds and may increase wound bacterial counts.
7. Although sterile normal saline solution has the lowest toxicity, tap water is safe and effective. There is no added benefit to the addition of povidone iodine or hydrogen peroxide to irrigation fluid.

## **DEBRIDEMENT**

1. Devitalized tissue may increase the risk of infection and delay healing. Debridement removes foreign matter, bacteria, and devitalized tissue and creates a sharp wound edge that is easier to repair.
2. Elliptical excision around the wound edges with a standard surgical blade is the most effective type of debridement. Tissue that has a narrow base or lacks capillary refill will require debridement.
3. Wounds with an extensive amount of nonviable tissue may require a large amount of tissue removal and will need more delayed wound closure or grafting. In general, a surgical specialist should be consulted to manage these wounds.

## **ANTIBIOTICS PRIOR TO MANIPULATION**

Although there is no clear evidence that antibiotic prophylaxis prevents wound infection in most emergency department patients, there may be a role in selected high-risk wounds and populations.

1. When used, antibiotic prophylaxis should be (*a*) started rapidly, before significant tissue manipulation; (*b*) performed with agents that are effective against predicted pathogens; and (*c*) administered by routes that rapidly achieve desired blood levels. Oral antibiotics may be as effective as intravenous if the agent has sufficient spectrum of coverage and rapid absorption.
2. Most non-bite infections are caused by staphylococci or streptococci and coverage with a  $\beta$ -lactam is reasonable. Use a first generation

cephalosporin cephalexin 25 to 50 milligrams/kilogram/day PO 4 times per day in children; 500 milligrams PO four times per day in adults. For  $\beta$ -lactam allergic patients, use clindamycin 8 to 25 milligrams/kilogram/day 3 times per day in children or 150 to 450 four times per day for adults.

3. Treat patients with human and mammalian bites with amoxicillin/clavulanate 25 to 50 milligrams/kilogram/day twice per day in children, 875 milligrams PO twice per day in adults for *Pasteurella*, *Eikenella*, or *Capnocytophaga* (see [Chapter 15](#)).
4. Effectiveness of antibiotics for oral lacerations is inconclusive. If you chose to cover with antibiotics, use penicillin 25 to 50 milligrams/kilogram/day PO divided over 3 doses per day; 500 milligrams PO 3 times daily in adults.
5. Prescribe ciprofloxacin 500 PO twice each day in adults with wounds contaminated by fresh water and plantar puncture wounds to cover for *Pseudomonas*.
6. Duration of prophylactic antibiotics is 3 to 5 days for non-bite wounds and 5 to 7 days for bite wounds.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 43, "Evaluation of Wounds," by Judd Hollander and Adam Singer; and Chapter 44, "Wound Preparation," by Shoma Desai, Susan C. Stone and Wallace A. Carter.

# **CHAPTER 10**

## **Methods for Wound Closure**

**David M. Cline**

Wounds can be closed primarily in the emergency department (ED) by the placement of sutures, surgical staples, skin closure tapes, and adhesives. All wounds heal with some scarring; however, preferred closure techniques make scars less noticeable. It is important to match each layer of a wound edge to its counterpart. Care must be taken to avoid having one wound edge roll inward. The rolled-in edge promotes wound infection as well as misaligns the dermis and epidermis causing wound dehiscence and increased scarring.

### **SUTURES**

Sutures are the strongest, most reliable and adaptable of all wound closure devices allowing the most accurate approximation of wound edges. Sutures are classified as nonabsorbable and absorbable. The latter lose all their tensile strength within 60 days. Monofilament synthetic sutures such as nylon or polypropylene have the lowest rates of infection and are the most commonly used suture material in the ED. Synthetic monofilament absorbable sutures (eg, Monocryl<sup>®</sup>) are preferred for closure of deep structures such as the dermis or fascia because of their strength and low tissue reactivity. Rapidly absorbing sutures (eg, Vicryl Rapide<sup>®</sup>) can be used to close the superficial skin layers or mucous membranes, especially when the avoidance of removal is desired.

Sutures are sized according to their diameter. For general ED use, 6-0 suture is the smallest and is used for percutaneous closure on the face and other cosmetically important areas. Suture sizes 5-0 and 4-0 are progressively larger; 5-0 is commonly used for closure of hand and finger lacerations, and 4-0 is used to close lacerations on the trunk and proximal

extremities. Very thick skin, as is found on the scalp and sole, may require closure with 3-0 sutures.

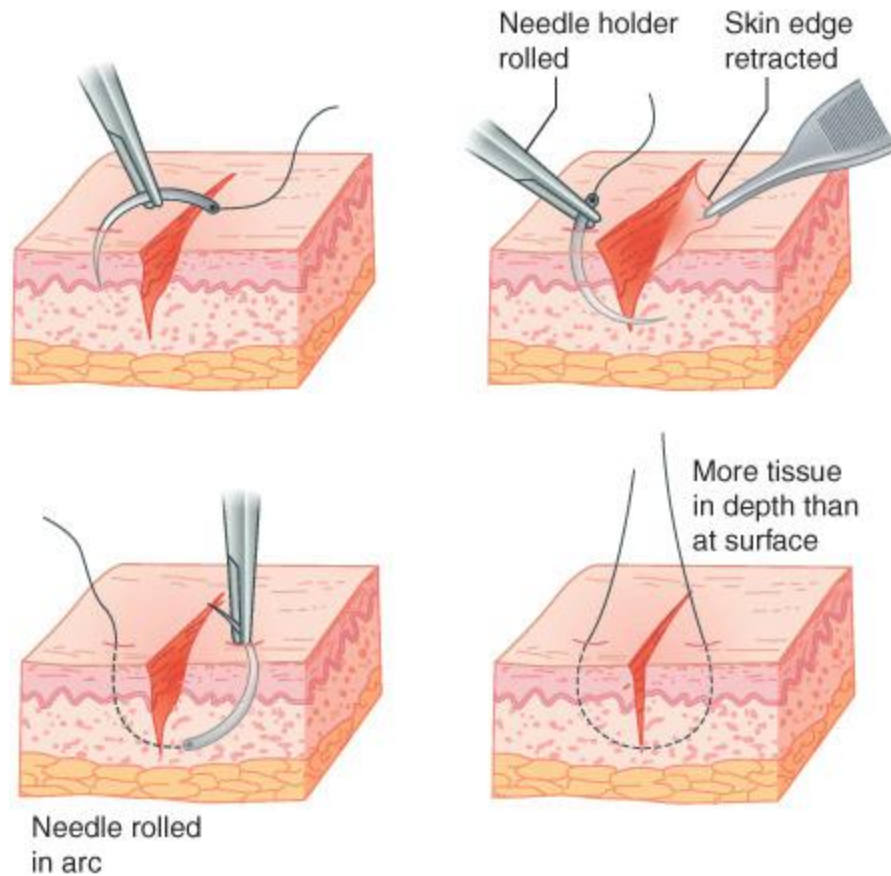
## **SUTURING TECHNIQUES**

Percutaneous sutures that pass through the epidermal and dermal layers are the most common sutures used in the ED. Dermal or subcuticular sutures reapproximate the divided edges of the dermis without piercing the epidermis. These two sutures may be used together in a layered closure as wound complexity demands. Sutures can be applied in a continuous fashion (“running” sutures) or as interrupted sutures.

### **Simple Interrupted Percutaneous Sutures**

Place percutaneous sutures to achieve eversion of the wound edges. The needle should enter the skin at a 90° angle and exit the opposite side at 90°. The depth of the suture should be wider than the width. Sutures placed in this manner will encompass a portion of tissue that will evert when the knot is tied ([Fig. 10-1](#)). Place an adequate number of interrupted sutures to close wound edges without gaping. In general, the number of ties should correspond to the suture size (ie, 4 ties for 4-0 suture and 5 ties for 5-0 suture).





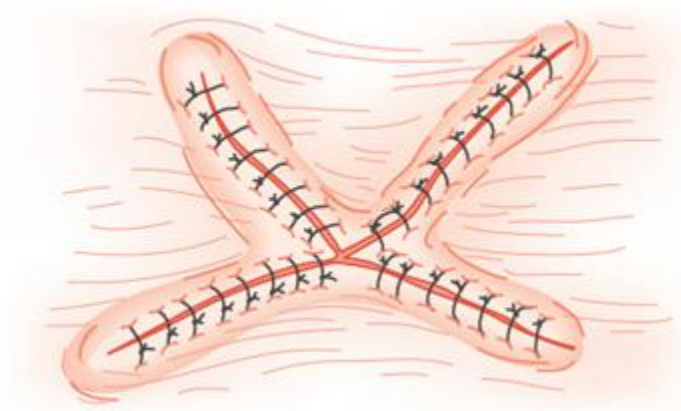
**FIGURE 10-1.** Placement of simple interrupted sutures. The suture path should gather more tissue at its base than at its surface. Therefore, the suture will evert its skin edges when tightened.

Straight, shallow lacerations must be closed with percutaneous sutures only, by sewing from one end toward the other and aligning edges with each suture bite. Deep, irregular wounds with uneven, unaligned, or gaping edges are more difficult to suture. Certain principles have been identified for these more difficult wounds:

1. Wounds in which the edges cannot be brought together without excessive tension should have dermal sutures placed to partly close the gap.
2. When suturing wound edges of different thicknesses, pass the needle through one side of the wound and then draw it out before reentry through the other side to ensure that the needle is inserted at a comparable level.

3. Align uneven edges by approximating the midportion of the wound with the first suture. Place subsequent sutures in the middle of each half, until the wound edges are aligned and closed.

Simple interrupted sutures are the most versatile and effective for realigning irregular wound edges and stellate lacerations ([Fig. 10-2](#)). An advantage of interrupted sutures is that only the involved sutures need to be removed in the case of wound infection.

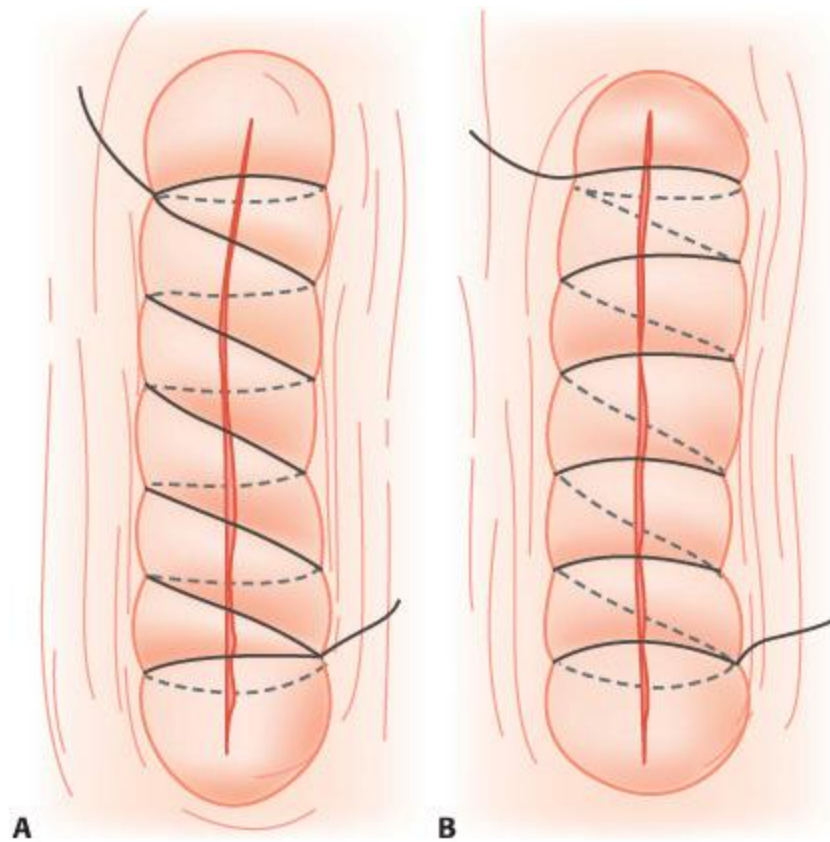


**FIGURE 10-2.** Stellate laceration closed with interrupted sutures.

### **Continuous “Running” Percutaneous Sutures**

Continuous “running” percutaneous sutures can be used when repairing linear wounds. An advantage of the continuous suture is that it accommodates to the developing edema of the wound edges during healing. However, a break in the suture may ruin the entire repair and may cause permanent marks if placed too tightly. Continuous suture closure of a laceration can be accomplished by 2 different patterns. In the first pattern, the needle pathway is at a 90° angle to the wound edges and results in a visible suture that crosses the wound edges at a 45° angle ([Fig. 10-3A](#)). In the other pattern, the needle pathway is at a 45° angle to the wound edges, so that the visible suture is at a 90° angle to the wound edges ([Fig. 10-3 B](#)). In

either case, the physician starts at the corner of the wound farthest away and sutures toward himself or herself.

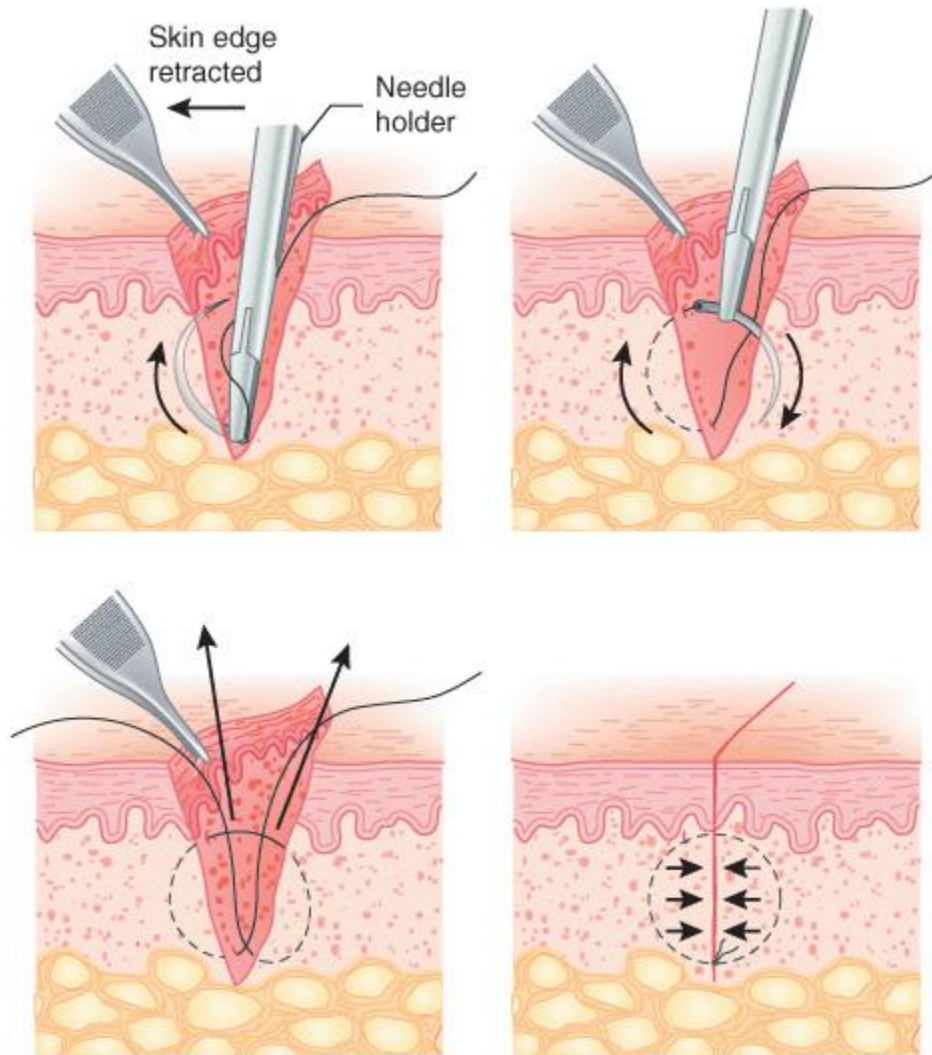


**FIGURE 10-3.** A. Running suture crossing wound at 45°. B. Running suture crossing wound at 90.

## Deep Dermal Sutures

The major role of these sutures is to reduce tension. They are also used to close dead spaces. However, their presence increases the risk of infection in contaminated wounds. Sutures through adipose tissues do not hold tension, increase infection rates, and should be avoided. With deep dermal sutures, the curved needle is inserted at the depth of the dermis and directed upward, exiting within the wound, just beneath the dermal-epidermal junction ([Fig. 10-4](#)). Then the needle is inserted across the wound and directed in a curve

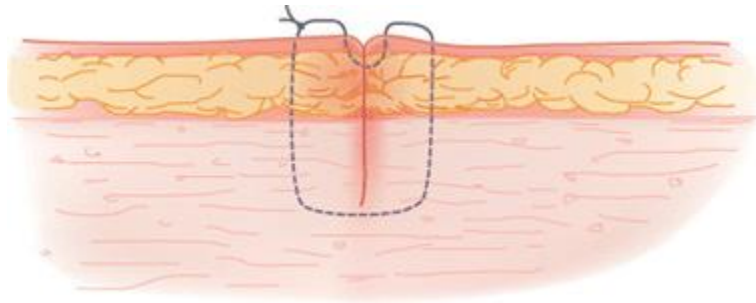
downward, exiting at the wound base. The suture is tied deep in the wound and will become buried in the depth of the tissue. The first suture is placed at the center of the laceration, and additional sutures sequentially bisect the wound. The number of deep sutures should be minimized.



**FIGURE 10-4.** Placement of deep dermal suture. The needle is inserted at the depth of the dermis and directed upward, exiting beneath the dermal-epidermal junction. Then the needle is inserted across the wound and directed downward, exiting at the wound base. The suture knot is then placed deep in the wound.

## Vertical Mattress Sutures

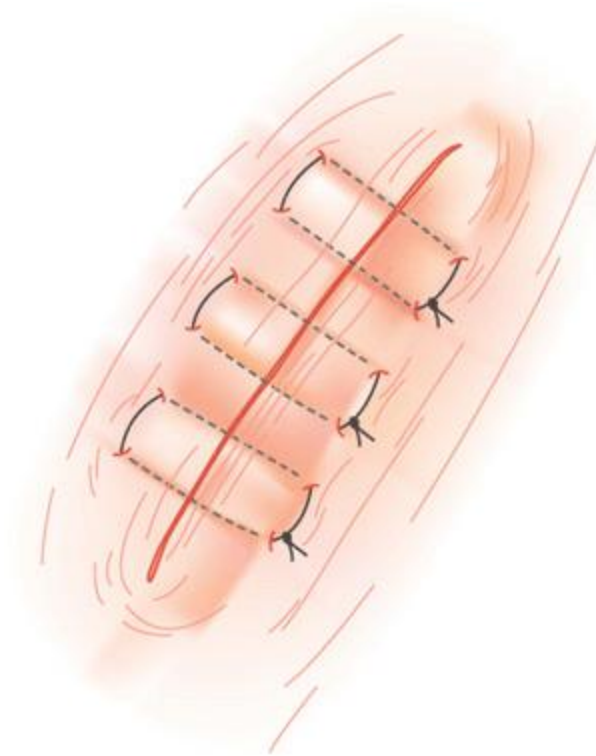
Vertical mattress sutures ([Fig. 10-5](#)) are useful in areas of lax skin (elbow and dorsum of the hand) where the wound edges tend to fold into the wound. It can act as an “all-in-one” suture, thus avoiding the need for a layered closure in gaping lacerations.



**FIGURE 10-5.** Vertical mattress suture.

## Horizontal Mattress Sutures

Horizontal mattress sutures are faster and accomplish eversion better than vertical mattress sutures. These are especially useful in areas of increased tension such as the fascia, joints, and callus skin ([Fig. 10-6](#)). To avoid tissue strangulation, care must be taken not to tie the individual sutures too tightly.



**FIGURE 10-6.** Horizontal mattress suture.

## **Delayed Closure**

Delayed primary closure is an option for contaminated wounds or for wounds presenting beyond 12 hours from injury. The wound is left open for 3 to 5 days, after which it may be closed if no infection supervenes.

## **STAPLES**

Skin closure by metal staples is quick and economical, with the advantage of low tissue reactivity. The skin staple should be reserved for lacerations in areas where the healing scar is not readily apparent (eg, scalp). When placing staples, use tissue forceps to slightly elevate and hold the wound edges together. Place the device gently against the skin, and squeeze the

trigger slowly. A properly placed staple should have its topside off the skin surface.

## **ADHESIVE TAPES**

Adhesive tapes are the least reactive of all wound closure devices. Skin closure tapes are used as an alternative to sutures and staples and for additional support after suture and staple removal. Tapes work best on flat, dry, nonmobile surfaces where the wound edges fit together without tension. Taped wounds are more resistant than sutured wounds to infection. They can be used for skin flaps, where sutures may compromise perfusion, and for lacerations with thin, friable skin that will not hold sutures. Application of benzoin to the skin surface 2 to 3 cm beyond the wound edges will enhance adherence. Maintain some space between individual tapes. The tapes will spontaneously detach as the underlying epithelium exfoliates.

## **CYANOACRYLATE TISSUE ADHESIVES**

Cyanoacrylate tissues adhesives close wounds by forming an adhesive layer on top of intact epithelium. Adhesives are most useful when they are used on wounds that close spontaneously, have clean or sharp edges, and are located on clean, nonmobile areas. Do not apply adhesives within wounds, to mucous membranes, infected areas, joints, areas with dense hair (eg, scalp), or on wounds exposed to body fluids. Wound closure with adhesives is faster and less painful than suturing and has comparable rates of infection, dehiscence, and cosmetic appearance.

Wounds with edges separated by more than 5 mm are unlikely to stay closed with tissue adhesives alone. Subcutaneous sutures can be inserted to relieve this tension. Lacerations longer than 5 cm are unlikely to remain closed with tissue adhesives alone.

The adhesive is carefully expressed through the tip of the applicator and gently brushed over the wound surface in a continuous steady motion. The

adhesive should cover the entire wound in addition to an area covering 5 to 10 mm on either side of the wound edges. After allowing the first layer of the adhesive to polymerize for 30 to 45 seconds, 2 to 3 additional layers of the adhesive are similarly brushed onto the surface of the wound, with pauses of 5 to 10 seconds between successive layers. Take care to position the patient parallel to the floor, cover the eyes, and use gentle squeezing of the applicator to avoid problematic runoff.

Once applied, Cyanoacrylate should not be covered with ointment, bandage, or dressing. Instruct patients not to pick at edges of the adhesive. The area can be gently washed with plain water after 24 hours but should not be scrubbed, soaked, or exposed to moisture for any length of time. The adhesive will spontaneously slough off in 5 to 10 days. Should a wound open, the patient should return immediately for closure.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 45, “Methods for Wound Closure,” by Adam J. Singer and Judd E. Hollander.



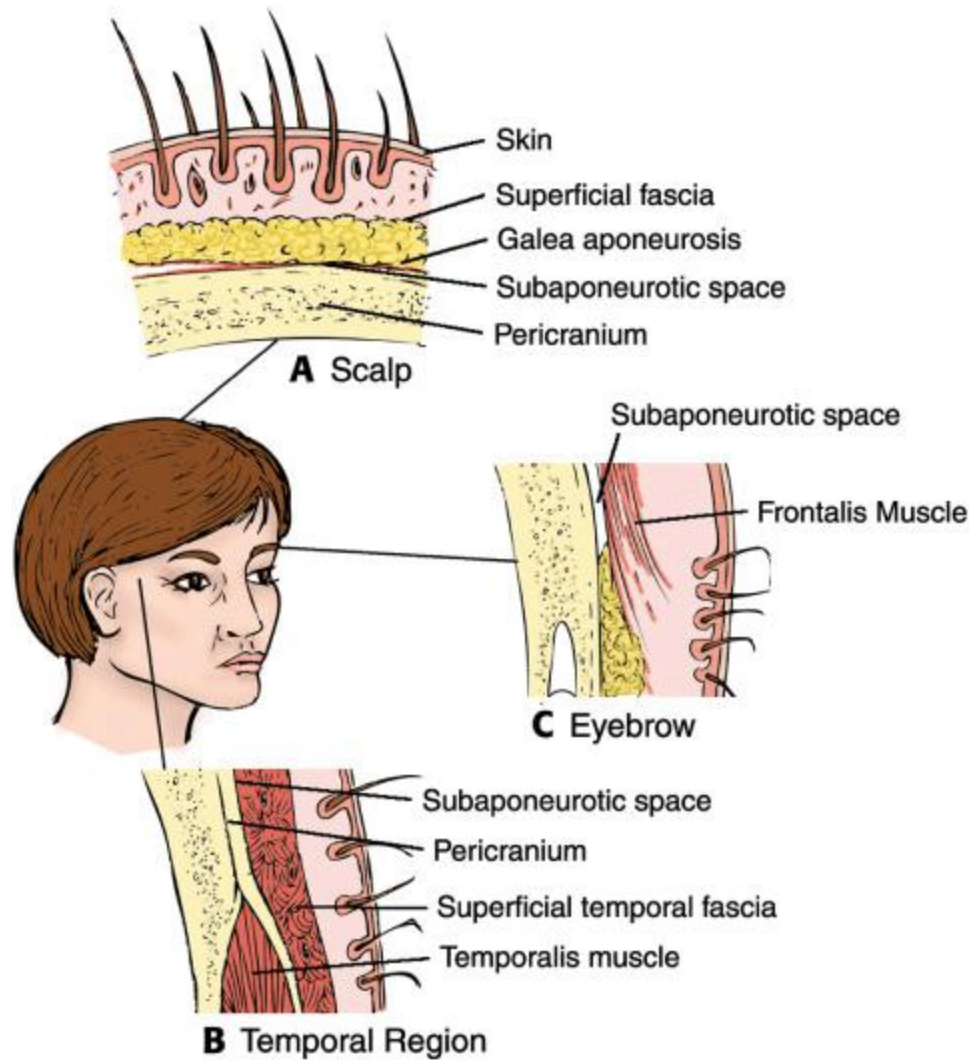
# **CHAPTER 11**

## **Lacerations to the Face and Scalp**

J. Hayes Calvert

### **SCALP AND FOREHEAD**

The scalp and forehead (which includes eyebrows) are parts of the same anatomic structure ([Fig. 11-1](#)). Eyebrows are valuable landmarks for the meticulous reapproximation of the wound edges and should never be clipped or shaved. After the wound cleaning and hemostasis are achieved, the base of the wound always should be palpated for possible skull fracture. All depressed fractures should be evaluated by computed tomography.



**FIGURE 11-1.** The layers of the **A.** scalp, **B.** temporal region, and **C.** eyebrow.

*Key* : TM = temporalis muscle.

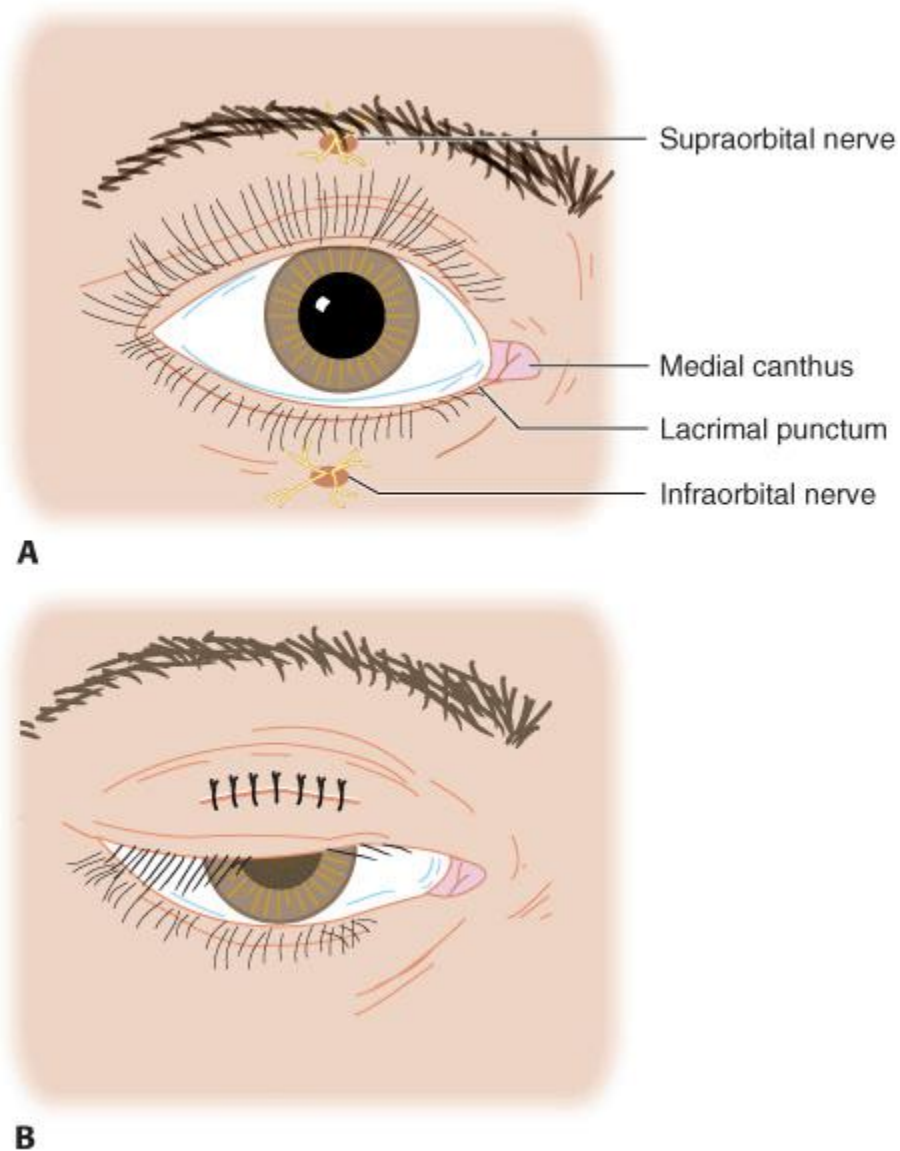
When the edges of a laceration of the eyebrow or the scalp are devitalized, debridement is mandatory. Debride at an angle that is parallel to that of the hair follicles, to prevent subsequent alopecia. Occasionally direct pressure or vessel clamping may be needed to control hemorrhage at the wound edges. Begin wound closure with approximation of the galea aponeurotica using buried, interrupted absorbable 4-0 sutures. Close the divided edges of muscle and fascia with buried, interrupted, absorbable 4-0 synthetic sutures to prevent further development of depressed scars. Close

the skin with staples or interrupted nylon sutures (sutures of a color different from the patient's hair should be considered).

Approximate the skin edges of anatomic landmarks on the forehead first with key stitches by using interrupted, nonabsorbable monofilament 5-0 synthetic sutures. Accurate alignment of the eyebrow, transverse wrinkles of the forehead, and the hairline of the scalp is essential. It may be necessary to have younger patients raise their eyebrows to create wrinkles for accurate placement of the key stitches. A firm pressure dressing placed around the head can close any potential dead space, encourage hemostasis, and prevent hematoma formation. Leave the pressure dressing in place for 24 hours. Scalp sutures and staples can be removed in 7 to 10 days, whereas facial sutures should be removed in 5 days.

## **EYELIDS**

A complete examination of the eye structure and function is essential, including an evaluation for foreign bodies (see [Chapter 149](#)). Examine the lid for involvement of the canthi, the lacrimal system, the supraorbital nerve, and the infraorbital nerve or penetration through the tarsal plate or lid margin ([Fig. 11-2](#)). The following wounds should be referred to an ophthalmologist: (*a*) those involving the inner surface of the lid, (*b*) those involving the lid margins, (*c*) those involving the lacrimal duct, (*d*) those associated with ptosis, and (*e*) those extending into the tarsal plate. Failure to recognize and properly repair the lacrimal system can result in chronic tearing.



**FIGURE 11-2.** External landmarks.

Uncomplicated lid lacerations can be readily closed by using nonabsorbable 6-0 sutures, with removal in 3 to 5 days. Do not use tissue adhesive near the eye.

## **NOSE**

Lacerations of the nose may be limited to skin or involve the deeper structures (sparse nasal musculature, cartilaginous framework, and nasal mucous membrane). Each tissue layer must be accurately approximated. Inexperienced operators should refer such cases to an otolaryngologist or a plastic surgeon. Local anesthesia of the nose can be difficult because of the tightly adhering skin. Topical anesthesia may be successful with lidocaine.

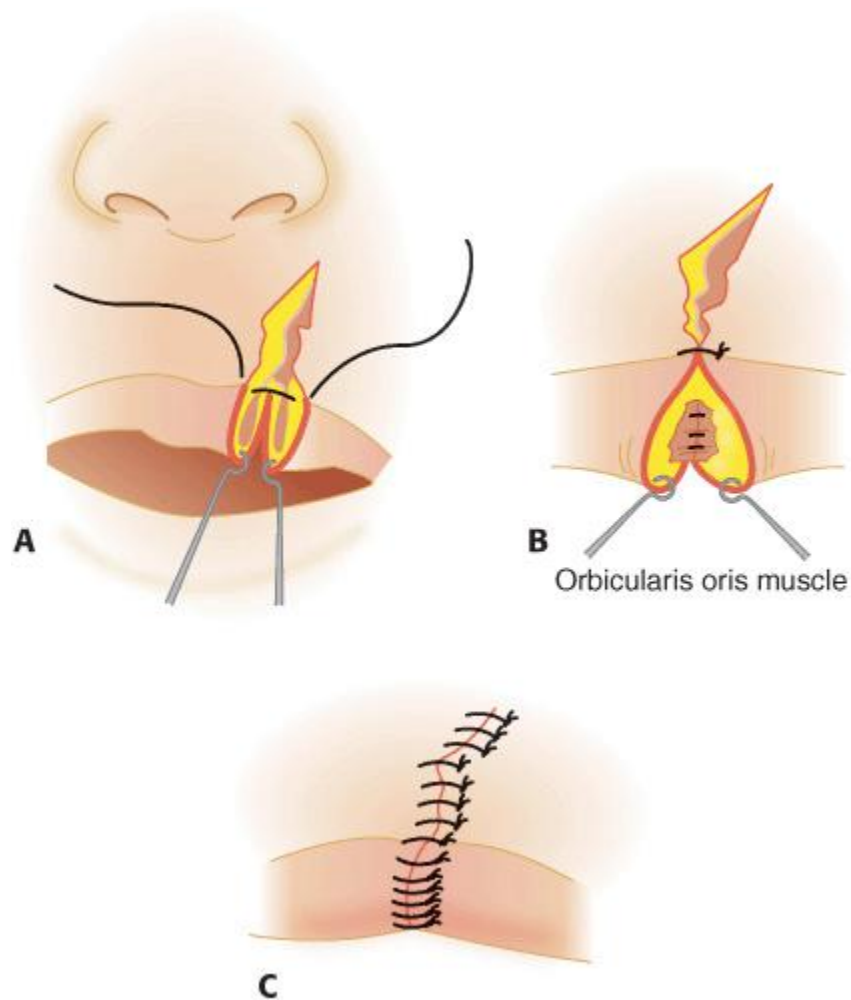
When the laceration extends through all tissue layers, begin closure with a nonabsorbable, monofilament 5-0 synthetic suture that aligns the skin surrounding the entrances of the nasal canals to prevent malposition and notching of the alar rim. Traction on the long, untied ends of this suture approximates the wounds and aligns the anterior and posterior margins of the divided tissue layers. Repair the mucous membrane with interrupted, braided, absorbable 5-0 synthetic sutures, burying the knots in the tissue. Re-irrigate the area gently from the outside. Rarely, the cartilage may need to be approximated with a minimal number of 5-0 absorbable sutures. In sharply marked linear lacerations, closure of the overlying skin is usually sufficient. Close the cut edges of the skin, with its adherent musculature, using interrupted, nonabsorbable, monofilament 6-0 synthetic sutures. Remove external sutures in 3 to 5 days.

Inspect the septum for hematoma formation with a nasal speculum. The presence of bluish swelling in the septum confirms the diagnosis of septal hematoma. Treatment of the hematoma is evacuation of the blood clot. Drainage of a small hematoma can be accomplished by aspiration of the blood clot through an 18 gauge needle. A larger hematoma should be drained through a horizontal incision at the base. Bilateral hematomas should be drained in the operating room by a specialist. Reaccumulation of blood can be prevented by nasal packing. Antibiotic treatment is recommended to prevent infection that may cause necrosis of cartilage. Use an oral penicillin, cephalosporin, or macrolide (in penicillin-allergic patients).

## **LIPS**

The technique of closure will depend largely on the type of lip wound. Isolated intraoral lesions may not need to be sutured. Through and through lacerations that do not include the vermilion border can be closed in layers. Begin repair with 5-0 absorbable suture for the mucosal surface, reirrigate and then close the orbicularis oris muscle with 4-0 or 5-0 absorbable suture. Close skin with 6-0 nonabsorbable suture or tissue adhesive. Remove sutures in 5 days.

Begin closure of a complicated lip laceration at the junction between the vermilion and the skin with a nonabsorbable, monofilament 6-0 synthetic suture ([Fig. 11-3](#)). The orbicularis oris muscle is then repaired with interrupted 4-0 or 5-0 absorbable sutures. Approximate the junction between the vermilion and the mucous membrane with a braided, absorbable 5-0 synthetic suture. Close the divided edges of the mucous membrane and vermilion with interrupted absorbable 5-0 synthetic sutures in a buried knot construction. Skin edges of the laceration may be jagged and irregular, but they can be fitted together as the pieces of a jigsaw puzzle by using interrupted, nonabsorbable, monofilament 6-0 synthetic sutures with their knots formed on the surface of the skin. Patients with sutured intraoral lacerations should receive prophylactic antibiotics, penicillin or clindamycin.

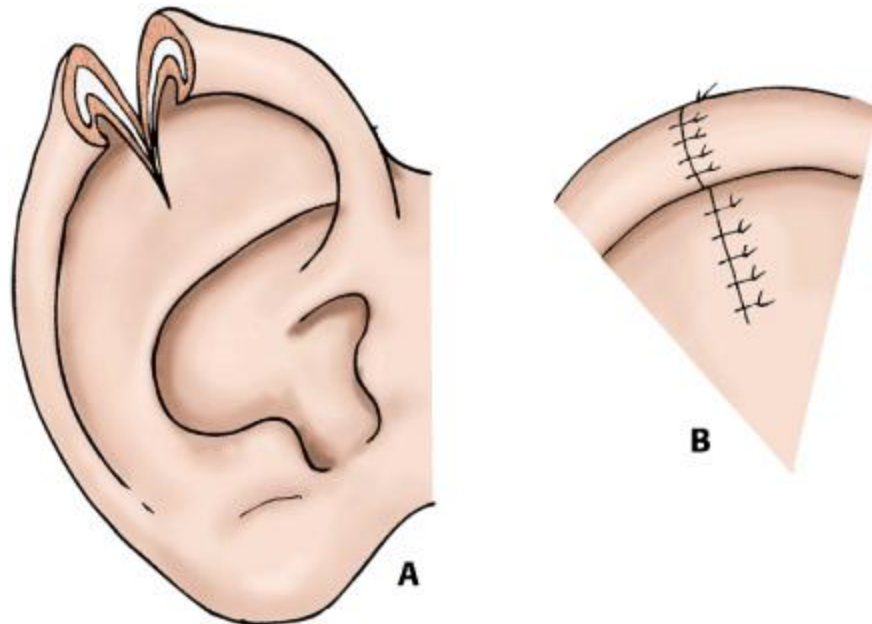


**FIGURE 11-3.** Irregular-edged vertical laceration of the upper lip. **A.** Traction is applied to the lips and closure of the wound is begun first at the vermilion-skin junction. **B.** The orbicularis oris muscle is then repaired with interrupted, absorbable 4-0 synthetic sutures. **C.** The irregular edges of the skin are then approximated.

## EAR

Close superficial lacerations of the ear with 6-0 nylon suture. Cover exposed cartilage. Debridement of the skin is not advisable because there is very little excess skin. In most through and through lacerations of the ear,

the skin can be approximated and the underlying cartilage will be supported adequately ([Fig. 11-4](#)). After repair of simple lacerations, place a small piece of nonadherent gauze over the laceration only and apply a pressure dressing. Place gauze squares behind the ear to apply pressure, and wrap the head circumferentially with gauze. Remove sutures in 5 days. Consult an otolaryngologist or plastic surgeon for more complex lacerations, ear avulsions, or auricular hematomas.

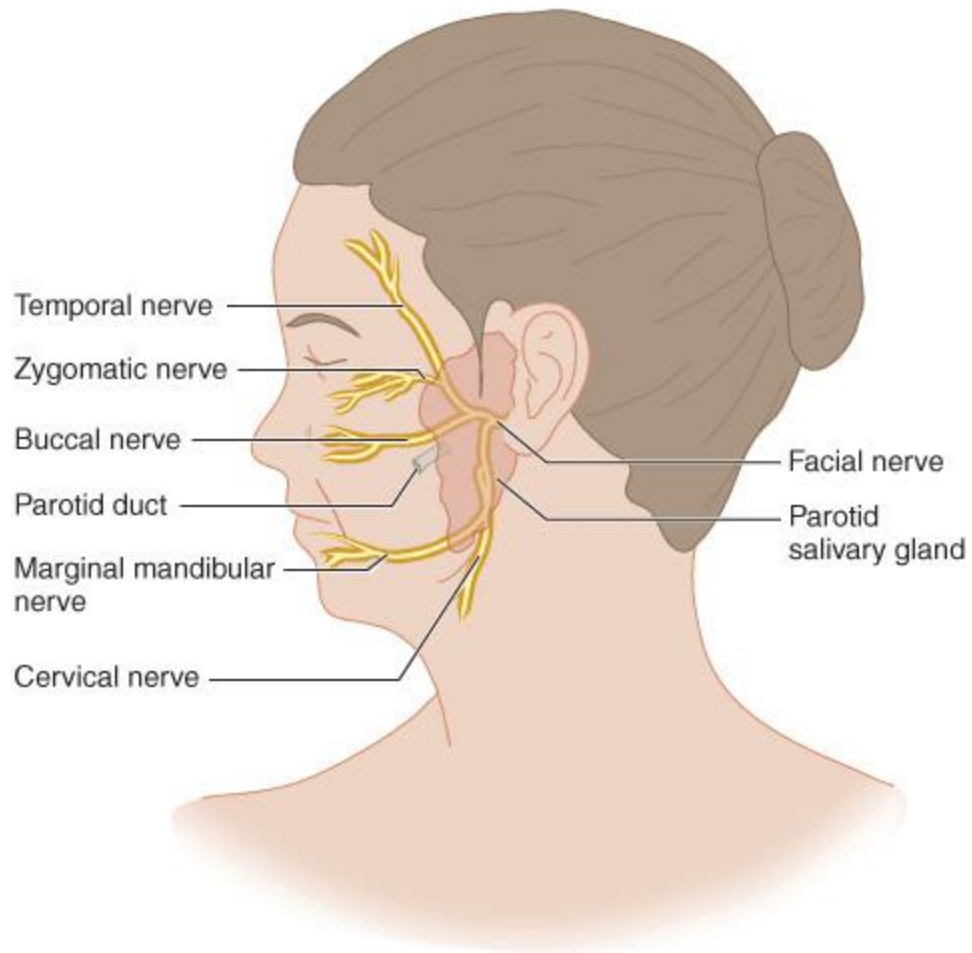


**FIGURE 11-4.** Repair of auricular laceration. **A.** Laceration through auricle. **B.** Interrupted 6-0 nonabsorbable sutures approximate the skin edges.

## CHEEKS AND FACE

In general, facial lacerations are closed with 6-0 nonabsorbable, simple interrupted sutures and are removed after 5 days. Tissue adhesive may also be used. Attention to anatomic structures including the facial nerve and parotid gland is necessary ([Fig. 11-5](#)). If these structures are involved, operative repair is indicated.





**FIGURE 11-5.** Anatomic structures of the cheek. The course of the parotid duct is deep to a line drawn from the tragus of the ear to the midportion of the upper lip. Branches of the facial nerve: temporal (**T**), zygomatic (**Z**), buccal (**B**), mental (**M**), and cervical (**C**).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 46, “Lacerations to the Face and Scalp,” by Wendy C. Coates.

# CHAPTER 12

## Injuries of the Arm, Hand, Fingertip, and Nail

David M. Cline

### DIAGNOSIS AND DIFFERENTIAL

History should include occupation and hand dominance. Examination of all arm and hand injuries includes inspection at rest, evaluation of motor, nerve and tendon functions, evaluation of sensory nerve function, and evaluation of perfusion. Examine active motion and resistance to passive motion. (See [Tables 12-1](#) and [12-2](#)) Examine all wounds for evidence of potential artery, nerve, tendon, bone injuries, and the presence of foreign bodies, debris, or bacterial contamination.

Nerve	Motor Exam
Radial	Dorsiflexion of wrist
Median	Thumb abduction away from the palm Thumb interphalangeal joint flexion
Ulnar	Adduction/abduction of digits

**TABLE 12-1** Motor Testing of the Peripheral Nerves in the Upper Extremity

Sensory Nerve	Area of Test
Radial	First dorsal web space
Median	Volar tip of index finger
Ulnar	Volar tip of little finger

**TABLE 12-2** Sensory Testing of Peripheral Nerves in the Upper Extremity

A bloodless field is needed to achieve adequate visualization. If a proximal tourniquet is needed, a Penrose drain can be used for distal finger injuries and a manual blood pressure cuff for more proximal injuries. Once

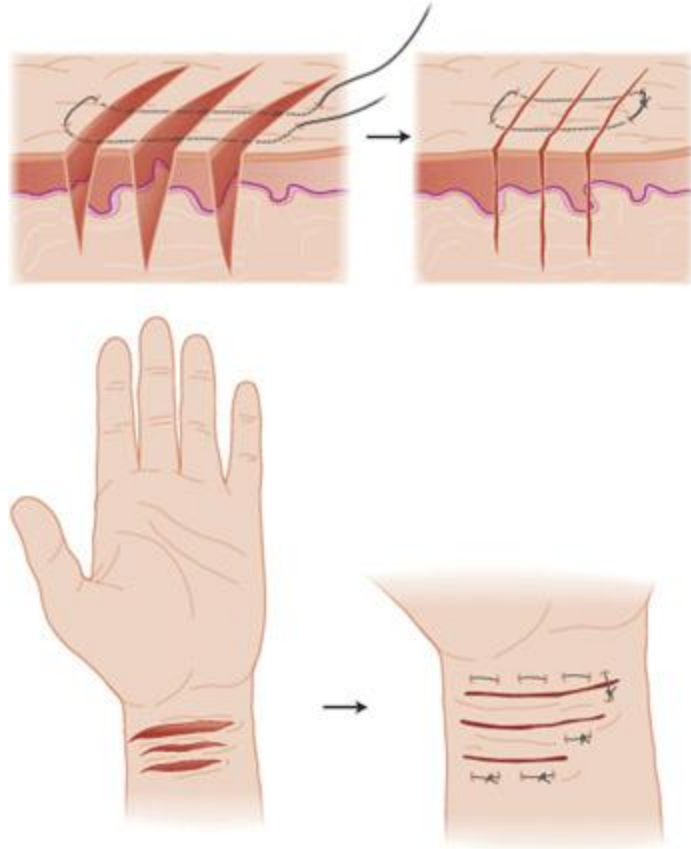
adequate visualization is obtained, examine the wound for foreign bodies and tendon and joint capsule injuries. Examine the hand and arm in the position of injury to avoid missing deep structure injuries that may have moved out of the field of view when examined in a neutral position. Obtain anteroposterior and lateral x-rays if bony injuries, retained radiopaque foreign bodies, or joint penetration are suspected.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. All wounds require scrupulous cleaning and irrigation after adequate anesthesia.
2. Provide tetanus prophylaxis as indicated (see [Chapter 16](#)).
3. Consult a plastic or hand surgeon for complex or extensive injuries, injuries requiring skin grafting, injuries requiring technically demanding skills, or when the hand is vital to patient's career (eg, a professional musician).
4. See below for additional care instructions of specific injuries.

### **Forearm and Wrist Lacerations**

1. Injury over the wrist raises the possibility of a suicide attempt. Question the patient about intent and a history of depression.
2. Injuries that involve more than 1 parallel laceration, classic for suicide attempts, may require horizontal mattress sutures to cross all lacerations to prevent compromising the vascular supply of the island of skin located between incisions ([Fig. 12-1](#)).



**FIGURE 12-1.** Horizontal mattress sutures for multiple parallel lacerations.

3. Examine tendons and distal nerves individually. (see [Tables 12-3](#) and [12-4](#))

Compartment	Muscle	Function
First compartment	Abductor pollicis longus Extensor pollicis brevis	Abducts and extends thumb Extends thumb at MCP joint
Second compartment	Extensor carpi radialis longus Extensor carpi radialis brevis	Extends and radially deviates wrist Extends and radially deviates wrist
Third compartment	Extensor pollicis longus	Extends thumb at interphalangeal joint
Fourth compartment	Extensor digitorum communis Extensor indicis proprius	Splits into four tendons at level of the wrist; extends index, long, ring, and little digits Extends index finger
Fifth compartment	Extensor digiti minimi	Extends little finger at MCP joint
Sixth compartment	Extensor carpi ulnaris	Extends and radially deviates wrist

Key: MCP = metacarpophalangeal.

**TABLE 12-3** Extensor Compartments in the Forearm

Flexor Tendon	Function
Flexor carpi radialis	Flexes and radially deviates wrist
Flexor carpi ulnaris	Flexes and ulnarly deviates wrist
Palmaris longus	Flexes wrist
Flexor pollicis longus	Flexes thumb at MCP and interphalangeal joints
Flexor digitorum superficialis	Flexes index, long, ring, and little digits at MCP and PIP joints
Flexor digitorum profundus	Flexes index, long, ring, and little digits at MCP, PIP, and DIP joints

Key: DIP = distal interphalangeal; MCP = metacarpophalangeal; PIP = proximal interphalangeal.

**TABLE 12-4** Flexor Tendons in the Forearm

## Palm Lacerations

1. Injuries to the palm may require a regional anesthetic, (eg, a median or ulnar nerve block).
2. If no deep injury is suspected, close the wound. Pay particular attention to re-opposing the skin creases accurately.
3. Avoid using deep “bites” with the needle because this risks injury to the underlying tendons or tendon sheaths. Interrupted horizontal mattress sutures with 5-0 monofilament suture are recommended.
4. Refer patients with deep injuries between the carpometacarpal joints and the distal creases of the wrist (“no-mans’ land”) to a specialist for exploration and repair.

## Dorsal Hand Lacerations

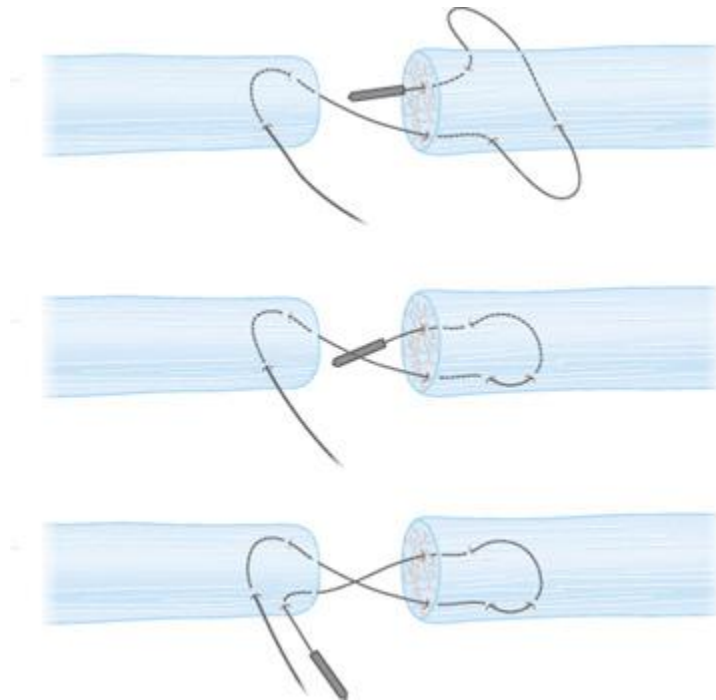
1. Lacerations over the metacarpophalangeal joint suggest a **closed fist injury** and require special care. Polymicrobial infections are the rule; *Staphylococcus aureus*, *Streptococcus* spp., *Corynebacterium* spp., and *Eikenella Corrodens* are the most common bacteria. Obtain x-rays to rule out fracture or embedded teeth. Irrigate thoroughly debride. Infected wounds require IV antibiotics (ampicillin-sulbactam 3 grams every 6

hours), consultation to a hand surgeon, and admission. Patients with noninfected, minor closed fist injuries can be treated as outpatients with immobilization in position of function (do not close), amoxicillin-clavulanic acid 875 PO twice daily (22.5 milligrams/kilogram per dose 2 or 3 times daily in children), and follow-up in 24 to 48 hours with strict return instructions in the event of erythema, drainage, or increased pain.

2. The pliable skin and extensive movements of the hand may hide tendon injuries.
3. Repair skin using 5-0 nonabsorbable sutures.

### **Extensor Tendon Lacerations**

1. Experienced emergency physicians may repair (non-bite) extensor tendon injuries over the dorsum of the hand, with the exception of the tendons to the thumb.
2. Discuss all tendon injuries with a hand specialist for preferred technique and to arrange follow-up.
3. Use a figure-of-8 knot, tied on the side of the lacerated tendon, using a 4-0 (5-0 for smaller tendons) nonabsorbable suture material such as polypropylene ([Fig. 12-2](#)). Close the skin with nonabsorbable suture and splint the limb.



**FIGURE 12-2.** Extensor tendon laceration repair with a figure-of-eight stitch.

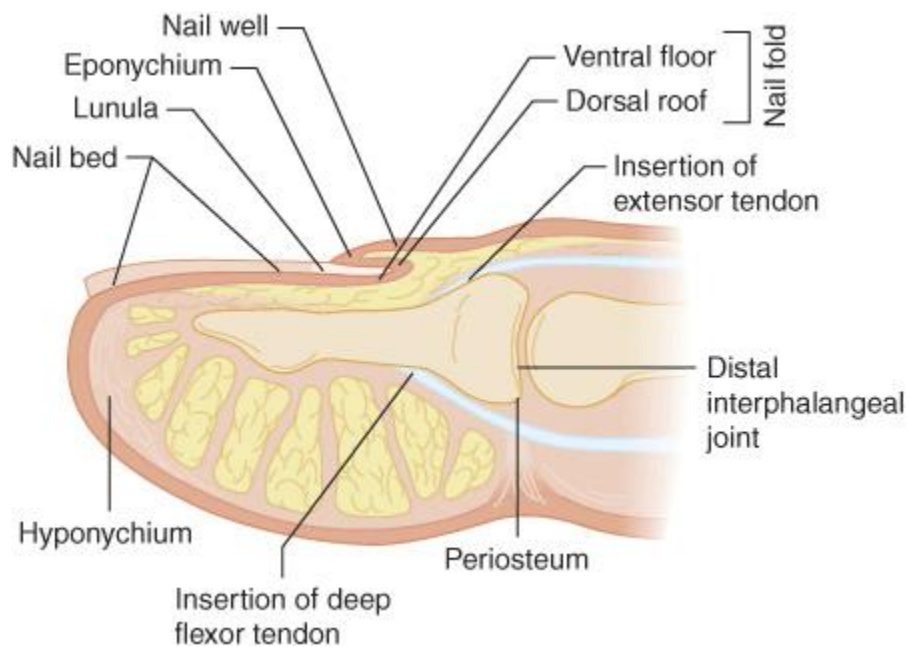
4. Lacerations to the extensor tendons over the distal interphalangeal joint may produce a *mallet deformity*, and if not repaired may result in a *swan neck deformity*; whereas lacerations over the proximal interphalangeal joint may produce a *boutonniere deformity*. Open tendon lacerations require operative repair; closed tendon injuries are either splinted in extension for up to 6 weeks or until operative repair. Refer to a hand surgeon.

## **Flexor Tendon Lacerations**

1. Refer all flexor tendon injuries to a hand specialist.
2. Some hand surgeons prefer to repair these injuries within 12 to 24 hours while others delay repair. If repair is delayed, clean the wound, repair the skin, splint the limb in a position of function, and arrange follow-up within 2 to 3 days with a hand surgeon.

## Finger and Finger Tip Injuries

1. Most finger lacerations are straightforward and can be repaired by using 5-0 nonabsorbable suture materials.
2. Suspect digital nerve injuries when static 2-point discrimination is distinctively greater on one side of the volar pad than on the other, or when it is greater than 10 mm (normal defined as <6 mm).
3. Successful repair of fingertip injuries requires knowledge of anatomy ([Fig. 12-3](#)) and an understanding of techniques of reconstruction.

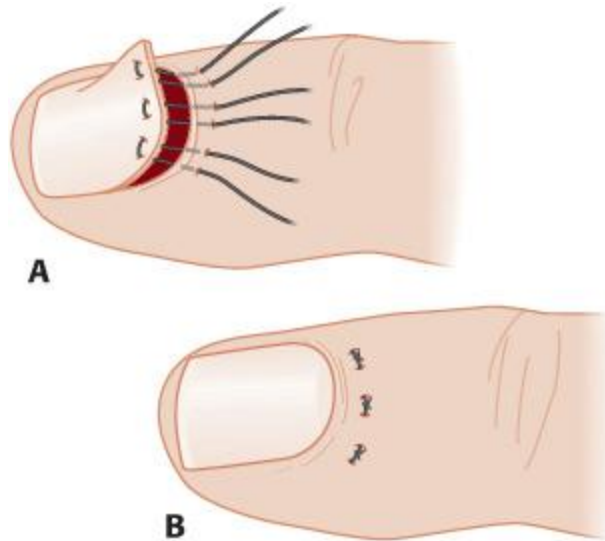


**FIGURE 12-3.** Anatomy of the perionychium.

4. Distal fingertip amputations with skin or pulp loss only are best managed conservatively, with serial dressing change only, especially in children.
5. In cases with larger areas of skin loss ( $> 1 \text{ cm}^2$ ), a skin graft using the severed tip itself or skin harvested from the hypothenar eminence may be required.
6. Complications of the skin graft technique include decreased sensation of the fingertip, tenderness at the injury and graft site, poor cosmetic result, and hyperpigmentation in dark-skinned patients.



7. Injuries with exposed bone are not amenable to skin grafting. Most of these injuries require specialist advice. If less than 0.5 mm of bone is exposed and the wound defect is small, the bone may be trimmed back and the wound left to heal by secondary intention. Injuries to the thumb or index finger with exposed bone nearly always require specialist attention.
8. Subungual hematomas require decompression by simple trephination of the nail plate. Use of heated paper clip delays healing. Use of nail drill, scalpel, or 18 gauge needle is recommended.
9. Simple trephination produces an excellent result in patients with subungual hematoma regardless of size, injury mechanism, or presence of simple fracture.
10. Injuries to the nail bed require careful repair to reduce scar formation. They are associated with fractures of the distal phalanx in 50% of cases.
11. Remove the nail if there is extensive crush injury, associated nail avulsion or surrounding nail fold disruption, or a displaced distal phalanx fracture on radiograph. Repair with 6-0 or 7-0 absorbable sutures. If the nail matrix is displaced from its anatomic position at the sulcus, the matrix should be carefully replaced and sewn in place with mattress sutures ([Fig. 12-4](#)). Alternatively, after nail bed repair, tissue adhesive can be used (dripping it onto the perionychium and into nailfold) can be used to secure the nail, avoiding suturing it in place. Apply mild downward pressure on the nail until the adhesive sets.
12. If there is extensive injury to the nail bed with avulsed tissue, consult a hand specialist.

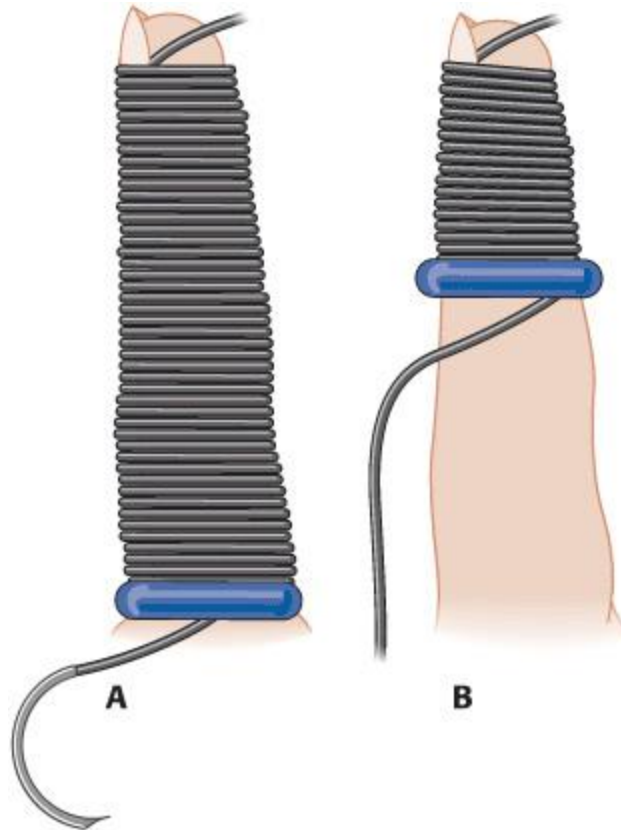


**FIGURE 12-4. A and B.** Technique for repair of an avulsion of the germinal matrix using three horizontal mattress sutures.

13. In children with fractures of the distal phalanx, the nail plate may come to lie on the eponychium. After careful cleaning and adequate anesthesia, replace the nail plate under the proximal nail fold.

## **Ring Tourniquet Syndrome**

1. Ring removal is required in all injured fingers. Swelling may require that the ring be cut off. If slower techniques are appropriate, simple lubrication may suffice.
2. The string technique is an alternative method ([Fig. 12-5](#)).



**FIGURE 12-5.** String technique for ring removal. **A.** Completely wrapped. **B.** Unwrapping with ring advancing off with the string.

- a. String, umbilical tape, or 0 gauge silk may be used.
- b. The string is passed under the ring and then wrapped firmly around the finger from proximal to distal.
- c. The proximal end of the string is then gently pulled, and the ring advances down the finger.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 47, “Injuries to the Arm, Hand, Fingertip and Nail,” by Moira Davenport.

## CHAPTER 13

# Lacerations to the Leg and Foot

Henderson D. McGinnis

## CLINICAL FEATURES

The mechanism of the injury determines the likelihood of disruption to underlying tissue, the risk of a retained foreign body, and the degree of potential contamination. The following circumstances are associated with specific pathogens: (a) farming accidents (*Clostridium perfringens*), (b) wading in a freshwater stream (*Aeromonas hydrophila*), and (c) high-pressure water systems used for cleaning surfaces (*Acinetobacter calcoaceticus*). Evaluation of wounds in general is discussed in [Chapter 9](#). It is important to determine the position of the limb at the time of injury, which will help to uncover occult tendon injuries.

## DIAGNOSIS AND DIFFERENTIAL

Assessment for associated nerve, vessel, or tendon injury is mandatory. Before anesthetizing the area, inspect the wound for position at rest, and assess sensory neurologic function using light touch and 2-point discrimination testing. Compare one side with the other. Motor function and wound exploration may be better assessed after the wound is anesthetized ([Tables 13-1](#) and [13-2](#)). Move the limb through its full range of motion to exclude tendon injury. Test each tendon function individually and inspect visually to rule out a partial laceration.

Nerve	Motor function
Superficial peroneal	Foot eversion
Deep peroneal	Foot inversion Ankle dorsiflexion
Tibial	Ankle plantar flexion

**TABLE 13-1** Motor Function of Lower Extremity Peripheral Nerves

Tendon	Motor function
Extensor hallucis longus	Great toe extension with ankle inversion
Tibialis anterior	Ankle dorsiflexion and inversion
Achilles tendon	Ankle plantar flexion and inversion

**TABLE 13-2** Tendon Function of the Lower Extremities

Laboratory studies usually are not indicated. Obtain an x-ray if there is a possibility of fracture or a radiopaque foreign body. Ultrasonography may be used to help identify a foreign body, a tendon injury, or bony abnormality. All injuries caused by glass should be radiographed unless physical examination can reliably exclude a foreign body (see [Chapter 14](#)).

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

### General Recommendations

1. See [Chapter 9](#) for discussion of wound preparation; thorough irrigation of lower extremity wounds is essential.
2. Wounds on the lower extremities are usually under greater tension than those on the upper limb. Consequently, a layered closure with 4-0 absorbable material to the fascia and interrupted 4-0 nonabsorbable sutures to the skin are preferred. The foot is an exception to this guideline.
3. Avoid deep sutures in diabetics and patients with stasis changes because of the increased risk of infection.
4. Always ask about tetanus immunization status. The elderly are at particular risk for being nonimmunized.

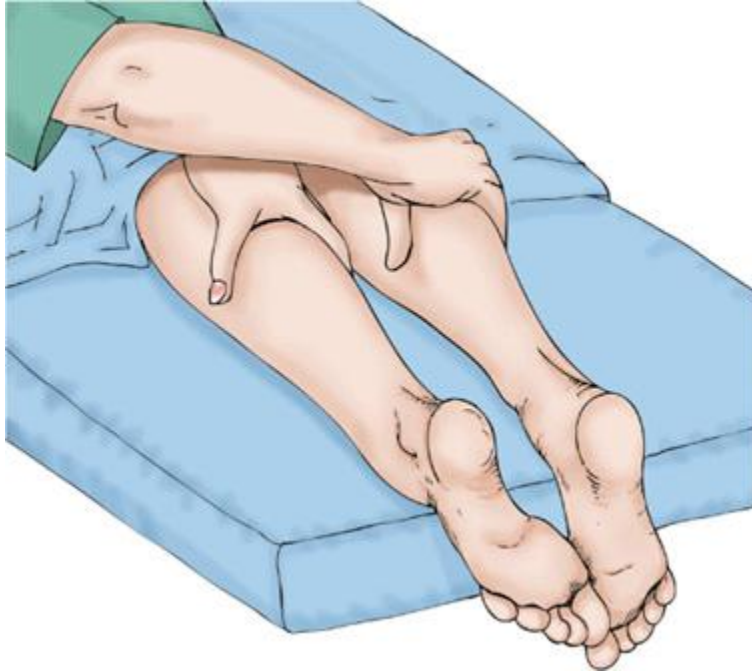
5. Cyanoacrylate glue is usually not used on the lower extremities because of greater wound tension.
6. Splint lacerations involving the joint or tendons in a position of function.

## **Knee injuries**

1. Examine knee wounds throughout the range of movement.
2. Evaluate the joint for possible penetration of the joint capsule and laceration of the patellar and quadriceps tendons. An x-ray may show air in the joint.
3. An alternative approach to diagnose joint penetration is to inject 60 mL of sterile saline, with or without a few drops of sterile fluorescein, into the joint by using a standard joint aspiration technique at a site separate from the laceration. Leakage of the solution from the wound indicates joint capsule injury.
4. The popliteal artery, the popliteal nerve, and the tibial nerve are at risk around the knee: always ascertain their integrity.
5. After closure, splint the knee to prevent excessive tension on the wound edges.

## **Ankle injuries**

1. Move the ankle through its full range of motion and directly inspect the wound to ensure there are no tendon injuries. Particularly at risk are the Achilles tendon, the tibialis anterior, and the extensor hallucis longus.
2. If any of these tendons are injured, they should be formally repaired.
3. The Achilles tendon can rupture without a penetrating injury when a tensed gastrocnemius is suddenly contracted. The Thompson test can be used to assess the Achilles tendon. While kneeling on a chair, or while the patient is supine with feet extending beyond the stretcher (see [Fig. 13-1](#)), the patient's calf is gently squeezed at the midpoint. Absent plantar flexion of the foot indicates complete Achilles tendon laceration (a partial injury may yield plantar flexion).



**FIGURE 13-1. Thompson Test** “With the patient’s feet extending beyond the stretcher, squeeze both calves at the midpoint. Normal response is shown on the patient’s left, absent plantar flexion (abnormal) shown on patient’s right.”

## Foot Injuries

1. Explore lacerations of the sole of the foot to ensure the absence of tendon injury and the absence of foreign bodies. Place the patient in a prone position, with the foot supported on a pillow or overhanging the bed.
2. Regional anesthesia is often best for exploration and repair of lacerations in this area.
3. Some wounds older than 6 hours and less than 12 hours at presentation can be repaired primarily. Each wound must be evaluated individually.
4. Large needles are required to penetrate the thick dermis of the sole. Use nonabsorbable 3-0 or 4-0 material for the sole and 4-0 or 5-0 nonabsorbable sutures for the dorsum.

5. Lacerations between the toes can be difficult to repair. The presence of an assistant who holds the toes apart can be a great help. An interrupted mattress suture often is required to ensure adequate skin apposition.
6. Crutches and a walking boot may be required after repair of any laceration on the foot.
7. Lawn mowers and bicycle spokes may cause extensive soft tissue injury, in addition to underlying fractures and tendon lacerations. These severe injuries require the services of an orthopedic specialist.
8. Consider antibiotic prophylaxis for patients at risk by history or injury mechanism.
9. Wounds caused while wading in fresh water are prone to infection with *Aeromonas*. Prescribe a fluoroquinolone such as **ciprofloxacin** 500 milligrams bid. In children, use **trimethoprim/sulfamethoxazole**, 5 mL suspension per 10 kilograms up to 20 mL bid. *Aeromonas* should be considered in any rapidly progressive case of cellulitis in the foot after an injury.

## **Hair Tourniquet Syndrome**

1. Hair tourniquet syndrome is an unusual type of injury seen in infants. A strand or strands of hair wrap around one of the toes, producing vascular compromise.
2. The hair must be completely cut to avoid compromising the neurovascular bundle to the toe.
3. This is best accomplished by making an incision perpendicular to the hair on the extensor surface of the toe down to the extensor ligament.

## **DISPOSITION**

1. Instruct patients to keep wounds clean and dry.
2. Remove sutures in 10 to 14 days for the lower limb and in 14 days for lacerations over joints.



3. Provide routine wound care instructions. Elevation of the affected limb will reduce edema and aid healing.
4. Recheck wounds after 48 hours if they were heavily contaminated or if a complex repair was required.
5. Crutches can be used for 7 to 10 days, as needed, to prevent additional tension on the wound.

For further reading in *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 48, "Lacerations of the Leg and Foot," by Timothy F. Platts-Mills.

# **CHAPTER 14**

## **Soft Tissue Foreign Bodies**

Rodney L. McCaskill

Retained foreign bodies may lead to a severe inflammatory response (from wood, thorns, and spines), chronic local pain (from glass, metal, and plastic), local toxic reactions (from sea urchin spines and catfish spines), systemic toxicity (from lead), or infection. Most foreign bodies can be located during clinical examination. High risk wounds will need diagnostic imaging. Most foreign bodies should be removed in the emergency department but some may be left in place due to risk of removal.

### **CLINICAL FEATURES**

Every wound has potential for containing a foreign body. The mechanism of injury, composition, and shape of the wounding object as well as the shape and location of the resulting wound determine the risk of a retained foreign body. Lacerating objects that splinter, shatter, or break increase the risk of a foreign body. Patient perception of the sensation of a foreign body more than doubles the likelihood of one being present. Every effort should be made to inspect all recesses of a wound. Extending the edges of the wound is often necessary for complete visualization. Wounds deeper than 5 mm and those whose depths cannot be investigated have a higher association with foreign bodies. Blind probing with a hemostat may be used if the wound is narrow and deep or if extending the wound is not desirable but is less effective.

Patients returning to the emergency department with retained foreign bodies may complain of sharp pain at the wound site with movement, a chronically irritated nonhealing wound, or a chronically infected wound.

### **DIAGNOSIS AND DIFFERENTIAL**

Imaging studies should be ordered if a foreign body is suspected or if multiple foreign bodies were extracted from a wound. Most foreign bodies (80%–90%) can be seen on plain radiographs. MRI, CT, or US may be needed in certain circumstances ([Table 14-1](#)) Using an underpenetrated soft tissue technique or adjusting the contrast and brightness using a digital system may increase the likelihood of identifying a foreign body by increasing the contrast between the foreign body and the surrounding tissue. Wooden foreign bodies can mimic air bubbles on initial CT imaging. Ultrasound is sensitive for detecting foreign bodies larger than 4 to 5 mm but has a high false positive rate and is dependent upon the composition of the foreign body, proximity to echogenic structures, and operator experience. High frequency probes are used for superficial depths and low frequency probes are used to search to depths of 10 cm. MRI is more accurate than the other modalities in identifying wood, plastic, spines, and thorns, but it is less available for emergency use.

Material	Plain Radiographs	High-Resolution US	CT	MRI
Wood	Poor	Good	Good	Good
Metal	Excellent	Good	Excellent	Poor
Glass	Excellent	Good	Excellent	Good
Organic (most plant thorns and cactus spines)	Poor	Good	Good	Good
Plastic	Moderate	Moderate to good	Good	Good
Palm thorn	Poor	Moderate	Good	Good

Adapted with permission from Chan C, Salam GA: Splinter removal. *Am Fam Physician* 67: 2559, 2003.

**TABLE 14-1** Comparison of Imaging Modalities for Detection of Soft Tissue Foreign Bodies

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

### General Principles

Indications for foreign body removal include potential for infection, toxicity, functional problems, or persistent pain. Not all foreign bodies need to be removed.

## **Specific Foreign Bodies and Removal Procedures**

### *Metallic Needles*

If the needle is superficial and can be palpated, an incision can be made over one end and the needle removed. If the needle is deeper, then the incision can be made at the midpoint of the needle and the needle grasped with a hemostat and pushed back out through the entrance wound. If the needle is perpendicular to the skin, the incision may need to be extended, and then pressure on the wound edges may deliver the needle so that it can be grasped and removed.

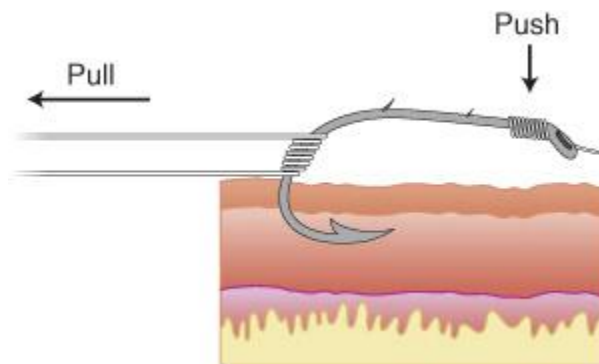
### *Wood Splinters and Organic Spines*

Wooden splinters and organic spines are difficult to remove because of their tendency to break. Only splinters that are superficial should be removed by longitudinal traction. Otherwise the wound should be enlarged and the splinter lifted out of the wound intact. If the splinter is small and localization is difficult, then a block of tissue may be removed in an elliptical fashion and the remaining wound closed primarily. Because of their tendency to become infected, subungual splinters should be removed by traction or by excising a portion of nail over the splinter. Cactus spines may be removed individually or with an adhesive such as facial gel, rubber cement, or household glue applied to the skin and removed when dry.

### *Fishhooks*

Several techniques have been established to remove fishhooks. When using any of the techniques, anesthesia should be injected around the fishhook

entry site. When using the string pull method, one hand depresses the shank of the hook to disengage the barb while the other gives a quick tug on a string that has been wrapped around the bend in the hook ([Fig. 14-1](#)). When the advance-and-cut technique is used, the point and the barb of the fishhook are pushed through the skin and clipped with wire cutters, and the remaining part of the hook is threaded back through the original wound. The final technique involves enlarging the wound down to the barb and then removing the hook. This technique allows for easier wound exploration and cleaning.



**FIGURE 14-1.** String-pull technique for fishhook removal. String or suture material is tied to the curve of the hook. Pressure is applied on the shank of the hook and a quick jerk on the string or suture will dislodge the hook.

## Post-Removal Treatment

Clean and irrigate the wound after removal of a foreign body. The wound may be closed primarily but if there is a significant risk of infection, delayed primary closure is preferred. Obtain a post procedure x-ray if multiple radiopaque foreign bodies are removed. Inform patients about the possibility of a retained foreign body.

## Delayed Removal

If a foreign body is suspected or identified by x-ray but cannot be located with thorough wound evaluation, or if the foreign body is located in an area that prohibits removal, refer the patient to a surgical specialist for delayed removal. If the foreign body is near a tendon or joint, splint the limb. Prophylactic antibiotics, although widely prescribed, may not be necessary in wounds with no sign of infection.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 49, “Soft Tissue Foreign Bodies,” by Richard L. Lammers.

## CHAPTER 15

# Puncture Wounds and Mammalian Bites

David M. Cline

## PUNCTURE WOUNDS

Infection occurs in 6% to 11% of puncture wounds, with *Staphylococcus aureus* predominating (including methicillin-resistant *S. aureus*—MRSA). *Pseudomonas aeruginosa* is the most frequent etiologic agent in postpuncture wound osteomyelitis, particularly when penetration occurs through the sole of an athletic shoe. Post-puncture wound infections with treatment failure suggest the presence of a retained foreign body.

### Clinical Features (See also [Chapter 9](#))

Wounds older than 6 hours with large and deep penetration and obvious visible contamination, which occurred outdoors with penetration through footwear, carry the highest risk of infectious complications. Patients with a history of diabetes mellitus, peripheral vascular disease, immunosuppression, or advanced age are at increased risk of infection.

On physical examination, the likelihood of injury to structures beneath the skin must be determined. Distal function of tendons, nerves, and vessels should be assessed carefully. The site should be inspected for location, condition of the surrounding skin, and the presence of foreign matter, debris, or devitalized tissue. Infection is suggested when there is evidence of pain, swelling, erythema, warmth, fluctuance, decreased range of motion, or drainage from the site.

### Diagnosis and Differential

Multiple view, “soft tissue,” plain film radiographs should be obtained of all infected puncture wounds and of any wound suspicious for a retained foreign body (see [Chapter 14](#) for recommendations on the diagnosis and management of retained foreign bodies).

## **Emergency Department Care and Disposition**

Many aspects of the treatment of puncture wounds remain controversial.

1. Uncomplicated, clean punctures less than 6 hours after injury require only low-pressure irrigation and tetanus prophylaxis, as indicated. Soaking has no proven benefit. Healthy patients do not appear to require prophylactic antibiotics.
2. Prophylactic antibiotics may benefit patients with peripheral vascular disease, diabetes mellitus, and immunosuppression. Plantar puncture wounds, deeper wounds, especially those in high-risk patients, or through athletic shoes should be treated with prophylactic antibiotics. Fluoroquinolones (such as **ciprofloxacin** 500 milligrams twice daily) are recommended for plantar wounds and are acceptable alternatives to parenteral administration of a cephalosporin and aminoglycoside. For other at risk wounds, **cephalexin** 500 milligrams four times daily, or a macrolide, are recommended. In general, prophylactic antibiotics should be continued for 5 to 7 days.
3. Ciprofloxacin is not recommended for routine use in children for prophylaxis. **Cephalexin** 12.5 to 25 milligrams/kilogram/dose 4 times daily up to 500 milligrams/dose can be used with close follow-up.
4. Wounds infected at presentation need to be differentiated into cellulitis, abscess, deeper spreading soft tissue infections, and bone or cartilage involvement. Plain radiographs are indicated to detect the possibility of radiopaque foreign body, soft tissue gas, or osteomyelitis. Bedside ultrasound may identify abscess.
5. Cellulitis usually is localized without significant drainage, developing within 1 to 4 days. There is no need for routine cultures, and antimicrobial coverage should be directed at gram-positive organisms,



especially *S. aureus*. Seven to ten days of a cephalexin (dose above) is usually effective.

6. A local abscess may develop at the puncture site, especially if a foreign body remains. Treatment includes incision, drainage, and careful exploration for a retained foreign body. The wound should be rechecked in 48 hours. Serious, deep, soft tissue infections require surgical exploration and debridement in the operating room.
7. Any patient who relapses or fails to improve after initial therapy should be suspected of having osteomyelitis, septic arthritis, or retained foreign body. Radiographs, white blood cell count, erythrocyte sedimentation rate, and orthopedic consultation should be obtained. Definitive management frequently necessitates operative intervention for debridement. Pending cultures, antibiotics that cover *Staphylococcus* including MRSA and *Pseudomonas* species are started. A reasonable regimen is parenteral **vancomycin** 1 gram IV every 12 hours (in children, 20 milligrams/kilogram per dose every 12 hours) and **ceftazidime** 1 to 2 grams IV every 8 hours (in children, 30 to 50 milligrams/kilogram/dose every 8 hours, not to exceed adult dose).
8. Conditions for admission include wound infection in patients with high risk for complications, immunocompromised states, wounds with progressive cellulitis and lymphangitic spread, osteomyelitis, septic arthritis, and deep foreign bodies necessitating operative removal.
9. Tetanus prophylaxis should be provided according to guidelines (see [Chapter 16](#)). Outpatients should avoid weight bearing, should elevate and soak the wound in warm water, and have follow-up within 48 hours.

## NEEDLE-STICK INJURIES

Needle-stick injuries carry the risk of bacterial infection in addition to the risk of infection with hepatitis B and C, and human immunodeficiency virus (HIV). Because recommendations in this area are complex and evolving, each hospital should have a predesigned protocol developed by infectious disease specialists for the expeditious evaluation, testing, and treatment of needle-stick injuries, including hepatitis B and HIV prophylaxis.

## **HIGH-PRESSURE-INJECTION INJURIES**

High-pressure-injection injuries may present as puncture wounds, usually to the hand or foot. High-pressure-injection equipment is designed to force liquids (usually paint or oil) through a small nozzle under high pressure. These injuries are severe owing to intense inflammation incited by the injected liquid spreading along fascial planes. Patients have pain and minimal swelling. Despite an innocuous appearance, serious damage can develop. Pain control should be achieved with parenteral analgesics; digital blocks are contraindicated to avoid increases in tissue pressure with resultant further compromise in perfusion. An appropriate hand specialist should be consulted immediately, and early surgical debridement should be implemented for an optimal outcome.

## **EPINEPHRINE AUTOINJECTOR INJURY**

These injuries typically occur when a patient attempts self-injection during a rushed attempt to treat an allergic reaction. Patients present with pain due to the needle stick, paresthesias, and epinephrine-induced vasospasm to the injected area; the entire digit may be blanched and cold. There is no clear evidence that active treatment is better than observation alone. The only treatment that has been shown to be beneficial is phentolamine with lidocaine. A mixture of 0.5 mL of standard phentolamine solution (5 milligrams/mL concentration) and 0.5 mL of 1% lidocaine solution will produce a 1 mL total volume containing 2.5 milligrams of phentolamine that can be subcutaneously injected directly through the site of autoinjector puncture. Once the ischemia is resolved (no blanching, warm), the patient can be discharged, as relapse appears very unlikely.

## **HUMAN BITES**

Human bites produce a crushing or tearing of tissue, with potential for injury to underlying structures and inoculation of tissues with normal human oral flora. Human bites are most often reported on the hands and upper extremities. Infection is the major serious sequelae, see [Table 15-1](#) for common organisms.

Animal	Organism	First-Line Antibiotic
Cat	<i>Pasteurella multocida</i> <i>Bartonella henselae</i> (cat-scratch fever)	Amoxicillin-clavulanate Azithromycin
Dog	<i>Pasteurella</i> , streptococci, staphylococci, <i>Capnocytophaga canimorsus</i>	Amoxicillin-clavulanate
Human	<i>Eikenella</i> , staphylococci, streptococci Herpes simplex (herpetic whitlow)	Amoxicillin-clavulanate Acyclovir or valacyclovir
Rats, mice, squirrels, gerbils	<i>Streptobacillus moniliformis</i> (North America) or <i>Spirillum minus/minor</i> (Asia)	Amoxicillin-clavulanate
Livestock, large game animals	Multiple organisms <i>Brucella</i> , <i>Leptospira</i> , <i>Francisella tularensis</i>	Amoxicillin-clavulanate or specific agent for disease
Bats, monkeys, dogs, skunks, raccoons, foxes	Rabies	Rabies immune globulin, rabies vaccine
Monkeys	Herpes B virus (Cercopithecine herpesvirus)	Acyclovir or valacyclovir
Freshwater fish	<i>Aeromonas</i> , staphylococci, streptococci	Fluoroquinolone or Trimethoprim/ sulfamethoxazole
Saltwater fish	<i>Vibrio</i> , staphylococci, streptococci	Doxycycline +/- fluoroquinolone

**TABLE 15-1** Common Bites and First Line Treatment

### Clinical Features (See also [Chapter 9](#))

Of particular concern is the closed fist injury (CFI, or clenched fist injury, or reverse bite injury), which occurs at the metacarpophalangeal (MCP) region as the fist strikes the mouth and teeth of another individual. These hand injuries are at increased risk for serious infection, and any questionable injury in the vicinity of the MCP joint should be considered a CFI until proven otherwise. See [Chapter 12](#) for more information.

The physical examination should include assessment of the direct injury and a careful evaluation of the underlying structures, including tendons, vessels, nerves, deep spaces, joints, and bone. Local anesthesia usually is required to perform a careful wound exploration. In a CFI, the wound must be examined through a full range of motion at the MCP joint to detect extensor tendon involvement, as the tendon may have retracted proximally in the unclenched hand. The examination also must assess a potential joint-space violation. Radiographs are recommended, particularly of the hand, to delineate foreign bodies and fractures.

Human bites to the hand frequently are complicated by cellulitis, lymphangitis, abscess formation, tenosynovitis, septic arthritis, and osteomyelitis. Infections from human bites are polymicrobial, with staphylococcal and streptococcal species being common isolates in addition to species-specific *Eikenella corrodens*.

## **Diagnosis and Differential**

History and physical examination usually will indicate a straightforward diagnosis. There are times, however, when a patient may try to conceal or deny the true etiology of a human bite, and a high degree of suspicion is warranted, particularly when the wound is on the hand. It is important to keep in mind that viral diseases also can be transmitted by human bites (eg, herpes simplex, herpetic whitlow, and hepatitis B). The potential risk of acquiring HIV through a human bite appears to be negligible due to low levels of HIV in saliva.

## **Emergency Department Care and Disposition**

1. Copious wound irrigation with a normal saline solution and judicious limited debridement of devitalized tissue are critical to initial management.
2. Human bites to the hand initially should be left open. Other sites can undergo primary closure unless there is a high degree of suspicion for infection.

3. Prophylactic antibiotics should be considered in all but the most trivial of human bites. **Amoxicillin/clavulanate** 500 to 875 milligrams PO twice daily (12.5 to 22.5 milligrams/kilogram/dose twice daily in children) is the antibiotic of choice.
4. See [Chapter 12](#) for management of closed/clenched fist injury. Herpetic whitlow is treated with acyclovir or valacyclovir (see [Chapter 182](#) for discussion).
5. Wounds that are infected at presentation require systemic antibiotics after cultures are obtained. Local cellulitis in healthy and reliable patients may be managed on an outpatient basis with immobilization, antibiotics, and close follow-up. Moderate to severe infections require admission for surgical consultation and parenteral antibiotics. Appropriate coverage includes **ampicillin/sulbactam** 3 g IV every 6 hours (in children, 25 to 37.5 milligrams/kilogram/dose every 6 hours) or **cefoxitin** 2.0 g IV every 8 hours (in children 27 to 33 milligrams/kilogram/dose IV or IM up to 2.0 g every 8 hours). Penicillin-allergic patients may be treated with **clindamycin** (5 to 10 milligrams/kilogram/dose IV 4 times daily, up to 600 milligrams/dose) *plus* **ciprofloxacin** (10 milligrams/kilogram/dose every 12 hours IV; maximum:400 milligrams/dose).
6. All patients should receive tetanus immunization according to guidelines.

## DOG BITES

### Clinical Features

Dog bites account for 80% to 90% of reported animal bites, with school-age children sustaining the majority of reported bites. Infection occurs in approximately 5% of cases and is more common in patients older than 50 years, those with hand wounds or deep puncture wounds, and those who delay in seeking initial treatment over a 24-hour period. A thorough history and examination as outlined in the section on human bites are required to assess the extent of the wound and the likelihood of infection. Infections from dog bite wounds are often polymicrobial and include aerobic and anaerobic bacteria.

## Diagnosis and Differential

Radiographs are recommended if there is evidence of infection, suspicion of a foreign body, bony involvement, or large dog intracranial penetration bites to the heads of small children.

## Emergency Department Care and Disposition

1. All dog bite wounds require appropriate local wound care with copious irrigation and debridement of devitalized tissue.
2. Primary closure can be used in wounds to the scalp, face, torso, and extremities other than the feet and hands. Lacerations of the feet and hands should be left open initially. Large, extensive lacerations, especially in small children, are best explored and repaired in the operating room.
3. Puncture wounds, wounds to the hands and feet, and wounds in high-risk patients should receive 3 to 5 days of prophylactic antibiotics with **amoxicillin/clavulanate** 500 to 875 milligrams PO twice daily (12.5 to 22.5 milligrams/kilogram/dose twice daily in children) or **clindamycin** (5 milligrams/kilogram/dose 4 times daily, up to 450 milligrams/dose PO *plus* **ciprofloxacin** (15 milligrams/kilogram/dose every 12 hours; maximum: 500 milligrams/dose PO). Clindamycin *plus* trimethoprim-sulfamethoxazole can be used for the penicillin allergic patient.
4. Wounds obviously infected at presentation need to be cultured and antibiotics initiated. Reliable, low-risk patients with only local cellulitis and no involvement of underlying structures can be managed as outpatients with close follow-up.
5. Significant wound infections require admission and parenteral antibiotics. Examples include infected wounds with evidence of lymphangitis, lymphadenitis, tenosynovitis, septic arthritis, osteomyelitis, systemic signs, and injury to underlying structures, such as tendons, joints, or bones. Cultures should be obtained from deep structures, preferably during exploration in the operating room. Initial antibiotic therapy should begin with **ampicillin/sulbactam** 3 g IV every 6 hours or

**clindamycin** (5 to 10 milligrams/kilogram/dose IV 4 times daily, up to 600 milligrams/dose) *plus* **ciprofloxacin** (10 milligrams/kilogram/dose every 12 hours IV; maximum: 400 milligrams/dose). If the Gram stain reveals gram-negative bacilli, a third or fourth generation cephalosporin or aminoglycoside should be added.

6. Tetanus prophylaxis should be provided according to standard guidelines.

## **CAT BITES**

Cat bites account for 5% to 18% of reported animal bites, with the majority resulting in puncture wounds on the arm, forearm, and hand. Up to 80% of cat bites become infected.

### **Clinical Features**

*Pasteurella multocida* is the major pathogen, isolated in 53% to 80% of infected cat bite wounds. *Pasteurella* causes a rapidly developing intense inflammatory response with prominent symptoms of pain and swelling. It may cause serious bone and joint infections and bacteremia. Many patients with septic arthritis due to *P. multocida* have altered host defenses due to glucocorticoids or alcoholism.

### **Diagnosis and Differential**

Radiographs are recommended if there is evidence of infection, suspicion of a foreign body, or bony involvement.

### **Emergency Department Care and Disposition**

1. All cat bite wounds require appropriate local wound care with copious irrigation and debridement of devitalized tissue.

2. Primary wound closure is usually indicated, except in puncture wounds and lacerations smaller than 1 to 2 cm, because they cannot be adequately cleaned. Delayed primary closure also can be used in cosmetically important areas. Factors favoring delayed closure or avoiding simple primary closure include presentation beyond 6 hours, lack of cosmetic concern, complex repair needed, and underlying injury requiring surgical intervention.
3. Prophylactic antibiotics should be administered to high-risk patients including those with punctures of the hand; immunocompromised patients; and patients with arthritis or prosthetic joints. The case can be made that all patients with cat bites should receive prophylactic antibiotics because of the high risk of infection. **Amoxicillin/clavulanate** 500 to 875 milligrams PO twice daily (12.5 to 22.5 milligrams/kilogram/dose 2 times daily in children), **cefuroxime** 500 milligrams PO twice daily (10 to 15 milligrams/kilogram/dose twice daily in children), or **doxycycline** 100 milligrams PO twice daily in adults (in children, 1 to 2 milligrams/ kilogram/dose twice daily, up to 100 milligrams/dose) administered 3 to 5 days are appropriate.
4. For cat bites that develop infection, evaluation and treatment are similar to those for dog bite infections. Penicillin is the drug of choice for *P. multocida* infections.
5. Tetanus prophylaxis should be provided according to standard guidelines.

## **RODENTS, LIVESTOCK, EXOTIC AND WILD ANIMALS**

Rodent bites are typically trivial, rodents are not known to carry rabies, and these bites have a low risk for infection. Livestock and large game animals can cause serious injury. There is also a significant risk of infection and systemic illness caused by brucellosis (see [Chapter 98](#)), leptospirosis (see [Chapter 98](#)), and tularemia (see [Chapter 97](#)). Aggressive wound care and broad-spectrum antibiotics are recommended. Specific agents are listed in [Table 15-1](#).



For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 50, “Puncture Wounds and Bites,” by Robert A. Schwab and Robert D. Powers.

# **CHAPTER 16**

## **Post Repair Wound Care**

Eugenia B. Quackenbush

After repair, proper care is focused on optimized healing and prevention of complications. Considerations include use of dressing, positioning, prophylactic antibiotics, and tetanus prophylaxis. Appropriate follow up and patient education regarding cosmetic results are important.

### **USE OF DRESSINGS**

Wound dressings provide a moist environment that promotes epithelialization and speeds healing. Semipermeable films such as OpSite® are available in addition to conventional gauze dressings. The disadvantages of these materials are their inability to absorb large amounts of fluid. Alternatively, topical antibiotics may be used to provide a warm, moist environment. Topical antibiotics may reduce the rate of wound infection and also may prevent scab formation. Wounds closed with tissue adhesives should not be treated with topical antibiotic ointment because it will loosen the adhesive.

### **PATIENT POSITIONING AFTER WOUND REPAIR**

The injured site should be elevated, if possible, to reduce edema around the wound and speed healing. Splints are useful for extremity injuries because they decrease motion and edema and increase attention paid to the body part. Pressure dressings minimize the accumulation of fluid and are most useful for ear and scalp lacerations (see [Chapter 11](#)).

# PROPHYLACTIC ANTIBIOTICS

Prophylactic oral antibiotics are only indicated in specific clinical circumstances. When deciding whether or not to prescribe antibiotics, consider the mechanism of injury (ie, crush injury), degree of bacterial or soil contamination, and host predisposition to infection.

Prophylactic antibiotics are recommended for human bites, dog or cat bites on the extremities (see [Chapter 15](#)), intraoral lacerations (see [Chapter 11](#)), open fractures, and wounds with exposed joints or tendons (see [Chapters 12, 13](#)). Patients with wounds in areas with lymphedema will likely benefit from prophylactic antibiotics as well. A 3 to 5 day course is adequate for non-bite injuries and a 5 to 7 day course is adequate for bite wounds.

# TETANUS PROPHYLAXIS

The need for tetanus prophylaxis should be considered for every wounded patient. Inquire about the mechanism of injury, age of the wound, and the patient's tetanus immunization status. The only contraindication to tetanus toxoid is a history of neurologic or severe systemic reaction after a previous dose (see [Table 16-1](#) for a summary of recommendations for tetanus prophylaxis).

History of Tetanus Immunization	Clean Minor Wounds		All Other Wounds*	
	Administer Tetanus Toxoid†	Administer TIG‡	Administer Tetanus Toxoid	Administer TIG
<3 or uncertain doses	Yes	No	Yes	Yes
≥3 doses				
Last dose within 5 y	No	No	No	No
Last dose within 5–10 y	No	No	Yes	No
Last dose >10 y	Yes	No	Yes	No

\*Especially if wound care delayed (>6 h), deep (>1 cm), grossly contaminated, exposed to saliva or feces, stellate, ischemic or infected, avulsions, punctures, or crush injuries.

†Tetanus toxoid: Tdap if adult and no prior record of administration, otherwise tetanus-diphtheria toxoid if >7 years old and diphtheria-tetanus toxoid if <7 years old, preferably administered into the deltoid.

‡Tetanus immune globulin: adult dose, 250–500 IU administered into deltoid opposite the tetanus-diphtheria toxoid immunization site.

**TABLE 16-1** Recommendations for Tetanus Prophylaxis

## WOUND CLEANSING

Sutured or stapled wounds may be cleansed as early as 8 hours after closure without increasing risk of wound infection. Wounds should be gently cleansed with soap and water and examined for signs of infection daily. Application of topical antibiotics for the first 3 to 5 days decreases scab formation and prevents edge separation. Patients with wounds closed with tissue adhesives may shower, but should not immerse the wound or apply topical antibiotics, as this will loosen the adhesive bond and cause earlier sloughing of the adhesive.

## WOUND DRAINS

Drains are placed in wounds for removal of interstitial fluid or blood, to keep an open tract for drainage of pus, or to prevent an abscess from forming by allowing drainage from a contaminated area. Closed drainage systems have largely replaced open wound drains, especially after surgery, because closed systems prevent bacteria access into the wound. Ribbon gauze packing in an abscess cavity after I&D is the most common drain

used in the ED. Advise patients to change packing daily until the wound stops producing exudate.

## **PAIN CONTROL**

Inform patients about the expected degree of pain and measures that might reduce pain. Splints help reduce pain and swelling in extremity lacerations. Analgesics may be needed although narcotic analgesia is rarely necessary after the first 48 hours.

## **HEALTH CARE PROVIDER FOLLOW-UP**

Provide specific instructions for wound examination or suture removal. Patients with high risk wounds or conditions or those unable to identify signs of infection should be instructed to return for re-exam, usually in 48 hours. Facial sutures should be removed in 3 to 5 days. Most other sutures can be removed in 7 to 10 days, except for sutures in the hands or over joints, which should remain for 10 to 14 days. When removing sutures or adhesive tapes, take care to avoid tension perpendicular to the wound that could cause dehiscence. Tissue adhesives will slough off within 5 to 10 days of application.

## **PATIENT EDUCATION ABOUT LONG-TERM COSMETIC OUTCOME**

Inform patients that all traumatic lacerations result in some scarring and that the short-term cosmetic appearance is not highly predictive of the ultimate cosmetic outcome. Instruct patients to avoid sun exposure while their wounds are healing because it can cause permanent hyperpigmentation. Patients should wear sunblock for at least 6 to 12 months after injury.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 51, “Postrepair Wound Care,” by Adam J. Singer and Judd E. Hollander.

## **SECTION 4 Cardiovascular Diseases**

# CHAPTER 17

## Chest Pain: Cardiac or Not

Thomas Rebbecchi

Patients with acute nontraumatic chest pain are among the most challenging patients cared for by emergency physicians. They may appear seriously ill or completely well and yet remain at significant risk for sudden death or an acute myocardial infarction (AMI).

### CLINICAL FEATURES

The typical pain of myocardial ischemia has been described as retrosternal or epigastric squeezing, tightening, crushing, or pressure-like discomfort. The pain may radiate to the left shoulder, jaw, arm, or hand. In many cases, particularly in the elderly, the chief complaint is not chest pain, but of a poorly described visceral sensation with associated dyspnea, diaphoresis, nausea, light-headedness, or profound weakness. The onset of symptoms may be sudden or gradual, and symptoms usually last minutes to hours. In general, symptoms that last less than 2 min or are constant over days are less likely to be ischemic in origin. Symptoms that are new or familiar to the patient but now occur with increasing frequency, severity, or at rest are called *unstable* and warrant urgent evaluation even if they are absent at the time of presentation. Cardiac risk factors should be used only to predict coronary artery disease within a given population and not in an individual patient. Women, diabetics, the elderly, and patients with psychiatric disorders may have more atypical symptoms of ischemia. Although some symptoms such as radiation of pain to the arms, an exertional component, associated diaphoresis, nausea and vomiting increase the likelihood that a patient is suffering from an AMI; there is no identifiable symptom complex that allows for a definitive diagnosis of the AMI patient without objective testing.



Patients with acute myocardial ischemia may appear clinically well or be profoundly hemodynamically unstable. The degree of hemodynamic instability is dependent on the amount of myocardium at risk, associated dysrhythmias, or preexisting valvular or myocardial dysfunction. Worrisome signs may be clinically subtle, particularly the presence of sinus tachycardia, which may be due to pain and fear or may be an early sign of physiologic compensation for left ventricular failure. Patients with acute ischemia often have a paucity of significant physical findings. Rales, a third or fourth heart sound, cardiac murmurs, or rub are clinically relevant and important findings. The presence of chest wall tenderness has been demonstrated in 5% to 10% of patients with AMI, so its presence should not be used to exclude the possibility of acute myocardial ischemia. Also, response to a particular treatment such as nitroglycerin or a “gastrointestinal (GI) cocktail” should not be taken as evidence of a certain disease.

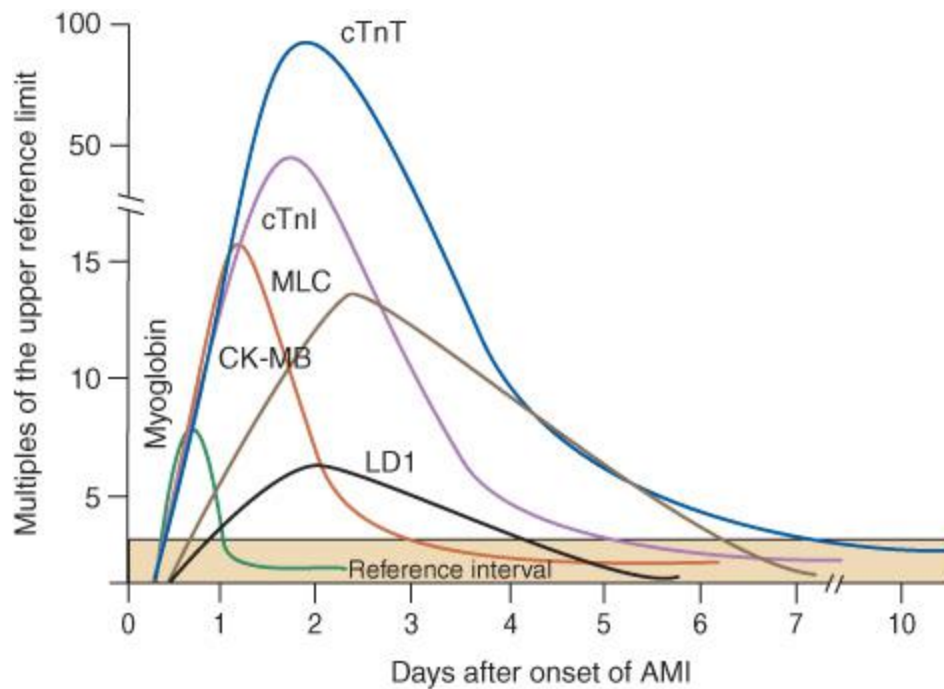
## **DIAGNOSIS**

### **Electrocardiography**

Of all the diagnostic tools clinically used in assessing chest pain, the electrocardiogram (ECG) is the most reliable when used and interpreted correctly. Patients with acute infarctions may have ECG findings that range from acute ST-segment elevations to completely normal. This range means that the ECG is useful only when it has a positive, or diagnostic, finding. New ST-segment elevations, Q waves, bundle branch block, and T-wave inversions or normalizations are strongly suggestive of ischemia and warrant aggressive management in the emergency department (ED). The presence of a normal or unchanged ECG does not rule out the diagnosis of acute myocardial ischemia.

### **Serum Markers**

Serum markers, if positive, are highly specific for AMI. Creatinine kinase and its MB isoenzyme constitute the historical gold standard for diagnosing AMI, but have been replaced by troponin as the standard for the diagnosis of AMI. Cardiac-specific troponin I and T are not found in skeletal muscle, and have a much greater sensitivity and specificity for AMI. Other clinical conditions such as aortic dissection, acute CHF, aortic valve disease, and some arrhythmias can be associated with an elevated troponin in the absence of ischemic heart disease. However, the documentation of normal serum markers in the bloodstream does not exclude the diagnosis of AMI. These enzymes will not become elevated in serious disease conditions such as unstable angina. The use of these markers can aid the clinician in assessing risk for patients with chest pain, including disposition within the hospital. A serial enzyme evaluation can be used to appropriately risk stratify individual patients ([Fig. 17-1](#)).



**FIGURE 17-1.** Serum biomarker curve. Typical pattern of serum marker elevation after acute myocardial infarction (AMI).

*Key:* CK-MB = MB fraction of creatine kinase; cTnI = cardiac troponin I; cTnT = cardiac troponin T; LD1 = lactate dehydrogenase isoenzyme 1; MLC = myosin light chain.

## **Echocardiography**

Emergency 2-dimensional echocardiography may have value in the evaluation of chest pain when the ECG is nondiagnostic, eg, in patients with pacemakers, have a bundle branch block, or have an abnormal ECG at baseline. The finding of regional wall motion abnormalities in the acutely symptomatic patient is strongly suggestive of active ischemia. Wall motion abnormality also may represent previous myocardial injury. Two-dimensional echocardiography also may aid in the diagnosis of other conditions that may mimic ischemic disease, such as pericarditis, aortic dissection, or hypertrophic cardiomyopathy.

## **Provocative Tests**

Many tests currently being performed in some EDs will unmask otherwise unrecognized but clinically significant ischemic disease. Patients with atypical chest pain and a normal stress thallium or technetium scan have a very low incidence of short- and long-term subsequent ischemic events. Thallium or sestamibi testing can be done in the ED to further risk stratify patients in the hospital and perhaps be used in consideration for patient discharge from the ED.

The priority must always be to exclude life-threatening conditions, and the ED physicians should organize their test-ordering strategies to screen for those conditions first. ([Table 17-1](#) lists possible causes of nontraumatic chest pain.)

Disorder	Character of Pain
Angina pectoris	Tightness, crushing, or squeezing pressure in the retrosternal or epigastric area, with radiation to R or L shoulder, arm, or jaw.
Massive pulmonary embolism	Heaviness, tightness involving the whole chest, in the setting of unstable vital signs, and/or syncope, with a sense of impending doom.
Segmental pulmonary embolism	Pleuritic chest pain in a focal area of the chest, with associated tachypnea or tachycardia and/or dyspnea.
Aortic dissection	Ripping or tearing substernal pain, radiating to the back (intrascapular), possibly with neurologic symptoms (focal hypoperfusion).
Pneumothorax	Sharp, sudden, lancinating, pleuritic pain on one side of the chest, associated with dyspnea.
Esophageal rupture	Sudden, sharp, substernal chest pain following forceful vomiting, with radiation to the back.
Pericarditis	Sharp, constant or pleuritic substernal chest pain, with radiation to the back or shoulder.
Pneumonia	Sharp, pleuritic focal chest pain associated with fever.
Perforated ulcer	Severe, sharp epigastric pain, radiating to the back, with acute distress.

\*Atypical presentations are common with all listed life-threatening disorders.

Key: L = left, R = right.

**TABLE 17-1** Life-Threatening Causes of Chest Pain: Classic Symptoms Compared\*

## Differential Diagnosis

What follows is list of common etiologies of chest pain that should be considered by the emergency physician ([Table 17-1](#)).

### *Angina Pectoris*

The typical pain of chronic stable angina is episodic and lasts 5 to 15 min. It is precipitated by exertion and relieved with rest or sublingual nitroglycerin within 3 min. The pain is typically visceral in nature (aching, pressure, and squeezing), with radiation to the neck, jaw, arm, or hand. In individual patients the character of each attack varies little with recurrent episodes. Most patients can differentiate their usual angina from other causes of pain. Physicians evaluating patients with stable angina should screen carefully for

changes in the pattern that would suggest a shift from stable to unstable angina or even suggest a different diagnosis.

### *Unstable Angina*

Patients who complain of recent onset of angina, changes in the character of the angina, or angina at rest are thought to have an unstable pattern of their angina. They are at risk for an AMI or sudden cardiac death (see [Chapter 18](#) for management).

### *Variant (Prinzmetal) Angina*

This form of angina is thought to be due to spasm of the epicardial vessels in patients with normal coronary arteries (one-third of cases) or in patients with underlying atherosclerotic disease (two-thirds of cases). Pain typically occurs at rest and may be precipitated by the use of tobacco or cocaine. The ECG typically shows ST-segment elevations during an acute attack.

### *Acute Myocardial Infarction*

Ischemic pain that lasts longer than 15 min, is not relieved by nitroglycerin, or is accompanied by diaphoresis, dyspnea, nausea, or vomiting suggests the diagnosis of AMI. The clinician must understand the limitations of the screening tools used in the ED and should have a high level of suspicion for AMI in patients with risk factors and prolonged or persistent symptoms for which there is no other clear diagnosis (see [Chapter 18](#)).

### *Aortic Dissection*

This diagnosis should be suspected in the patient who complains of sudden onset of severe, tearing pain in the retrosternal or midscapular area. High-risk patients are also those at risk for AMI, specifically the middle-age hypertensive male. The patient may be hypertensive or hypotensive in shock. There may be a diastolic murmur of aortic regurgitation, indicating a

proximal dissection, or distal pulse deficits, indicating a distal dissection. The dissection may occlude coronary ostia, resulting in myocardial infarction, or the carotids, resulting in cerebral ischemia and stroke. Chest x-ray, computed tomography, transesophageal echocardiography (TEE), and angiography can aid in the diagnosis of this condition (see [Chapter 27](#) for a complete discussion of aortic dissection).

### *Pericarditis*

The patient with pericarditis typically will complain of pain that is constant, retrosternal, and radiating to the back, neck, or jaw. Pain is classically worsened by lying supine and is relieved by sitting forward. The presence of a pericardial friction rub supports the diagnosis. ECG may show PR-segment depressions, diffuse ST-segment elevations, or T-wave inversions that are typically diffuse (see [Chapter 24](#) for a complete discussion of pericarditis).

### *Acute Pericardial Tamponade*

Patients with acute tamponade may complain of positional or pleuritic chest pain, dyspnea, and palpitations. Physical examination will show tachycardia, hypotension, jugular venous distention, and distant heart sounds. If cardiovascular collapse is imminent, emergent pericardiocentesis is indicated.

### *Pulmonary Embolus*

Patients typically complain of sudden onset of pleuritic chest pain associated with dyspnea, tachypnea, tachycardia, or hypoxemia. The absence of any of these findings does not preclude the diagnosis, and a high index of suspicion is essential (see [Chapter 25](#) for a complete discussion of pulmonary embolism).

### *Musculoskeletal Causes*

Chest pain due to irritation or inflammation of structures in the chest wall is commonly seen in the ED. Possible causes include *costochondritis*, intercostal strain due to severe coughing, and pectoralis muscle strain in the setting of recent physical exertion. Patients will complain of sharp pain that is worsened with movement of the chest wall (eg, coughing, and some pain that can be elicited by palpation of the chest wall). These findings in patients without any other symptoms and no history of significant cardiac disease support the diagnosis of musculoskeletal pain. This pain is generally responsive to nonsteroidal anti-inflammatory drugs. It is important to emphasize that the presence of chest wall tenderness does not rule out the possibility of myocardial ischemia.

### *Gastrointestinal Causes*

Esophageal reflux, dyspepsia syndromes, and esophageal motility disorders can produce chest pain that is difficult to distinguish from ischemic pain. Patients may complain of burning, gnawing pain associated with an acid taste radiating into the throat. Pain may be exacerbated by meals, worsen when supine, and may be associated with belching. Clinicians should determine whether the symptoms are due to a GI disorder based on the clinical presentation and the absence of findings and/or risk factors suggesting an ischemic cause. Diagnostic decisions should not be made on the basis of a response to a therapeutic trial of antacids, GI cocktails, or nitroglycerin (see [Chapters 35](#), [40](#), and [41](#) for more discussion on GI causes of chest pain).

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. It should be assumed that every patient with any risk complaining of chest pain may be having an AMI and should be evaluated for this diagnosis.

2. Patients with suspicious histories should have a large-bore intravenous line established, a cardiac monitor, and supplemental oxygen. Vital signs and pulse oximetry should be monitored continuously.
3. An ECG should be obtained on all patients for whom there is a reasonable suspicion of myocardial ischemia.
4. Aspirin should be given early as other testing is being accomplished in patients considered at risk for AMI (see [Chapter 18](#) for details and additional medications).
5. If the etiology of chest pain remains unclear, clinicians should consider further testing and observation/admission as guided by clinical suspicion and findings.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 52, “Chest Pain: Cardiac or Not,” by Gary B. Green and Peter M. Hill.



## CHAPTER 18

# Acute Coronary Syndromes: Management of Myocardial Infarction and Unstable Angina

Jim Edward Weber

Mortality and morbidity reduction in acute coronary syndromes (ACS) is predicated upon minimizing the time interval between onset of ischemia and provision of treatment.

## CLINICAL FEATURES

Chest pain is the main symptom associated with ischemic heart disease. History should include its severity, location, radiation, duration, and quality. In addition, clinicians should ask about onset and duration of symptoms, provocative or palliative activities, and prior evaluations.

Seven major risk factors for coronary artery disease (CAD) have been identified: age, male sex, family history, cigarette smoking, hypertension (HTN), hypercholesterolemia, and diabetes (DM). However, cardiac risk factors are poor predictors of acute myocardial infarction (AMI) in ED patients. One risk factor, cocaine use, is noteworthy. Cocaine is directly myotoxic, accelerates atherosclerosis and CAD, and may cause myocardial infarction (MI) in patients.

Angina pectoris represents cardiac *ischemia*, a form of ACS. ACS symptoms may include: chest pain or discomfort, nausea, vomiting, diaphoresis, dyspnea, light-headedness, syncope, and palpitations. Reproducible chest wall tenderness is not uncommon. Angina is typically precipitated by exercise, stress, or cold temperature; pain lasts <10 min and is relieved by rest or nitroglycerin (NTG). Unstable angina, an ACS, represents a clinical state between stable angina and AMI. Unstable angina is present when anginal symptoms meet any of the following criteria: (a) new-onset angina (within 2 months); (b) increasing angina (increased

frequency or duration, or decreased threshold for symptom occurrence); (c) angina at rest (within one week).

As compared to angina, AMI is usually accompanied by more severe and prolonged chest discomfort. Symptoms tend to be less responsive to nitroglycerin, and associated symptoms (eg, diaphoresis) are more prominent.

Atypical presentations are common. Elderly patients and those with diabetes may have silent (painless) ischemia. Easy fatigability and/or shortness of breath are common ACS presenting symptoms in women and elderly men. Patients with inferior AMI may have abdominal pain, nausea, or vomiting.

Physical exam findings in patients with ACS range from normal to overt distress. The pulse rate, cardiac rhythm and blood pressure should be assessed and addressed. The first and second heart sounds may be diminished with LV dysfunction. An  $S_3$  implies myocardial dysfunction and an  $S_4$  suggests longstanding hypertension or myocardial dysfunction. A new murmur may signify papillary muscle dysfunction, valve regurgitation, or a ventricular septal defect. Similarly, the presence of rales is associated with LV dysfunction and left-sided CHF. JVD and peripheral edema suggest right heart failure.

## **DIAGNOSIS AND DIFFERENTIAL**

The differential diagnosis of cardiac ischemia is particularly broad (see [Chapter 17](#)). Entities that should be considered include pericarditis, cardiomyopathies, cardiac valvular disease, pulmonary embolism, pneumonia, pneumothorax, asthma or chronic obstructive pulmonary disease, gastrointestinal disorders (especially esophageal disease), chest trauma, chest wall disorders, hyperventilation, aortic aneurysm and dissection, and mediastinal disorders.

The diagnosis of ST-elevation myocardial infarction (STEMI) is based upon appropriate ECG changes occurring in a suggestive clinical setting; early treatment and disposition decision-making do not require serum cardiac marker results. The diagnosis of a non-ST-segment elevation

myocardial infarction (NSTEMI) is made when abnormal elevation of cardiac markers is accompanied by ECG findings (eg, T-wave inversion, ST-segment depression) that fail to meet criteria for STEMI. The diagnosis of unstable angina is clinical.

The ECG is the single best test for identification of AMI patients at the time of ED presentation. An initial ECG should be obtained and interpreted within 10 min of ED presentation. Normal or nonspecific findings do not rule out ischemia or negate the need for hospitalization.

Diagnostic ECG criteria for AMI are shown in [Table 18-1](#). *STEMI* changes in the listed distributions suggest acute transmural injury. *ST-segment depressions* in these distributions suggest ischemia. In general, the greater the number and degree of ST-elevations, the more extensive the myocardial injury. In the setting of acute inferior wall STEMI, an ECG with right-sided lead placement is recommended; ST-segment elevation in  $V_{4R}$  is suggestive of RV infarction.

Anterior	rS deflection in $V_1$ with Q waves in $V_{2-4}$ or decrease in amplitude of initial R waves in $V_{1-4}$
Inferior	Q waves in II, III, aVF
Anteroseptal	QS deflections in $V_1$ - $V_3$ , and possibly $V_4$
Lateral	Q waves in I and aVL
Anterolateral	Q waves in $V_4$ - $V_6$ , I, and aVL
Inferolateral	Q waves in II, III, aVF, and $V_5$ and $V_6$
Right ventricular	Q waves in II, III, and aVF, and ST elevation in right-side $V_4$
True posterior*	Initial R waves in $V_1$ and $V_2$ > 0.04s and R/S ratio $\geq 1$

\*Posterior wall infarction does not produce Q wave abnormalities in conventional leads and is diagnosed in the presence of tall R waves in  $V_1$  and  $V_2$ .

**TABLE 18-1** Electrocardiographic Q-Wave-Based Criteria for AMI

Patients with persistent or suggestive symptoms and a *nondiagnostic* initial ECG should undergo repeat ECG every 15 to 30 min. Previous ECG tracings are useful for risk stratification. ECG evidence of new ischemia increases the risk of both unstable angina and AMI ([Table 18-1](#)). A rise in serum troponin I or T above the institutional threshold for infarction is diagnostic for AMI in patients with symptoms consistent with ACS. Serial marker sampling increases both the sensitivity and specificity of cardiac enzyme testing for AMI.

ECG evaluation for ischemia can be rendered difficult in the setting of paced rhythms or bundle branch block. MI in the setting of left bundle branch block or paced rhythms is suggested by: (a) ST elevation of > 1 mm concordant with the QRS complex; (b) ST depression of > 1 mm in leads V<sub>1</sub>, V<sub>2</sub>, or V<sub>3</sub>; or (c) discordant ST elevation of at least 5 mm.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The primary goal of initial treatment is early reperfusion, achieved by either fibrinolytics or percutaneous coronary intervention (PCI). Institutional goals for reperfusion are PCI within 90 min of ED arrival (preferred), or fibrinolysis within 30 min of ED arrival. Patients ineligible for fibrinolytic therapy should be considered for transfer to a PCI facility, even if arrival at the cardiac center will be delayed beyond the target time frame.

Maintenance of coronary artery patency is achieved by anticoagulation and administration of antiplatelet agents as discussed below. Treatment strategies to achieve immediate reperfusion and limit infarct size are listed in [Table 18-2](#), including drug doses.

<b>Antiplatelet agents</b>	
Aspirin	160 to 325 milligrams PO
Clopidogrel	Loading dose of 300 to 600 milligrams PO followed by 75 milligrams/d
<b>Anticoagulants</b>	
Heparin	60 units/kilogram bolus (max, 4000 U) followed by infusion of 12 units/kilogram/h (max, 1000 U/h) titrated to aPTT 1.5 to 2.5 times control
Enoxaparin (LMWH)	1 milligram/kilogram SC q12h
Bivalirudin	(dose prior to PCI) 0.75 milligram/kilogram IV bolus followed by 1.75 milligrams/kilogram/h infusion for duration of procedure
<b>Fibrinolytic agents</b>	
Streptokinase	1.5 million units over 60 min
Alteplase (tPA)	> 67 kilograms: 15 milligrams initial IV bolus; 50 milligrams infused over next 30 min; 35 milligrams infused over next 60 min < 67 kilograms: 15 milligrams initial IV bolus; 0.75 milligram/kilogram infused over next 30 min; 0.5 milligram/kilogram infused over next 60 min
Retepase (rPA)	10 units IV bolus followed by a second 10 unit IV bolus 30 min later
Tenecteplase (tPA)	Single bolus that is weight based, 30 to 50 milligrams
<b>Glycoprotein IIb/IIIa inhibitors</b>	
Abciximab	0.25 milligram/kilogram bolus followed by infusion of 0.125 microgram/kilogram/min (max, 10 micrograms/min) for 12 to 24 h
Eptifibatide	180 micrograms/kilogram bolus followed by infusion of 2.0 micrograms/kilogram/min for 72 to 96 h
Tirofiban	0.4 microgram/kilogram/min for 30 min followed by infusion of 0.1 microgram/kilogram/min for 48 to 96 h
<b>Other therapies</b>	
Nitroglycerin	SL: 0.4 milligram q5 min × 3 PRN for pain IV: start at 10 micrograms/min, titrate to 10% reduction in MAP if normotensive, 30% reduction in MAP if hypertensive
Morphine	2 to 5 milligrams IV q5 to 15 min PRN for pain
Metoprolol	50 milligrams PO
Atenolol	25 to 50 milligrams PO

\*Dosage may vary by indication such as presence or absence of ST-segment elevation

Key: LMWH = low-molecular-weight heparin, MAP = mean arterial pressure, max = maximum, PCI = percutaneous coronary intervention, PTT = partial thromboplastin time, q = every, rPA = recombinant plasminogen activator, SL = sublingual, tPA = tissue-type plasminogen activator.

**TABLE 18-2** Recommended Doses\* of Drugs Used in the Emergency Treatment of Acute Coronary Syndromes

1. All patients suspected of having cardiac pain should be placed on a cardiac monitor, receive an intravenous line, and supplemental oxygen. Dysrhythmias should be treated if their effect on heart rate exacerbates oxygen supply/demand imbalance, or if the dysrhythmia seems capable of electrical deterioration (eg, to a nonperfusing rhythm; see [Chapter 2](#)). The process of risk stratification using tools such as the TIMI Risk Score should be rapidly undertaken.
2. **Aspirin should be administered in a dose of 160 to 325 milligrams (chewed) in patients with suspected ACS, unless contraindicated or already taken by the patient.**
3. Oral and transdermal **nitroglycerin** (NTG) are useful in treating angina. A sublingual dose should be repeated twice, for a total of 3 tablets, administered at 2 to 5 min intervals. If there is no improvement with sublingual NTG, intravenous NTG should be started at 5 to 10 micrograms/min. IV NTG is recommended for MI or recurrent ischemia. The dose should be adjusted by 5 to 10 micrograms/min increments every 3 to 5 min, titrated to pain level and blood pressure reduction up to 200 micrograms/min maximum. NTG should be used cautiously in the setting of borderline-low blood pressure, as hypotension may worsen ischemia. NTG is contra-indicated in the setting of RV infarction, due to risk of hypotension related to loss of preload. A common side effect of NTG is headache.
4. **Morphine sulfate** can be used if there is uncontrolled ischemic chest discomfort despite NTG. Morphine may decrease cardiac output, and should be used with caution in the presence of hypotension, and in patients with inferior MI.
5. The thienopyridine **clopidogrel**, when added to ASA, reduces the composite risk of cardiovascular death, MI, or stroke. Thienopyridines bind to the ADP receptor and inhibit platelet aggregation. Clopidogrel should be considered in addition to standard care (ASA, anticoagulants) for patients with moderate to high-risk NSTEMI and STEMI, and in patients in whom PCI is planned. Clopidogrel is used without aspirin in patients allergic to aspirin. Clopidogrel increases risk of bleeding, and should be withheld at least 5 days before coronary artery bypass grafting (CABG). **Prasugrel** is an oral thienopyridine prodrug with no current indications for ED use.

6. **Unfractionated heparin (UFH)** is used for its anticoagulant properties. UFH has several disadvantages, including (1) the need for IV administration, (2) the requirement for frequent monitoring of the activated partial thromboplastin time (aPTT), (3) an unpredictable anticoagulant response in individual patients, (4) heparin-induced thrombocytopenia (HIT), and (5) increased risk of bleeding. Anticoagulation due to UFH can be reversed with protamine. The dosage is 1 milligram of **protamine** per 100 U of UFH infused in the previous 4 hours.
7. As compared to UFH, **low molecular weight heparins (LMWH)** offer greater bioavailability, lower protein binding, longer half-life, improved safety, and more reliable anticoagulant effect. They are administered in fixed subcutaneous doses and do not require laboratory monitoring. As compared to UFH, LMWH administration for ACS is associated with decreased ischemia and MI although there is an increase in minor bleeding complications. For patients with UA/NSTEMI, enoxaparin (a LMWH) or UFH are both reasonable choices for patients undergoing PCI revascularization. Improved outcomes are demonstrated with consistent therapy (use of a single antithrombin from the ED through the catheterization laboratory) and increased bleeding is seen when patients are switched from one antithrombin to another. In patients in whom CABG is planned, LMWH should be avoided (due to its half-life) in favor of UFH. In patients > 75 years of age, enoxaparin must be used with caution due to an increased risk of ICH. Enoxaparin dosing adjustments are recommended in patients with impaired renal function (creatinine clearance <30 mL/min).
8. Factor Xa inhibitors such as **fondaparinux**, a synthetic pentasaccharide, have similar efficacy to UFH in patients with UA/NSTEMI; bleeding risk is lower than that with enoxaparin. Current ACC/AHA guidelines consider fondaparinux an option as an antithrombin. In STEMI patients lacking renal impairment, fondaparinux may be considered for those patients treated with thrombolytics that are not fibrin specific (ie, streptokinase).
9. Direct thrombin inhibitors bind directly to thrombin in clot and are resistant to agents that degrade heparin. Comparison of bivalirudin with UFH found no outcomes benefit in NSTEMI patients, but less bleeding

occurred and no dosage adjustment is required in renal impairment. For patients with STEMI, **bivalirudin** may be considered as an alternative to UFH and GP IIb/IIIa inhibitors.

10. **Percutaneous coronary intervention (PCI)**, coronary angioplasty with or without stent placement, is the treatment of choice for the management of STEMI when PCI can be performed within 90 min of initial ED presentation. PCI may be offered to patients presenting to a non-PCI facility when prompt transfer can result in acceptable door-to-balloon times. **Early invasive therapy (PCI) within 48 hours is recommended in high-risk patients with UA/STEMI**, in patients with recurrent angina/ischemia, and in those who have elevated troponin, new or presumably new ST-segment depression, or high risk findings on stress testing. PCI is also more likely to be beneficial in the setting of depressed LV function, hemodynamic instability, sustained ventricular tachycardia, PCI within the previous 6 months, or prior CABG.
11. In treatment settings without timely access to PCI, **fibrinolytics** are indicated for patients with STEMI if time to treatment is <6 to 12 hours from symptom onset, and the ECG has at least 1 mm ST-segment elevation in two or more contiguous leads. The dosages of individual fibrinolytic agents are listed in [Table 18-2](#). STEMI patients who have received fibrinolytics should receive full-dose anticoagulants, started in the ED and maintained for a minimum of 48 hours. Similar efficacy and safety profiles have been demonstrated for tPA, rtPA, and TNK. Contraindications for fibrinolytics are listed in [Table 18-3](#). Before administering thrombolytics, informed consent should be obtained (with particular attention paid to an understanding of the risks). Arterial puncture should be avoided, as should venipuncture or central line placement in areas which are not readily compressible.



Absolute
Any prior intracranial hemorrhage
Known structural cerebral vascular lesion (eg, AVM)
Known malignant intracranial neoplasm (primary or metastatic)
Ischemic stroke within 3 months EXCEPT acute stroke within 3 h
Suspected aortic dissection
Active bleeding or bleeding diathesis (excluding menses)
Significant closed head trauma or facial trauma within 3 months
Relative
History of chronic, severe, poorly controlled hypertension
Severe uncontrolled hypertension on presentation (SBP > 180 mm Hg or DBP > 110 mm Hg)
History of prior ischemic stroke > 3 months, dementia, or known intracranial pathology not covered in contraindications
Traumatic or prolonged (> 10 min) CPR or major surgery (< 3 weeks)
Recent (within 2 to 4 weeks) internal bleeding
Noncompressible vascular punctures
Current use of anticoagulants: the higher the INR, the higher the risk of bleeding
Pregnancy
Active peptic ulcer
Streptokinase/anistreplase should not be given a second time

**TABLE 18-3** Contraindications to Fibrinolytic Therapy in ST-segment Elevation Myocardial Infraction

- a. **Tissue plasminogen activator (tPA)** is a naturally occurring human protein and is not antigenic. tPA is fibrin-specific and has a half-life of 5 min. When compared with traditional dosing, front-loaded tPA has been shown to have superior 90 min patency rates and reocclusion rates, with no increase in bleeding risk.
- b. **Retepase (rPA)** is a nonfibrin-specific deletion mutant of tPA with a prolonged half-life of 18 min (as compared to tPA's half-life of 3 min). Reteplase may have a faster time to perfusion. The main advantage of reteplase is that it is given as a (double) bolus rather than infusion.
- c. **Tenecteplase (TNK)** is a fibrin-specific substitution mutant of tPA that is given as a single weight-based bolus.
- d. **Streptokinase (SK)** activates circulating plasminogen, is not fibrin-specific, and is capable of generating an allergic reaction (minor: 5% to 5.7%, anaphylaxis: <0.2% to 0.7%). Hypotension occurs in up to 15% of patients and is usually responsive to fluids and slowing of SK infusion. Contraindications to SK include hypotension, prior SK administration (within 6 months), and streptococcal infection within a

year. SK's half-life is 23 min, but systemic fibrinolysis persists for 24 hours. Heparin must be given within 4 hours of starting SK.

e. The most significant complication of thrombolytics is hemorrhage, particularly ICH. Significant bleeding, especially internal, requires cessation of thrombolytics, heparin, and aspirin. Crystalloid and red blood cell infusion may be necessary. **Cryoprecipitate** (cryo) and **fresh frozen plasma** (FFP) may be used in an attempt to reverse fibrinolysis due to thrombolytics. Initially, 10 U of cryo are given, and fibrinogen levels are obtained. If the fibrinogen level is <1 gram/L, the dose of cryo should be repeated. If bleeding continues despite a fibrinogen > 1 gram/L, or if the fibrinogen level is <1 gram/L after 20 U of cryo, then 2 U of FFP should be administered. If this does not control hemorrhage, then platelets or antifibrinolytic agents (aminocaproic acid or tranexamic acid) are indicated.

12. Recent evidence shows no particular benefit to the early IV administration of  **$\beta$ -blockers** on cardiac rhythm, infarct size, reinfarction, or mortality. Oral  $\beta$ -blocker therapy does not need to be initiated in the ED unless there is a specific indication (eg, tachycardia), but  $\beta$ -blockers may be initiated within the first 24 hours of hospitalization for patients lacking contraindications, alternatives include metoprolol and atenolol among others.
13. **Glycoprotein IIb/IIIa (GP IIb/IIIa) antagonists** bind to platelets and inhibit their aggregation. Abciximab, eptifibatide, and tirofiban are currently available. There is no current evidence supporting the routine use of GP IIb/IIIa inhibitor therapy *prior* to angiography in patients with STEMI, and the use of these agents upstream is uncertain. Use of GP IIb/IIIa inhibitors should be guided by local interdisciplinary review of ongoing clinical trials, guidelines, and recommendations.

AMI patients with continued hemodynamic instability and pain or those who have not reperfused after administration of thrombolytics are candidates for rescue angioplasty (see [Chapter 19](#)). Emergent CABG may also be indicated for these patients. Patients in refractory cardiogenic shock should undergo emergent angioplasty. Intraaortic balloon pump or other LV-assisting devices may also be indicated for these patients.

Patients with AMI or UA who have ongoing chest pain, ECG changes, dysrhythmias, or hemodynamic compromise require cardiac intensive care. Patients with UA and resolved chest pain, normal or nonspecific ECG changes, and no complications should be admitted to a monitored bed. Certain patients, usually those with low risk, may undergo rule-out protocols in chest pain observation units (see [Chapter 20](#)).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 53, “Acute Coronary Syndromes: Unstable Angina, Myocardial Ischemia, and Infarction,” by Judd E. Hollander and Deborah B. Diercks.

# **CHAPTER 19**

## **Cardiogenic Shock**

Brian Hiestand

Cardiogenic shock occurs when there is insufficient cardiac output to meet the metabolic demands of the tissues. It is most commonly due to an acute myocardial infarction (AMI) that is extensive, impairs right ventricular contractility, or causes a rupture of a papillary muscle. Other etiologies to consider include cardiotoxic drug effects, infection (myopericarditis, endocarditis), and mechanical dysfunction (valvular disease, pulmonary embolism, cardiac tamponade, myocardial contusion). Early stabilization and treatment are critical, as mortality approaches 50% for AMI complicated by cardiogenic shock.

### **CLINICAL FEATURES**

The hallmark of all shock states is hypoperfusion. Cardiogenic shock generally presents with hypotension (systolic blood pressure [SBP] <90 mm Hg), although SBP may be greater than 90 mm Hg if there is preexisting hypertension or a compensatory increase in systemic vascular resistance. Sinus tachycardia is frequently seen, but may be absent in the setting of preceding calcium channel or beta-blockade. Evidence of hypoperfusion may include cool, mottled skin, oliguria, or altered mental status due to decreased cerebral perfusion and hypoxemia. Left ventricular failure may present with evidence of pulmonary edema: tachypnea, rales, wheezing, and frothy sputum. Jugular venous distention without pulmonary edema in the setting of hypotension should raise the suspicion of right ventricular failure due to infarction, tamponade, or pulmonary embolism. It is crucial to listen for the presence of a murmur that may represent acute valvular dysfunction (papillary muscle dysfunction or chordae rupture) or new ventricular septal defects as these findings may prompt life-saving surgery.

## **DIAGNOSIS AND DIFFERENTIAL**

The key task is to differentiate cardiogenic shock from shock due to hypovolemia or distributive causes (sepsis, neurogenic). A search for GI bleeding, obvious sources of infection, and focal neurologic deficits may establish an alternate diagnosis.

The first and most important test to order is an electrocardiogram (ECG). The ECG will aid in the detection of ischemia or infarction, arrhythmias, electrolyte abnormalities, or drug toxicity. ST-segment depression in the lateral leads should prompt consideration of a right ventricular infarction, which may occur without ST-segment elevation in the standard twelve lead ECG. Right ventricular infarction increases mortality from approximately 6% to 31%. A chest radiograph also should be obtained to look for pulmonary edema, abnormally wide mediastinum, or other abnormalities of the cardiac silhouette, or suggest alternate/confounding diagnoses such as pneumonia or pneumothorax.

There is no single laboratory test that is diagnostic for shock. A complete blood count and chemistries (including liver function tests) should be obtained. In the absence of ST-segment elevation, cardiac markers such as troponin may establish the diagnosis of non-ST-segment elevation MI (NSTEMI); in addition, they add prognostic value in non-AMI states such as acute heart failure and sepsis. Given their high negative predictive value, natriuretic peptides, such as BNP or n-terminal pro-BNP, should prompt a search for a noncardiac etiology if normal. Serum lactate will indicate the degree of hypoperfusion present. Blood gas measurements will provide insight into acid-base status and CO<sub>2</sub> retention. The decision to obtain toxicology testing should be guided by the specific clinical situation. Transthoracic echocardiography (TTE) is a useful bedside tool when evaluating a patient in shock without a clear etiology.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Airway management, circulatory stabilization, and arrangements for definitive cardiac care must occur simultaneously. Cardiology and cardiac surgery should be consulted early. Transfer should be arranged if indicated.

1. Supplemental oxygen should be provided. Endotracheal intubation should be considered as needed. Noninvasive positive pressure ventilation may provide temporary support, but most patients in cardiogenic shock are simply too ill to benefit from this.
2. Intravenous access should be obtained. Cardiac rhythm and pulse oximetry monitoring should be initiated. Rhythm disturbances, hypoxemia, hypovolemia, and electrolyte abnormalities should be identified and treated.
3. Early revascularization is required for cardiogenic shock due to ischemia. Percutaneous coronary intervention (PCI) is superior to fibrinolysis in the setting of cardiogenic shock. However, when PCI is not available (or prolonged transfer times are anticipated) fibrinolysis is superior to supportive measures alone.
4. Especially with concomitant right ventricular ischemia, anti-anginal therapies may precipitate cardiovascular collapse. For chest pain, titrated intravenous **nitroglycerin** 5 to 100 micrograms/min or **morphine sulfate** given in 2 milligram increments may be administered with caution. *Do not give  $\beta$ -blockers in cardiogenic shock.*
5. For mild hypotension without pulmonary congestion, a small fluid challenge (250 to 500 mL) may be considered. For hypotension in the setting of right ventricular ischemia, a more robust fluid resuscitation is warranted.
6. **Norepinephrine** may be considered for severe hypotension as a vasopressor and positive inotrope. An infusion should begin at 2 micrograms/min and titrated to the desired effect.
7. For mild to moderate hypotension without hypovolemia, **dobutamine** 2.5 to 20.0 micrograms/kilogram/min should be administered. Dobutamine may cause peripheral vasodilatation, requiring the concomitant use of **dopamine** 2.5 to 20.0 micrograms/kilogram/min, titrated to the desired effect with the lowest dose possible.
8. **Milrinone** may be considered as a positive inotrope. Start with a loading dose of 50 micrograms/kilogram IV over 10 min followed by an infusion

of 0.5 microgram/kilogram/min.

9. As a temporizing measure, intraaortic balloon pump counterpulsation (if available) should be considered to decrease afterload and to augment coronary perfusion.
10. In the setting of acute mitral regurgitation, afterload reduction via intravenous **sodium nitroprusside** 0.5 to 10.0 micrograms/kilogram/ min should be combined with inotropic support via **dobutamine** 2.5 to 20.0 micrograms/kilogram/min. An intraaortic balloon pump may also be indicated to augment forward blood flow (contraindicated in severe aortic regurgitation).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 54, “Cardiogenic Shock,” by James Edward Weber and W. Frank Peacock.

## CHAPTER 20

# Low Probability Acute Coronary Syndromes

Chadwick D. Miller

Patients with chest pain or other symptoms suggesting coronary ischemia require stratification based upon the probability of acute coronary syndrome (ACS) for proper treatment and disposition. This chapter discusses the features of low probability ACS, or possible ACS. By definition, patients classified into this group have no objective evidence of acute coronary ischemia or infarction—no characteristic electrocardiogram (ECG) ST-segment elevation or depression, and normal levels of cardiac markers.

## CLINICAL FEATURES

A key determination by the emergency physician is whether to pursue further evaluation for possible ACS. Currently 3% to 6% of patients thought to have noncardiac chest pain or a clear-cut alternative diagnosis will have a short-term adverse cardiac event.

The clinical features of patients with possible ACS are the same as discussed in [Chapter 17](#) “Chest Pain: Cardiac or Not.” High-risk historical features include chest pain with any of the following descriptors: radiating, occurs with exertion, described as pressure, similar to prior cardiac pain, or is accompanied by nausea or diaphoresis. However, even patients without high-risk features have some risk of ACS. Therefore absence of high-risk features should not solely be used to exclude ACS. Significant coronary disease is rare in patients <30 years old but youth does not completely eliminate ACS as a cause of acute chest pain. The physical exam should focus on excluding alternative diagnoses and detecting signs of cardiac failure.

A previous negative cardiac test should not prevent an appropriate evaluation for ACS in a patient with a concerning history or ischemic ECG findings. Plaque rupture is a major cause of ACS and commonly occurs in



lesions that were previously nonobstructive. Previous stress testing results cannot determine whether the patient's current symptoms represent new ischemia from a recent plaque rupture. In contrast, previous cardiac catheterization results can be of benefit in determining whether a patient should undergo stress testing after exclusion of myocardial infarction. It is unlikely that a patient with previously normal or near-normal coronary arteries has developed significant epicardial stenosis within 2 years of the procedure.

## **DIAGNOSIS AND DIFFERENTIAL**

The evaluation of patients with possible ACS can be conceptualized into a primary and secondary assessment. The primary evaluation must detect patients with ST-segment elevation that require emergent revascularization and distinguish between patients with definite ACS, possible ACS, and those with symptoms that are definitely not ACS. Alternative causes of chest pain should be considered (see [Chapter 17](#)).

The primary evaluation should include a history, physical examination, ECG, chest radiography, and cardiac biomarkers if ACS remains in the differential diagnosis. Serial ECGs should be obtained in patients with ongoing symptoms. All available data should be used to create a composite picture for decision making. Some have calculated that when the pretest probability of ACS is  $\leq 2\%$ , further testing is not indicated. Others have suggested a threshold of  $<1\%$ .

At the conclusion of the primary evaluation, patients should be classified as having acute myocardial infarction (AMI), possible acute ischemia, or definitely not ischemia. Patients with possible acute ischemia are further stratified into high, intermediate, and low risk for adverse events based on the pattern of symptoms, clinical features, and ECG findings. ([Table 20-1](#)) The TIMI risk score may also assist with this assessment of risk, but should not be used to determine if a patient is above or below the testing threshold.

<b>I. Acute myocardial infarction: immediate revascularization candidate</b>
<b>II. Probable acute ischemia: high risk for adverse events</b> Clinical instability, ongoing pain thought to be ischemic, pain at rest associated with ischemic ECG changes
<b>III. Possible acute ischemia: intermediate risk for adverse events</b> History suggestive of ischemia with absence of high risk features, and any of the following: pain at rest, new onset of pain, crescendo pattern of pain, ischemic pattern on ECG not associated with pain (may include ST depression < 1 mm or T-wave inversion greater than 1 mm)
<b>IV. Possible acute ischemia: low risk for adverse events</b> History not strongly suggestive of ischemia, ECG normal, unchanged from previous, or nonspecific changes
<b>V. Definitely not ischemia: very low risk for adverse events</b> Clear objective evidence of non-ischemic symptom etiology, ECG normal, unchanged from previous, or nonspecific changes, clinician estimate of ACS probability $\leq 2\%$

**TABLE 20-1** Risk Stratification Scheme for Patients With Possible Acute Coronary Syndrome

The secondary assessment can be conducted in an observation unit or in the inpatient arena. This assessment should exclude both components of ACS, myocardial infarction and unstable angina. Myocardial infarction is excluded through the use of serial troponin measurements to detect myocardial necrosis. Serum troponin levels can take as long as 8 hours from the time of infarction to become elevated. Therefore, a cardiac biomarker approach should take into account the time from symptom onset and generally should include multiple measurements. A traditional approach is to obtain troponin measurements at the time of arrival and 6 to 8 hours after arrival. An interim 3 to 4 hours measurement may be added if desired.

Normal serial myocardial marker measurements reduce the likelihood of AMI but do not exclude unstable angina, which still puts the patient at high risk for a subsequent adverse cardiac event. Therefore, patients with possible ACS should undergo some form of objective cardiac testing. Objective cardiac testing defines either the patient's coronary anatomy, cardiac function, or both. Common modalities used include stress electrocardiography, stress echocardiography, resting and/or stress nuclear imaging, stress cardiac magnetic resonance imaging (MRI), and computed tomography coronary angiography (CTCA). Most patients undergo testing during the initial encounter. However, outpatient testing is an option for low-risk patients in whom AMI has been excluded. This option is most

useful in reliable patients presenting to a facility where a mechanism exists to arrange this testing.

Selection of an objective cardiac testing approach should be guided by general principles discussed below and will also need to take into account the modalities available at each institution.

1. The first determination is whether stress testing or coronary imaging with CTCA is desired. The most promising application of CTCA is the exclusion of coronary disease in low-risk patients, and its use in this population is supported in the 2007 ACC/AHA Guidelines. Upcoming data will further define the role of this modality. At present, a functional assessment with stress testing is preferable in patients likely or known to have coronary atherosclerosis.
2. If a stress testing approach is desired, the method of stress (exercise or pharmacologic) should be determined next. In general, patients able to exercise should undergo an exercise-based testing modality. Those unable to exercise should receive a pharmacologic stress strategy.
3. Finally, the method of cardiac assessment during stress testing should be selected. Options include electrocardiography, nuclear imaging, echocardiography, or magnetic resonance imaging-based strategies. Selection from these strategies is often based on local expertise at the study institution, testing availability, matching pretest probability of disease with sensitivity of the imaging modality, and minimizing radiation exposure. ECG-based exercise treadmill testing is the least costly and is widely available, but has the lowest sensitivity (68%) of the imaging options and therefore should not be used in patients with high pretest probability for disease. Further, ECG-based exercise treadmill testing should not be used in patients with abnormal baseline ECGs due to difficulties in interpretation. Stress echocardiography has the advantages of no radiation exposure, improved sensitivity (80%), and wide availability. Nuclear imaging is also widely available, allows assessment of myocardial perfusion, and has high accuracy, but is associated with radiation exposure and radioisotope related delays. Cardiac MRI is also highly accurate and does not expose patients to radiation, but is less available.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

The treatment of patients with possible ACS is confined to the short period during which the care provider is uncertain whether the patient has ACS or some alternative diagnosis. Once the diagnosis of ACS is confirmed, treatment is started for that disease process ([Chapter 18](#)). When an alternative diagnosis is confirmed and ACS is excluded, treatment should begin for the alternative diagnosis. While testing is ongoing for possible ACS, patients should receive supplemental oxygen plus the following treatment:

1. **Aspirin** 160 to 325 milligrams PO.
2. **Nitroglycerin** 0.4 milligram spray or sublingual.
3. If symptoms continue, administer anti-ischemic therapy using  $\beta$ -blockers (**metoprolol** 25 to 50 milligrams PO in the first 24 hours) and/or morphine sulfate 1 to 5 milligrams IV. There are several contraindications to  $\beta$ -blockade including heart failure, low cardiac output, heart blocks, active reactive airway disease, tachycardia, and hypotension. (See [Chapter 18](#) “Acute Coronary Syndrome,” for further details.)
4. In addition to the core therapy above, there are other adjunctive treatment options for patients at intermediate risk. The decision to administer these medications should be institution specific, balanced with the patient’s bleeding risk and potential benefit, and determined through multidisciplinary discussions. These options include (a) dual antiplatelet therapy: a common regimen is **clopidogrel** 300 to 600 milligrams PO in addition to aspirin, (b) anti-thrombin therapy: common regimens are **heparin** 60 units/kilogram IV bolus (maximum bolus 4000 units) 12 units/kilogram/h IV infusion (maximum infusion 1000 units/h) or **enoxaparin** 1 milligram/kilogram SC every 12 hours.
5. Patients with negative serial cardiac markers, without diagnostic ECG changes, and who have normal objective cardiac testing are unlikely to have ACS as a cause of their symptoms. Consideration should be given to alternative life-threatening causes with further evaluation conducted as

appropriate. Those with positive cardiac markers, diagnostic ECG changes, or diagnostic testing supporting ACS are admitted to the hospital for cardiology care. Those with nondiagnostic testing should be handled on a case-by-case basis and most should be discussed with a cardiologist.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 55, “Low Probability Acute Coronary Syndromes,” by Chadwick D. Miller.

# CHAPTER 21

## Syncope

Michael G. Mikhail

Syncope accounts for up to 2% of all ED visits and 6% of hospital admissions. *Syncope* is defined as a transient loss of consciousness accompanied by loss of postural tone, followed by complete resolution without intervention. Although syncope typically is a benign vasovagal event, it may represent a life-threatening dysrhythmia/condition, particularly in the elderly. In up to half of syncope cases presenting to the ED, there is no definite etiology established for the syncopal episode.

### CLINICAL FEATURES

Syncope is most commonly reflex mediated. A sympathetic response to stress is suddenly withdrawn, leading to pronounced vagal tone with hypotension or bradycardia. The hallmark of vasovagal syncope is the occurrence, in a standing patient, of a prodrome of dizziness, nausea, pallor, diaphoresis, and diminished vision. The history should include a search for stimuli (eg, phlebotomy, injury, fear) known to be associated with vasovagal syncope. Carotid sinus hypersensitivity, which is more common in the elderly, is suggested by a history of presyncopal shaving, head-turning, or wearing of a constricting collar. Carotid sinus hypersensitivity should be considered in patients with syncope that is recurrent despite a negative cardiac workup. In situational syncope, the autonomic reflexive response may result from a specific physical stimulus such as micturition, defecation, or extreme coughing.

Orthostatic syncope occurs when a sudden change in posture after prolonged recumbence is associated with inadequate compensatory increases in heart rate and peripheral vascular resistance. Orthostatic syncope is often due to autonomic dysfunction, which has a myriad of potential causes (eg, peripheral neuropathy, spinal cord injury, Shy-Drager

syndrome). Any disorder causing volume depletion may also cause orthostatic syncope.

Cardiac syncope is due to a dysrhythmia or a structural cardiopulmonary lesion. Tachydysrhythmias (eg, ventricular tachycardia, torsades des pointes, supraventricular tachycardia) are common causes of syncope, but the most likely finding on ED evaluation is incidental bradycardia. Syncope from dysrhythmias is typically sudden and without prodrome. Drug- or exercise-induced vasodilation may cause syncope as underlying cardiac structural abnormalities are unmasked. In the elderly, this scenario is most commonly due to aortic stenosis, a diagnosis which must be rigorously investigated as a cause of syncope. In the young patient, the cardiac structural abnormality is most commonly hypertrophic cardiomyopathy. Approximately 10% of patients with pulmonary embolism will have pulmonary outflow obstruction that leads to syncope.

Cerebrovascular disorders are rare as a cause of syncope. If brainstem ischemia is the cause, the patient usually reports other posterior circulation deficits (eg, diplopia, vertigo, nausea) associated with the “drop attack.” If patients report that upper extremity exercise preceded the event, there may be intermittent obstruction of the brachiocephalic or subclavian artery (ie, subclavian steal syndrome). Subarachnoid hemorrhage may also present with syncope, which is likely due to a transient rise in intracranial pressure.

Because of poor autonomic responses and multiple medications, the elderly are particularly prone to syncope, which is usually due to cardiac causes. Cardiovascular responses to orthostatic or vasodilatory challenges may be blunted by antihypertensive agents such as  $\beta$ -blockers and calcium channel antagonists. Cardiovascular medications may also cause conduction abnormalities or life-threatening dysrhythmias. Diuretics also contribute to the risk of orthostatic hypotension, due to their volume-depleting effect. Syncope is a dual threat to the elderly, in that causes are more serious and injury risks are higher.

Analysis of the San Francisco syncope cohort of 1418 consecutive patients led to generation of a decision rule that may contribute to risk stratification decision-making in syncope patients. Presence of any one of five variables identified a patient at risk for serious outcome, with 89% sensitivity and 52% specificity for death at 1 year: an abnormal ECG, a

complaint of shortness of breath, hematocrit less than 30%, systolic blood pressure <90 mm Hg on arrival, or a history of congestive heart failure.

## **DIAGNOSIS AND DIFFERENTIAL**

Although an etiology for the syncopal episode may be difficult to establish, the most important tools in the syncope workup are a comprehensive history, physical examination, and ECG. From there, risk stratification is the most practical approach. The history should be directed to high-risk factors, including age, medications, and prodromal events. Sudden events that occur without warning suggest dysrhythmias; exertion may imply a structural cardiopulmonary lesion. Associated symptoms are helpful for indicating a syncopal episode's etiology as cardiac (eg, palpitations and chest pain) or neurologic (eg, vertigo and focal weakness). Back or abdominal pain may suggest a leaking abdominal aortic aneurysm or ruptured ectopic pregnancy. Single-vehicle crashes or trauma in the absence of defensive injuries should prompt an investigation for syncope as a precipitating event. The medical history is useful in revealing likely cardiac or psychiatric (eg, hyperventilation) causes for syncope. When present, family history of cardiac disease or sudden death may also be informative.

Physical examination may occasionally reveal the cause of syncope. The cardiac examination may uncover a ventricular flow obstruction. A cardiac murmur may represent aortic stenosis or hypertrophic cardiomyopathy. An accentuated pulmonary gallop can suggest pulmonary embolism. A complete neurologic assessment and rectal examination (with Hemoccult testing) may yield further secondary causes for syncope.

An ECG should be obtained to assess for signs of ischemia or dysrhythmia. Suggestive ECG findings include prolonged QT interval (indicating propensity for torsades des pointes) or PR interval shortening with delta wave (ie, Wolf-Parkinson-White syndrome). Prolonged monitoring may show a transient, but recurring, dysrhythmia.

Laboratory testing should be selective. A hematocrit may explain orthostatic syncope and direct further workup (eg, for occult gastrointestinal bleeding). Women of childbearing potential warrant a pregnancy test.



Although not recommended routinely, electrolytes may show a decreased bicarbonate after a seizure. Electrolytes are also indicated in patients with weakness or irritable myocardium (eg, as can occur with hypomagnesemia).

There are a variety of bedside tests that may provide clues in selected cases. A significant ( $> 20$  mm Hg) blood differential between upper extremities suggests subclavian steal syndrome. Carotid sinus massage can be performed in selected patients (without bruits) if one suspects carotid sinus hypersensitivity; a positive test requires reproduction of symptoms as well as bradycardic or hypotensive response. *Orthostatic hypotension* (autonomic instability from drugs or disease) is defined as a systolic blood pressure drop of at least 20 mm Hg upon standing; this diagnosis should only be made if postural hemodynamic changes are associated with reproduction of symptoms.

Seizure is the most common disorder mistaken for syncope. Some hallmarks of seizure (eg, tongue biting and incontinence) may also be seen with convulsive syncope, which occurs when cerebral anoxia causes brief clonus. The most helpful factor in differentiating syncope from seizures is the latter's postictal confusion and slow return to normal consciousness.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

By definition, syncope results in spontaneous recovery of consciousness. Therefore, the main goal of ED care is to identify those patients at risk for further medical problems. Patients can be categorized into 1 of 3 classes after a careful history, physical examination, and ECG:

1. *If the diagnosis is established (eg, pulmonary embolus, ectopic pregnancy, GI bleed)* then the patient can be appropriately managed by directing attention to the underlying cause of the syncopal event. Patients for whom a life-threatening etiology is identified, including those with neurologic or cardiac causes, warrant admission.
2. *Patients with unclear diagnosis who are high-risk* are those for whom there is concern about sudden cardiac death or ventricular dysrhythmia.

As suggested by the San Francisco Syncope Rule, concerning findings placing patients in the high-risk category include: abnormal ECG, complaint of shortness of breath, systolic blood pressure of <90 mm Hg on arrival, hematocrit less than 30%, age older than 45 years, or a history of ventricular dysrhythmia or congestive heart failure. Admission of these patients is warranted, for observation and to facilitate expedited workup (usually focused on cardiac etiologies).

3. *Patients with unclear diagnosis who are low-risk* are unlikely to have a cardiac etiology for their syncope. These patients lack the high-risk criteria noted above. They are young (<45 years), have few comorbidities, and have a normal physical examination and ECG. In this group, syncope is usually vasovagal and no further workup is required if the episode is isolated. Low-risk patients can be safely discharged home with instructions to return for any recurrence of presyncopal symptoms. Worrisome or recurrent cases may benefit from further outpatient workup including Holter or loop-event monitoring. These patients should also be advised that, pending further outpatient workup, they are considered at-risk for syncope and should modify behavior accordingly (eg, avoid driving).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 56, “Syncope,” by James Quinn.

## **CHAPTER 22**

# **Congestive Heart Failure and Acute Pulmonary Edema**

Lori Whelan

Acute pulmonary edema is one of the most critical presentations of the many clinical effects of heart failure. The most common precipitating factors of heart failure are atrial fibrillation, acute myocardial infarction or ischemia, discontinuation of medications (diuretics), increased sodium load, drugs that impair myocardial function, and physical overexertion.

## **CLINICAL FEATURES**

Patients with acute pulmonary edema usually present with symptoms of left ventricular heart failure, severe respiratory distress, frothy pink or white sputum, moist pulmonary rales, and a third heart sound (S<sub>3</sub>) or fourth heart sound (S<sub>4</sub>). Patients frequently are tachycardic and hypertensive. Cardiac dysrhythmias, such as atrial fibrillation or premature ventricular contractions, are common. There may be a history of exertional dyspnea, paroxysmal nocturnal dyspnea, or orthopnea. Patients with right ventricular heart failure have dependent edema of the extremities and may have jugular venous distention, hepatic enlargement, and a hepatojugular reflex. The traditional distinction between right and left heart failure does not have great bearing on ED management, as volume overload and respiratory distress will be approached in the same manner. However, consideration must be given to patients in whom there is a suspicion of valvular pathology or acute right ventricular infarction.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of acute pulmonary edema is made with clinical findings and the chest x-ray. The severity of illness may dictate need for a portable (anterior-posterior) chest x-ray be taken. Additional tests that assist in management include an electrocardiogram, serum electrolytes, serum urea nitrogen, creatinine, complete blood cell count, arterial or venous blood gas, B-type natriuretic peptide, and cardiac markers (eg, troponin). The diagnosis of right-side heart failure is made clinically. In left-side heart failure, the chest x-ray will show enlargement of the cardiac silhouette.

In the differential diagnosis, consider the common causes of acute respiratory distress: asthma, chronic obstructive pulmonary disease, pneumonia, pulmonary embolus, allergic reactions, and other causes of respiratory failure. Also in the differential are other causes of noncardiogenic pulmonary edema, such as drug-related alveolar capillary damage or acute respiratory distress syndrome.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The treatment of patients in acute pulmonary edema includes oxygen, preload reducers, diuretics, afterload reducers, and inotropic agents.

1. Oxygen therapy, up to 100% delivered by non-rebreather mask should be administered to achieve an oxygen saturation of at least 95% by pulse oximetry.
2. If hypoxia persists despite oxygen therapy, or if the patient is showing signs of respiratory distress (eg, tripod stature, accessory muscle use, inability to speak), **continuous positive airway pressure (CPAP)** or **biphasic positive airway pressure (BiPAP)** should be applied.
3. Immediate intubation is indicated for unconscious or visibly tiring patients.
4. **Nitroglycerin** 0.4 milligram should be administered sublingually (may be repeated every 1 to 5 min). If the patient does not respond, or if the electrocardiogram shows ischemia, nitroglycerin 0.4

microgram/kilogram/ min should be initiated as an intravenous drip and titrated rapidly to BP and symptom reduction.

5. After nitrates, an intravenous diuretic can be administered, such as **furosemide** 40 to 80 milligrams intravenously, **bumetanide** 0.5 to 1 milligram intravenously, or torsemide 10 milligrams intravenously. Electrolytes (especially serum potassium) should be monitored.
6. Transient hypotension may develop after nitroglycerin is started. This may be due to marked clinical improvement, and should improve with decreasing the dose or cessation of nitroglycerin. If hypotension persists, initiate a fluid bolus (250 to 1000 cc) and be suspicious of a right ventricular infarction, valvular pathology (severe aortic stenosis), hypovolemia, or recent use of medications for erectile dysfunction.
7. For patients with resistant hypertension or those who are not responding well to nitroglycerin, **nitroprusside** may be used, starting at 0.3 microgram/ kilogram/min and titrated.
8. For hypotensive patients or patients in need of additional inotropic support, see [Chapter 19](#) “Cardiogenic Shock,” for management.
9. Coexisting dysrhythmias (see [Chapter 2](#)) or electrolyte disturbances (see [Chapter 4](#)) should be treated, and those therapies that impair the inotropic state of the heart should be avoided.

Acute mitral valve or aortic valve regurgitation should be considered, especially if the heart is of normal size, because the patient may need emergency surgery. Until excluded, acute myocardial infarction should be considered as the cause of pulmonary edema exacerbation. Because the initial electrocardiogram may fail to demonstrate ischemic changes, repeat electrocardiograms are indicated for those patients with initially normal tracings, who fail to improve.

**Morphine** can be given (2 to 5 milligrams intravenously) and repeated as needed for pain control. Its use is controversial, however, and may cause respiratory depression. For anuric (dialysis) patients, sorbitol and phlebotomy may have some benefit; however, dialysis is the treatment of choice in these patients who prove resistant to nitrates.

Patients with acute pulmonary edema may require admission to the intensive care unit for ongoing invasive hemodynamic monitoring. In the presence of new dysrhythmias, uncontrolled hypertension, or suspected

myocardial infarction, the patient should be admitted to a telemetry bed for evaluation and optimization of drug therapy.

Long-term treatment of congestive heart failure includes dietary salt reduction, chronic use of diuretics such as furosemide, afterload reducers such as angiotensin-converting enzyme inhibitors,  $\beta$ -blockers, or digoxin. Patients with an exacerbation of chronic congestive heart failure without chest pain or complicating factors who respond to diuretics may be discharged home if close follow-up is arranged.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 57, "Congestive Heart Failure and Acute Pulmonary Edema," by W. Frank Peacock.

# CHAPTER 23

## Valvular Emergencies

Boyd Burns

Typically the diagnosis of valvular disease and dysfunction has been previously established but emergency physicians must be alert to the presenting signs and symptoms to aid the undiagnosed patient, and with bedside echocardiography becoming more common, initial diagnosis will occur more commonly in the ED.

### MITRAL STENOSIS

#### Clinical Features

As with all valvular diseases, exertional dyspnea is the most common presenting symptom (80% of patients with mitral stenosis). In the past, hemoptysis was the second most common presenting symptom, but it is less common now with earlier recognition and treatment. Systemic emboli may occur and result in myocardial, kidney, central nervous system, or peripheral infarction. Most patients eventually develop atrial fibrillation because of progressive dilation of the atria. The classic murmur of mitral stenosis and associated signs are listed in [Table 23-1](#).

Valve Disorder	Murmur	Heart Sounds and Signs
Mitral stenosis	Mid-diastolic rumble, crescendos into S <sub>1</sub>	Loud snapping S <sub>1</sub> , apical impulse is small, tapping due to under-filled ventricle
Mitral regurgitation	Acute: harsh apical systolic murmur that starts with S <sub>1</sub> and may end before S <sub>2</sub> . Chronic: high-pitched apical holosystolic murmur that radiates into S <sub>2</sub>	S <sub>3</sub> and S <sub>4</sub> may be heard
Mitral valve prolapse	Click may be followed by a late systolic murmur that crescendos into S <sub>2</sub>	Midsystolic click; S <sub>2</sub> may be diminished by the late systolic murmur
Aortic stenosis	Harsh systolic ejection murmur	Paradoxical splitting of S <sub>2</sub> ; S <sub>3</sub> and S <sub>4</sub> may be present; pulse of small amplitude; pulse has a slow rise and sustained peak
Aortic regurgitation	High-pitched blowing diastolic murmur immediately after S <sub>2</sub>	S <sub>3</sub> may be present; wide pulse pressure

Key: S<sub>1</sub> = first heart sound, S<sub>2</sub> = second heart sound, S<sub>3</sub> = third heart sound, S<sub>4</sub> = fourth heart sound.

**TABLE 23-1** Comparison of Heart Murmurs, Sounds, and Signs

## Diagnosis and Differential

The electrocardiogram (ECG) may demonstrate notched or diphasic P waves and right axis deviation. On the chest radiograph, straightening of the left heart border, indicating left atrial enlargement, is a typical early radiographic finding. Eventually, findings of pulmonary congestion are noted: redistribution of flow to the upper lung fields, Kerley B lines, and an increase in vascular markings. The diagnosis of mitral stenosis should be confirmed with echocardiography or consultation with a cardiologist. The urgency for an accurate diagnosis and appropriate referral depends on the severity of symptoms.

## Emergency Department Care and Disposition



1. The medical management of mitral stenosis includes intermittent diuretics, such as **furosemide** 40 milligrams intravenously (IV), to alleviate pulmonary congestion, the treatment of atrial fibrillation (see [Chapter 2](#)), and anticoagulation (international normalized ratio [INR] goal of 2:3) for patients at risk for arterial embolic events.
2. Frank hemoptysis may occur in the setting of mitral stenosis and pulmonary hypertension. Bleeding may be severe enough to require blood transfusion, consultation with a thoracic surgeon, and emergency surgery.

## MITRAL INCOMPETENCE

### Clinical Features

Acute mitral incompetence secondary to rupture of the chordae tendineae or papillary muscles presents with dyspnea, tachycardia, and pulmonary edema. Patients may quickly deteriorate to cardiogenic shock or cardiac arrest. Intermittent mitral incompetence usually presents with acute episodes of respiratory distress due to pulmonary edema and may be asymptomatic between attacks. Chronic mitral incompetence may be tolerated for years or even decades. The first symptom is usually exertional dyspnea, sometimes prompted by atrial fibrillation. If patients are not anticoagulated, systemic emboli occur in 20% and are often asymptomatic. The classic murmur and signs of mitral incompetence are listed in [Table 23-1](#).

### Diagnosis and Differential

In acute rupture, the ECG may show evidence of acute inferior wall infarction (more common than anterior wall infarction in this setting). On chest radiography, acute mitral incompetence from papillary muscle rupture may result in a minimally enlarged left atrium and pulmonary edema, with less cardiac enlargement than expected. In chronic disease, the ECG may demonstrate findings of left atrial and left ventricular hypertrophy (LVH).

On chest radiography, chronic mitral incompetence produces left ventricular and atrial enlargements that are proportional to the severity of the regurgitant volume.

Echocardiography is essential to make the diagnosis with certainty, and bedside technique may be mandatory in the acutely ill patient. However, transthoracic echocardiography may underestimate lesion severity, and the transesophageal technique should be undertaken as soon as the patient is adequately stable to leave the department. In stable patients, echocardiography can be scheduled electively.

## **Emergency Department Care and Disposition**

1. Pulmonary edema should be treated initially with oxygen, noninvasive ventilation, diuretics such as **furosemide** 40 milligrams IV, and nitrates as the patient tolerates. Intubation is reserved for those patients with who fail clinically despite these measures.
2. Nitroprusside increases forward output by increasing aortic flow and partly restoring mitral valve competence as left ventricular size diminishes. **Nitroprusside** 5 micrograms/kilogram/min IV can be started unless the patient is hypotensive. There may be a subset of patients whose mitral regurgitation is worsened by nitroprusside (those patients who respond with a dilation of the regurgitant orifice); thus, careful monitoring is essential.
3. Hypotensive patients should receive inotropic agents such as **dobutamine** 2.5 to 20 micrograms/kilogram/min in addition to nitroprusside.
4. Aortic balloon counter pulsation increases forward flow and mean arterial pressure and diminishes regurgitant volume and left ventricular filling pressure and can be used to stabilize a patient while awaiting surgery.
5. Emergency surgery should be considered in cases of acute mitral valve rupture.

## **MITRAL VALVE PROLAPSE**

## **Clinical Features**

Most patients are asymptomatic. Symptoms include atypical chest pain, palpitations, fatigue, and dyspnea unrelated to exertion. The abnormal heart sounds are listed in [Table 23-1](#). In patients with mitral valve prolapse without mitral regurgitation at rest, exercise provokes mitral regurgitation in about one-third of patients and predicts a higher risk for morbid events.

## **Diagnosis and Differential**

The ECG and chest x-ray are usually normal. Echocardiography is recommended to confirm the clinical diagnosis of mitral valve prolapse and to identify any associated mitral regurgitation. Echocardiography or consultation with a cardiologist can be performed on an outpatient basis.

## **Emergency Department Care and Disposition**

1. Initiating treatment for mitral valve prolapse is rarely required for patients seen in the emergency department. Patients with palpitations, chest pain, or anxiety frequently respond to  $\beta$ -blockers such as **atenolol** 25 milligrams daily.
2. Avoiding alcohol, tobacco, and caffeine also may relieve symptoms.

# **AORTIC STENOSIS**

## **Clinical Features**

The classic triad is dyspnea, chest pain, and syncope. Dyspnea is usually the first symptom, followed by paroxysmal nocturnal dyspnea, syncope on exertion, angina, and myocardial infarction. The classic murmur and

associated signs of aortic stenosis are listed in [Table 23-1](#). Blood pressure is normal or low, with a narrow pulse pressure.

Brachioradial delay is an important finding in aortic stenosis. The examiner palpates simultaneously the right brachial artery of the patient with the thumb and the right radial artery of the patient with the middle or index finger. Any palpable delay between the brachial artery and radial artery is considered abnormal.

## **Diagnosis and Differential**

The ECG usually demonstrates criteria for LVH and, in 10% of patients, left or right bundle branch block. The chest radiograph is normal early, but eventually LVH and findings of congestive heart failure are evident if the patient does not have valve replacement.

Echocardiography should be undertaken to confirm the suspected diagnosis of aortic stenosis and in the hospital if the murmur is associated with syncope.

## **Emergency Department Care and Disposition**

1. Patients presenting with pulmonary edema can be treated with oxygen and diuretics such as **furosemide** 40 milligrams IV, but nitrates should be used with caution because reducing preload may cause significant hypotension. Nitroprusside is not well tolerated in patients with aortic stenosis.
2. New onset atrial fibrillation may severely compromise cardiac output, especially at higher rates and will require anticoagulation and cardioversion in the appropriate clinical setting.
3. Patients with profound symptoms secondary to aortic stenosis such as syncope are usually admitted to the hospital.

## **AORTIC INCOMPETENCE**

## **Clinical Features**

In acute disease, dyspnea is the most common presenting symptom, seen in 50% of patients. Many patients have acute pulmonary edema with pink frothy sputum. Patients may complain of fever and chills if endocarditis is the cause. Dissection of the ascending aorta typically produces a “tearing” chest pain that may radiate between the shoulder blades. The classic murmur and signs of aortic incompetence are listed in [Table 23-1](#). In the acute state, chest radiography demonstrates acute pulmonary edema with less cardiac enlargement than expected.

In the chronic state, about one-third of patients will have palpitations associated with a large stroke volume and/or premature ventricular contractions. In the chronic state, signs include a wide pulse pressure with a prominent ventricular impulse, which may be manifested as head bobbing. “Water hammer pulse” may be noted; this is a peripheral pulse that has a quick rise in upstroke followed by a peripheral collapse. Other classic findings may include accentuated precordial apical thrust, pulsus bisferiens, Duroziez sign (a to-and-fro femoral murmur), and Quincke pulse (capillary pulsations visible at the proximal nailbed while pressure is applied at the tip).

## **Diagnosis and Differential**

ECG changes may be seen with aortic dissection, including ischemia or findings of acute inferior myocardial infarction, suggesting involvement of the right coronary artery.

In patients with acute regurgitation, the chest radiograph demonstrates acute pulmonary edema with less cardiac enlargement than expected. In chronic aortic incompetence, the ECG demonstrates LVH, and the chest radiograph shows LVH, aortic dilation, and possibly evidence of congestive heart failure. Echocardiography is essential for confirming the presence of and evaluating the severity of valvular regurgitation. Bedside transthoracic echocardiography should be undertaken in the unstable patient potentially in need of emergency surgery. Transesophageal echocardiography is

recommended when aortic dissection is suspected but may not be possible in acutely unstable patients.

## **Emergency Department Care and Disposition**

1. Pulmonary edema should be treated initially with oxygen and noninvasive ventilation with intubation for failing respiratory effort or clinical deterioration. Diuretics and nitrates can be used but cannot be expected to be effective.
2. **Nitroprusside** (start at 5 micrograms/kilogram/min) and inotropic agents such as **dobutamine** (start at 2.5 micrograms/kilogram/min) or **dopamine** (start 2 micrograms/kilogram/min) can be used to augment forward flow and reduce left ventricular end-diastolic pressure in an attempt to stabilize a patient before emergency surgery.
3. Intraaortic balloon counter pulsation is contraindicated.
4. Although  $\beta$ -blockers are often used in treating aortic dissection, these drugs should be used with great caution, if at all, in the setting of acute aortic valve rupture because they will block the compensatory tachycardia. When used, typically **labetalol** 20 milligrams IV is given.
5. Emergency surgery may be lifesaving.
6. Chronic aortic regurgitation is typically treated with vasodilators such as angiotensin-converting enzyme inhibitors or nifedipine (initiated by a patient's private physician).

## **PROSTHETIC VALVE DISEASE**

Prosthetic valves are implanted in 40,000 patients per year in the United States. There are approximately 80 types of artificial valves, each with advantages and disadvantages. Patients who receive prosthetic valves are instructed to carry a descriptive card in their wallets.

### **Clinical Features**

Many patients have persistent dyspnea and reduced effort tolerance after successful valve replacement. This is more common in the presence of preexisting heart dysfunction or atrial fibrillation. Large paravalvular leaks usually present with congestive heart failure. Patients with new neurologic symptoms may have thromboembolism associated with the valve thrombi or endocarditis. Patients with prosthetic valves usually have abnormal cardiac sounds. Mechanical valves have loud, metallic closing sounds. Systolic murmurs are commonly present with mechanical models. Loud diastolic murmurs are generally not present with mechanical valves. Patients with bioprostheses usually have normal S<sub>1</sub> and S<sub>2</sub>, with no abnormal opening sounds. The aortic bioprosthesis is usually associated with short midsystolic murmur.

## **Diagnosis and Differential**

New or progressive dyspnea of any form, new onset or worsening of congestive heart failure, decreased exercise tolerance, or a change in chest pain compatible with ischemia suggest valvular dysfunction. Persistent fever in patients with prosthetic valves should be evaluated with blood cultures for possible endocarditis. Blood studies that may be helpful include a blood count with red blood cell indices and coagulation studies if the patient is on warfarin. Emergency echocardiographic studies should be requested if there is any question about valve dysfunction. Ultimately, echocardiography and/or cardiac catheterization may be required for diagnosis.

## **Emergency Department Care and Disposition**

1. It is critical that patients with suspected acute prosthetic valvular dysfunction have immediate referral to a cardiac surgeon for possible emergency surgery.
2. The need for the intensity of anticoagulation therapy varies with each type of mechanical valve, but for those patients taking warfarin its INR

goal ranges from 2 to 3.5.

3. Acute prosthetic valvular dysfunction due to thrombotic obstruction has been treated successfully with thrombolytic therapy, but the diagnosis generally requires consultation with a cardiologist. Lesser degrees of obstruction should be treated with optimization of anticoagulation.
4. Disposition of patients with worsening of symptoms can be problematic, and consultation with the patient's regular physician may be needed before consideration for discharge.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 58, "Valvular Emergencies" by Simon A. Mahler.



## **CHAPTER 24**

# **The Cardiomyopathies, Myocarditis, and Pericardial Disease**

N. Stuart Harris

## **THE CARDIOMYOPATHIES**

Cardiomyopathies are the third most common form of heart disease in the United States and are the second most common cause of sudden death in the adolescent population. It is a disease process that directly affects the cardiac structure and alters myocardial function. Four types are currently recognized: (a) dilated cardiomyopathy (DCM), (b) hypertrophic cardiomyopathy (HCM), (c) restrictive cardiomyopathy, and (d) dysrhythmogenicity of right ventricular cardiomyopathy.

## **DILATED CARDIOMYOPATHY**

Dilation and compensatory hypertrophy of the myocardium result in depressed systolic function and pump failure leading to low cardiac output. Eighty percent of cases of DCM are idiopathic. Idiopathic DCM is the primary indication for cardiac transplant in the United States.

### **Clinical Features**

Systolic pump failure leads to signs and symptoms of congestive heart failure (CHF) including dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Chest pain due to limited coronary vascular reserve also may be present. Mural thrombi can form from diminished ventricular contractile force, and there may be signs of peripheral embolization (eg,

focal neurologic deficit, flank pain, hematuria, or pulseless, cyanotic extremity). Holosystolic regurgitant murmur of the tricuspid and mitral valve may be heard along the lower left sternal border or at the apex. Other findings include a summation gallop, an enlarged and pulsatile liver, bibasilar rales, and dependent edema.

## **Diagnosis and Differential**

Chest x-ray usually shows an enlarged cardiac silhouette, biventricular enlargement, and pulmonary vascular congestion (“cephalization” of flow and enlarged hila). The electrocardiogram (ECG) shows left ventricular hypertrophy, left atrial enlargement, Q or QS waves, and poor R wave progression across the precordium. Echocardiography confirms the diagnosis and demonstrates ventricular enlargement, increased systolic and diastolic volumes, and a decreased ejection fraction. Differential diagnosis includes acute myocardial infarction, restrictive pericarditis, acute valvular disruption, sepsis, or any other condition that results in a low cardiac output state.

## **Emergency Department Care and Disposition**

Patients with newly diagnosed, symptomatic DCM require admission to a monitored bed or intensive care unit. Initial management is directed by symptoms.

1. Intravenous access, supplemental oxygen, and continuous monitoring should be established.
2. Intravenous diuretics (eg, **furosemide** 40 milligrams IV) and **digoxin** (maximum dose, 0.5 milligram IV) may improve symptoms.
3. Angiotensin-converting enzyme (ACE) inhibitors, such as **enalaprilat** 1.25 milligrams IV every 6 hours, and the  $\beta$ -blocker **carvedilol**, 3.125 milligrams orally, improve survival long term.
4. **Amiodarone** (loaded 150 milligrams IV over 10 min and then 1 milligrams/ min for 6 hours) for complex ventricular ectopy can be

administered.

5. Anticoagulation should be considered to reduce risk of mural thrombus formation.

Patients with mild to moderate exacerbation of symptoms can be managed with intravenous diuretics, reinstatement of their medications if noncompliant, counseling, and prompt follow-up.

It is important to search for other causes of exacerbations of DCM such as myocardial ischemia or infarction, anemia, infection, new-onset atrial fibrillation, bradydysrhythmia, valvular insufficiency, renal dysfunction, pulmonary embolism, or thyroid dysfunction.

## **HYPERTROPHIC CARDIOMYOPATHY**

This condition is characterized by left ventricular and/or right ventricular hypertrophy that is usually asymmetric and involves primarily the intraventricular septum. There is no ventricular dilatation. The result is decreased compliance of the left ventricle leading to impaired diastolic relaxation and diastolic filling. Cardiac output is usually normal and occasionally elevated. Fifty percent of cases are hereditary.

### **Clinical Features**

Symptom severity progresses with age. Dyspnea on exertion is the most common symptom, followed by angina-like chest pain, palpitations, and syncope. Patients may be aware of forceful ventricular contractions and call these *palpitations*. Physical examination may show a fourth heart sound, hyperdynamic apical impulse, a precordial lift, and a systolic ejection murmur best heard at the lower left sternal border or apex. The murmur may be increased with the Valsalva maneuver or standing after squatting. The murmur can be decreased by squatting, forceful hand gripping, or passive leg elevation with the patient supine (see [Chapter 23](#) “Valvular Emergencies,” for contrasting murmurs).

## Diagnosis and Differential

The ECG demonstrates left ventricular hypertrophy in 30% of patients and left atrial enlargement in 25% to 50%. Large septal Q waves ( $> 0.3$  mV) are present in 25%. Another ECG finding is upright T waves in those leads with QS or QR complexes (T-wave inversion in those leads would suggest ischemia). Chest x-ray is usually normal. Echocardiography is the diagnostic study of choice and will demonstrate disproportionate septal hypertrophy.

## Emergency Department Care and Disposition

Symptoms of HCM may mimic ischemic heart disease and treatment of those symptoms is covered in [Chapter 18](#). Otherwise, general supportive care is indicated.

1.  $\beta$ -Blockers, such as **atenolol** 25 to 50 milligrams orally every day, are the mainstay of treatment for patients with HCM and chest pain.
2. Patients should be discouraged from engaging in vigorous exercise.
3. Those with suspected HCM who have syncope should be hospitalized and monitored.

## RESTRICTIVE CARDIOMYOPATHY

This is one of the least common cardiomyopathies. In this form of the disease, the ventricular volume and wall thickness are normal, but there is decreased diastolic volume of both ventricles.

### Clinical Features

The predominant symptoms are those of CHF: dyspnea, orthopnea, and pedal edema. Chest pain is uncommon. Physical examination may show

third or fourth heart sound, cardiac gallop, pulmonary rales, jugular venous distension, Kussmaul sign (inspiratory jugular venous distention), hepatomegaly, pedal edema, or ascites.

## **Diagnosis and Differential**

The chest x-ray may show signs of CHF without cardiomegaly. Nonspecific ECG changes are most likely. However, in cases associated with amyloidosis or sarcoidosis, conduction disturbances and low-voltage QRS complexes are common.

Differential diagnosis includes constrictive pericarditis and diastolic left ventricular dysfunction (most commonly due to ischemic or hypertensive heart disease). Differentiating between restrictive cardiomyopathy and constrictive pericarditis (using echocardiography) is critical because constrictive pericarditis can be cured surgically.

## **Emergency Department Care and Disposition**

1. Treatment is symptom directed with the use of diuretics and ACE inhibitors.
2. Corticosteroid therapy is indicated for sarcoidosis.
3. Chelation is used for the treatment of hemochromatosis.
4. Admission is determined by the severity of the symptoms and the availability of prompt subspecialty follow-up.

## **DYSRHYTHMOGENICITY OF RIGHT VENTRICULAR CARDIOMYOPATHY**

This is the rarest form of cardiomyopathy and is characterized by progressive replacement of the right ventricular myocardium with fibrofatty tissue. The typical presentation is that of sudden death or ventricular dysrhythmia in a young or middle-age patient. All these patients require extensive workup and hospitalization.

# MYOCARDITIS

Inflammation of the myocardium may be the result of a systemic disorder or an infectious agent. Viral etiologies include coxsackie B, echovirus, influenza, parainfluenza, Epstein-Barr virus, and human immunodeficiency virus. Bacterial causes include *Corynebacterium diphtheriae*, *Neisseria meningitidis*, *Mycoplasma pneumoniae*, and  $\beta$ -hemolytic streptococci. Pericarditis frequently accompanies myocarditis.

## Clinical Features

Systemic signs and symptoms predominate, and include myalgias, headache, rigors, and fever; heart rate is elevated disproportionate to the degree of temperature elevation. Chest pain due to coexisting pericarditis is frequently present. A pericardial friction rub may be heard in patients with concomitant pericarditis. In severe cases, there may be symptoms of progressive heart failure (CHF, pulmonary rales, pedal edema, etc) or cardiogenic shock.

## Diagnosis and Differential

ECG may be normal, or nonspecific ECG changes, atrioventricular block, prolonged QRS duration, or ST-segment elevation (in the setting of associated pericarditis) are seen. Chest x-ray is typically normal. Cardiac enzymes may be elevated. Differential diagnosis includes cardiac ischemia or infarction, valvular disease, and sepsis.

## Emergency Department Care and Disposition

1. Supportive care is the mainstay of treatment.
2. If a bacterial cause is suspected, antibiotics are appropriate.

3. Many patients have progressive CHF; therefore, hospitalization in a monitored environment is usually indicated (see [Chapter 23](#) for management of CHF), or supportive care for cardiogenic shock (see [Chapter 19](#)).

## ACUTE PERICARDITIS

Inflammation of the pericardium may be the result of infection with a virus (eg, coxsackie virus, echovirus, human immunodeficiency virus), bacteria (eg, *Staphylococcus*, *S pneumoniae*,  $\beta$ -hemolytic *Streptococcus*, *Mycobacterium tuberculosis*), or fungus (eg, *Histoplasmosis capsulatum*). Other etiologies include malignancy (leukemia, lymphoma, melanoma, and metastatic breast cancer), drugs (procainamide and hydralazine), radiation, connective tissue disease, uremia, myxedema, or postmyocardial infarction (Dressler syndrome). Pericarditis may be idiopathic.

### Clinical Features

The most common symptom is sudden or gradual onset of sharp or stabbing chest pain that radiates to the back, neck, left shoulder or arm. Radiation to the left trapezial ridge (due to inflammation of the adjoining diaphragmatic pleura) is distinctive. The pain may be aggravated by movement or inspiration. Typically, chest pain is made more severe by lying supine, and lessened by sitting up and leaning forward. Associated symptoms include low-grade intermittent fever, dyspnea, and dysphagia. A transient, intermittent friction rub heard best at the lower left sternal border or apex is the most common physical finding.

### Diagnosis and Differential

ECG changes of acute pericarditis and its convalescence have been divided into 4 stages. During stage 1, the acute phase, there is ST-segment elevation

in leads I, V<sub>5</sub>, and V<sub>6</sub>, with PR-segment depression in leads II, aV<sub>F</sub> and V<sub>4-6</sub>. As the disease resolves (stage 2), the ST segment normalizes and T-wave amplitude decreases. In stage 3, T-wave inversion appears in leads previously showing ST elevations. The final phase, stage 4, is characterized by resolution of repolarization abnormalities and a return to a normal ECG.

When sequential ECGs are not available, it can be difficult to distinguish pericarditis from the normal variant with “early repolarization.” In these cases, the finding of a ST-segment/T-wave amplitude ratio greater than 0.25 in leads I, V<sub>5</sub>, or V<sub>6</sub> is indicative of acute pericarditis. Pericarditis without other underlying cardiac disease does not typically produce dysrhythmias. Chest x-ray is usually but may show enlarged cardiac silhouette. Echocardiography is the best diagnostic test ([Fig. 24-1](#)) to assess for pericardial effusion. Other tests that may be of value in specific cases include complete blood cell count with differential, serum urea nitrogen and creatinine levels (to rule out uremia), streptococcal serology, appropriate viral serology, other serology (eg, antinuclear and anti-DNA antibodies), thyroid function studies, erythrocyte sedimentation rate, and creatinine kinase levels with isoenzymes (to assess for myocarditis).



**FIGURE 24-1.** Pericardial effusion on parasternal long-axis view.

*Key:* Ant Eff = anterior effusion; AV = aortic valve; LA = left atrium; LV = left ventricle; Post Eff = posterior effusion; RV = right ventricle.



[Reprinted with permission from Reardon RF, Joing SA: Cardiac, in Ma OJ, Mateer JR, Blaivas M (eds): *Emergency Ultrasound, 2nd ed.* Copyright © 2008, The McGraw-Hill Companies, Inc., all rights reserved, Figure 6-24.]

## **Emergency Department Care and Disposition**

1. Stable patients with idiopathic or presumed viral etiologies are treated as outpatients with nonsteroidal anti-inflammatory agents (eg, **ibuprofen** 400 to 600 milligrams orally 4 times daily) for 1 to 3 weeks.
2. Patients should be treated for a specific cause if one is identified.
3. Any patient with myocarditis, uremic pericarditis, enlarged cardiac silhouette on chest x-ray, or hemodynamic compromise should be admitted into a monitored environment.

## **NONTRAUMATIC CARDIAC TAMPONADE**

Tamponade occurs when the pressure in the pericardial sac exceeds the normal filling pressure of the right ventricle, resulting in restricted filling and decreased cardiac output. Causes include metastatic malignancy, uremia, hemorrhage (excessive anticoagulation), bacterial or tubercular disorder, chronic pericarditis, and others (eg, systemic lupus, postradiation, or myxedema). The cause may be idiopathic.

### **Clinical Features**

The most common complaints are dyspnea and decreased exercise tolerance. Other nonspecific symptoms include weight loss, pedal edema, and ascites. Physical findings include tachycardia, low systolic blood pressure, and a narrow pulse pressure. Pulsus paradoxus (apparent dropped beats in the peripheral pulse during inspiration), neck vein distention, distant heart sounds, and right upper quadrant pain (due to hepatic congestion) also may be present. Pulmonary rales are usually absent.

## **Diagnosis and Differential**

Low-voltage QRS complexes and ST-segment elevation with PR-segment depression may be present on the ECG. Electrical alternans (beat-to-beat variability in the amplitude of the P and R waves unrelated to inspiratory cycle) is a classic but uncommon finding (about 20% of cases). Chest x-ray may or may not show an enlarged cardiac silhouette. Echocardiography is the diagnostic test of choice and bedside ultrasound may facilitate a rapid diagnosis.

## **Emergency Department Care and Disposition**

Tamponade is a true emergency.

1. Standard supportive measures as previously discussed should be instituted promptly.
2. An intravenous fluid bolus of 500 to 1000 mL normal saline will facilitate right heart filling and may temporarily improve hemodynamics.
3. Pericardiocentesis is diagnostic and therapeutic.
4. These patients require admission to an intensive care unit or monitored setting.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 59, “The Cardiomyopathies, Myocarditis, and Pericardial Disease,” by James T. Niemann.

# CHAPTER 25

## Thromboembolism

Christopher Kabrhel

Venous thromboembolism (VTE) is a common and deadly disease that includes deep vein thrombosis (DVT) and pulmonary embolism (PE). DVT occurs when blood coagulates inside a deep vein—usually in the leg, but occasionally in the arm or proximal vein. PE occurs when a portion of a clot breaks off and travels to a pulmonary artery. The clinical presentation of VTE is highly variable, thus clinicians must maintain a high index of suspicion.

There are numerous risk factors for VTE including: advanced age, obesity, pregnancy, malignancy, inherited thrombophilia, recent surgery or major trauma, immobility/bed rest, an indwelling central venous catheter, long distance travel, smoking, congestive heart failure, stroke, estrogen use, and inflammatory conditions. The absence of known risk factors does not confer absolute protection from VTE.

### CLINICAL FEATURES

**Deep vein thrombosis:** Classically, DVT presents as calf or leg pain, redness, swelling, tenderness, and warmth. Unfortunately, this constellation of findings is present in fewer than 50% of DVT patients. A difference in lower leg diameter is predictive, but Homan sign (ie, pain in the calf with forced dorsiflexion) is neither sensitive nor specific for DVT. The presence of upper extremity swelling associated with an indwelling venous catheter should raise suspicion of an upper extremity DVT.

Uncommon but severe presentations of DVT include *phlegmasia cerulea dolens* and *phlegmasia alba dolens*. *Phlegmasia cerulea dolens* is a high-grade obstruction that elevates compartment pressures and can compromise limb perfusion. It presents as a massively swollen, cyanotic limb. *Phlegmasia alba dolens* is usually associated with pregnancy and has a

similar pathophysiology but presents as a pale limb secondary to arterial spasm.

**Pulmonary embolism:** The diagnosis of PE should be considered in any patient who experiences acute dyspnea, chest pain, unexplained tachycardia, hypoxemia, syncope, or shock. The most common symptoms include dyspnea, pleuritic or nonpleuritic chest pain, anxiety, cough, and syncope, though PE can present as confusion or even seizure. Common signs include hypoxemia, tachypnea, tachycardia, hemoptysis, diaphoresis, and low-grade fever. Clinical signs of DVT occur in about 50% of patients with PE. Massive PE can cause hypotension, severe hypoxemia, or cardiopulmonary arrest. However, the clinical presentation of VTE can be insidious; there is poor correlation between the size of a PE and the severity of symptoms. In fact, patients with sizable PE may be asymptomatic.

## **DIAGNOSIS AND DIFFERENTIAL**

The leg pain and swelling associated with DVT are similar to that seen with cellulitis, congestive heart failure, musculoskeletal injuries, and venous stasis without thrombosis. The differential diagnosis of PE includes many pulmonary disorders, including: asthma, chronic obstructive pulmonary disease, pleural effusion, pneumonia, and pneumothorax. Cardiac disorders that may mimic PE include angina/myocardial infarction, congestive heart failure, pericarditis, and tachydysrhythmia. Muscle strain and costochondritis can mimic the chest pain of PE. Anxiety and hyperventilation syndrome may mimic PE but should be considered diagnoses of exclusion.

**Pretest probability assessment:** The clinician should consider the patient's probability of PE prior to the decision to initiate testing. Testing should be reserved for patients whose probability of PE is higher than a predetermined "test threshold," approximately 1.4% to 2.0%. When the clinician feels that a patient is low risk for PE and the Pulmonary Embolism Rule-Out Criteria (PERC Rule) ([Table 25-1](#)) is negative, the risk of PE is 1% at 45 days. In these cases no further testing for PE is required.

Age < 50 years
Pulse oximetry > 94% (breathing room air)
Heart rate < 100 beats/min
No prior venous thromboembolism
No recent surgery or trauma (requiring hospitalization, intubation, or epidural anesthesia within 4 weeks prior)
No hemoptysis
No estrogen use
No unilateral leg swelling

**TABLE 25-1** Pulmonary Embolism Rule-Out Criteria (PERC Rule)

When PE cannot be excluded with the PERC Rule, the patient’s pretest probability should guide the clinician’s choice and interpretation of diagnostic testing. Pretest probability can be based on the clinician’s prior experience, or by using an objective and validated instrument such as found in [Table 25-2](#). When signs and symptoms are consistent with DVT without PE, pretest probability can be determined using the DVT decision rule developed by Wells et al, [Table 25-3](#).

Objective Criteria	Points	Subjective Criteria	Points
Heart rate > 100 beats/min	1.5	Clinician considers alternative diagnoses to be less likely than PE	3
Hemoptysis	1		
History of venous thromboembolism	1.5		
Malignancy (active)	1		
Leg swelling, pain with palpation of deep veins (clinical signs of DVT)	3		

Risk for PE: >6 = high risk (78.4%); 2 to 6 = moderate risk (27.8%); <2 points = low risk (3.4%).  
Key: DVT = deep vein thrombosis.

**TABLE 25-2** Wells Score for Pulmonary Embolism (PE)

**Diagnostic Testing:** For patients with low or intermediate pretest probability (from [Tables 25-2](#) and [25-3](#)), D-dimer testing is the recommended first test. The diagnostic sensitivity of automated quantitative D-dimer assays ranges from 94% to 98% and the specificity from 50% to 60% for PE and DVT. Clinicians should know the performance of the local assay. Advanced age, active malignancy, pregnancy, recent surgery and rheumatologic and sickle cell disease can all elevate D-dimer levels in the absence of VTE.

Clinical Feature	Points*
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for >3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of >3 cm (below tibial tuberosity)	1
Unilateral pitting edema (greater in symptomatic leg)	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis as likely as or more likely than DVT	-2

\*Risk score interpretation (probability of DVT):  $\geq 3$  points: high risk (75%); 1 or 2 points: moderate risk (17%);  $< 1$  point: low risk (3%).

(Reproduced with permission from Wells PS, Anderson DR, Bormanis J, et al. Value of assessment of pretest probability of deep-vein thrombosis in clinical management. *The Lancet*. 1997;350(9094):1795-1798.)

**TABLE 25-3** Wells Score for Deep Vein Thrombosis (DVT)

For patients with high pretest probability of VTE, those with a positive D-dimer, or those in whom D-dimer testing is very likely to be positive, imaging is indicated. Duplex ultrasonography is the test of choice for evaluating DVT with high sensitivity (95%) and specificity (95%) for lower extremity DVT. Sensitivity is lower for pelvic and isolated calf DVT and in obese patients. To rule out DVT in patients with high clinical probability, some algorithms require both a negative initial ultrasound and a concurrent negative D-dimer or a follow up ultrasound performed 1 week after the initial negative ultrasound. The performance of 2 negative duplex ultrasounds 1 week apart is associated with  $< 1\%$  risk of symptomatic DVT or PE in 3 months.

Lower extremity ultrasound may also be a cost-effective first test for evaluating possible PE. Patients with signs or symptoms of PE who have DVT demonstrated on venous ultrasound can be assumed to have PE. This strategy may be particularly helpful when CT pulmonary angiography is relatively or absolutely contraindicated, such as when the patient is pregnant, has renal insufficiency, or is allergic to intravenous contrast.

In most cases, contrast-enhanced computed tomography (CT) of the chest, with or without lower extremity venography, is the test of choice to

rule out PE. Chest CT angiography identifies a clot as a filling defect in a contrast-enhanced pulmonary artery ([Fig. 25-1](#)). The sensitivity of a technically adequate CT for PE is 83% to 90%, and specificity is 95%. The negative likelihood ratio is between 0.11 and 0.18, similar to that of the D-dimer. Therefore, as with D-dimer testing, CT pulmonary angiography should be interpreted in the context of pretest probability. Compared to other imaging modalities, CT has the advantage of demonstrating important alternative diagnoses in about 15% of patients imaged for possible PE.



**FIGURE 25-1.** Axial image from a chest CT angiogram demonstrating a filling defect consistent with acute pulmonary embolism. Two white arrowheads outline a circular filling defect in the right middle lobar pulmonary artery. The long white arrow projecting in the left lung points to a filling defect in a segmental artery in the posterior medial segmental artery.

Ventilation-perfusion (V/Q) scanning is performed by comparing emission of radioisotope that has been injected into the pulmonary arteries

to emission of radioisotope that has been inhaled into the alveoli. A V/Q scan that demonstrates homogeneous scintillation throughout the lung in the perfusion portion rules out PE in 96% to 100% of cases. However, only about one-third of V/Q scans demonstrate findings sufficient to diagnose or rule out PE with certainty. Therefore, the clinical utility of V/Q scanning is generally limited to patients who cannot undergo CT (eg, contrast allergy).

**Ancillary testing:** Patients suspected of having PE should undergo routine cardiopulmonary testing; findings may include hypoxemia on pulse oximetry, or decreased end-tidal carbon dioxide. Chest radiographs frequently demonstrate cardiomegaly or atelectasis, although are not specific for PE. Focal oligemia (Westermarck sign) or a peripheral dome-shaped dense opacification (Hampton hump) are relatively specific, but present in fewer than 5% of cases. Chest radiographs can be helpful when they fail to demonstrate an alternative diagnosis, and therefore increase the concern for PE.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The treatment of VTE consists of initial stabilization, anticoagulation, and thrombolytic therapy in severe cases. All patients suspected of having PE should have their cardiac rhythm, blood pressure, and pulse oxygenation measured continuously.

1. Patients should be placed on supplemental oxygen to maintain a pulse oximetry reading greater than 95%.
2. Intravenous crystalloid fluids should be given as needed to augment preload and correct hypotension.
3. Anticoagulation may be achieved by administering intravenous unfractionated heparin (UFH) or subcutaneous low molecular weight heparin (LMWH). Outcomes, complications, and cost may be improved with use of LMWH for PE or DVT, although the benefits are small. LMWH can result in unpredictable anticoagulation in patients with severe renal insufficiency, so UFH is preferred in this setting. Potentially



decreased absorption of LMWH in obese patients or high risk of bleeding in selected patients may also favor the use of UFH. Dosing of **unfractionated heparin** should be weight-based, with 80 units/kilogram given as an initial bolus followed by 18 units/kilogram/h. The activated partial thromboplastin time should be maintained between 55 and 80 seconds (1.5 to 2.5 times normal). The loading dose should be reduced for morbid obesity; give UFH 80 units/kilogram for ideal body weight, plus 24 to 32 units UFH for each kilogram above ideal body weight. Dosing of LMWH is also weight-based. Examples include: **dalteparin** 100 units/ kilogram subcutaneous every 12 hours or 200 units/kilogram subcutaneous every day; **enoxaparin** 1 milligram/kilogram SC every 12 hours or 1.5 milligrams/kilogram subcutaneous every day; and **tinzaparin** 175 units/kilogram subcutaneous every day. There are few absolute contraindications to anticoagulation with heparin for acute PE, although patients with recent intracranial hemorrhage or active gastrointestinal hemorrhage may have anticoagulation withheld. For patients with moderate to high pretest probability and no contraindications, the benefits of initiating heparin therapy prior to diagnostic testing outweigh the risks. The appropriate use of anticoagulation for isolated calf vein thrombosis is unresolved. Options include withholding anticoagulation pending a repeat ultrasound in one week to assess for clot progression into a proximal vein, or therapeutic anticoagulation.

4. For patients with contraindications to heparin, such as documented heparin-induced thrombocytopenia, an alternative anticoagulant should be used. **Fondaparinux** inhibits factor Xa and may be dosed as follows: <50 kilograms, 5 milligrams subcutaneous every day; 50 to 100 kilograms, 7.5 milligrams subcutaneous every day; > 100 kilograms, 10 milligrams subcutaneous every day. **Lepirudin** inhibits thrombin and may be dosed as follows: 0.4 milligram/kilogram slow bolus up to 44 milligrams followed by an infusion of 0.1 to 0.15 milligram/kilogram/h. Both fondaparinux and lepirudin are contraindicated in severe renal insufficiency.
5. Oral anticoagulation with **warfarin** can be initiated simultaneously with heparin therapy. Usual initial dosing is 5 milligrams every day with a target international normalized ratio of 2 to 3.

6. **Thrombolytic therapy** should be considered for selected patients with VTE. Severe DVT that causes *phlegmasia cerulea dolens* can lead to loss of limb and requires immediate treatment. The affected limb should be maintained at neutral level, constrictive clothing, casts or dressings should be removed, and anticoagulation should be initiated. Catheter-based thrombectomy/thrombolysis should be discussed with an interventional radiologist, peripheral interventional cardiologist, or vascular surgeon. If this service is not available, intravenous thrombolysis should be considered. Major contraindications to thrombolytic therapy include intracranial disease, uncontrolled hypertension, recent major surgery or trauma, ongoing bleeding, and metastatic disease. The recommended dose is 50 to 100 milligrams of **alteplase** infused IV over 4 hours, though few data are available to support one dosing regimen over another.

Currently, the only patients with PE who have been shown to clearly benefit from thrombolytic therapy are those with **massive PE**, defined as a large PE associated with hemodynamic instability (systolic blood pressure below 90 mm Hg, or below 100 mm Hg in a patient with preexisting hypertension). Patients may be defined as having **submassive PE** based on the presence of right heart strain on echocardiogram, elevated cardiac biomarkers (troponin or brain-natriuretic peptide), a shock index (heart rate/systolic blood pressure) > 1, or severe hypoxemia and respiratory distress. While thrombolysis does not appear to improve mortality in patients with submassive PE, it may improve cardiac function and quality of life. Patients with smaller PE probably do not benefit from thrombolysis. Therefore, when there the risk of bleeding is low, thrombolysis should be strongly considered in cases of massive PE, and considered in patients with submassive PE. There are three thrombolytic regimens approved by the United States FDA for the treatment of PE. The most commonly used drug is tissue plasminogen activator (tPA, alteplase). The recommended dose of tPA for PE is 100 milligrams infused over two hours. However, in cases of cardiac arrest where prolonged drug administration is impractical, a slow bolus may be administered. Either enoxaparin or unfractionated heparin can be started after the thrombolytic infusion.

7. An inferior vena cava filter should be considered when anticoagulation has failed, is contraindicated, or when submassive PE is associated with persistent large DVT.
8. In specialized centers, surgical thrombectomy may be an option for patients with massive PE that does not respond to thrombolysis.
9. Patients with DVT may be treated as outpatients using a combination of LMWH and oral anticoagulation (warfarin). However, practical limitations such as the ability to obtain and inject LMWH at home, should be considered. Stable patients with PE should be admitted to a telemetry bed. Patients who exhibit signs of circulatory compromise and all patients who receive thrombolytic therapy should be admitted to an intensive care unit.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 60, "Thromboembolism," by Jeffrey A. Kline.

## CHAPTER 26

# Systemic and Pulmonary Hypertension

David M. Cline

Classification of acute systemic hypertension into categories facilitates management:

1. **Hypertensive emergency:** elevated blood pressure (BP) associated with target organ dysfunction such as aortic dissection, acute pulmonary edema, acute coronary syndrome, acute renal failure, severe preeclampsia, hypertensive encephalopathy, subarachnoid hemorrhage, intracranial hemorrhage, acute ischemic stroke, and sympathetic crisis. Immediate recognition and treatment are required but therapeutic goals vary considerably.
2. **Hypertensive urgency:** a clinical presentation associated with severe elevations in blood pressure without progressive target organ dysfunction. The arbitrary numerical criterion of  $> 180/110$  mm Hg is often cited as an indication for treatment, when in fact the clinical benefit of such treatment is not well defined (see Emergency Department Care and Disposition section).

The clinician must ensure that the BP cuff size is appropriate for the patient's size; a small cuff relative to the arm size produces a falsely elevated reading.

## CLINICAL FEATURES

Essential historic features include a prior history of HTN; noncompliance with BP medications; cardiovascular, renal, or cerebrovascular disease; diabetes; hyperlipidemia; chronic obstructive pulmonary disease or asthma; and a family history of HTN.

Precipitating causes such as pregnancy, illicit drug use (cocaine and methamphetamines), or decongestants should be considered. Patients

should be asked about central nervous system symptoms (headaches, visual changes, weakness, seizures, and confusion), cardiovascular symptoms (chest pain, palpitations, dyspnea, syncope, pedal edema, or tearing pain radiating to the back or abdomen), and renal symptoms (anuria, edema, or hematuria). The patient should be examined for evidence of papilledema, retinal exudates, neurologic deficits, seizures, or encephalopathy; the presence of these findings may constitute a hypertensive emergency in the setting of elevated blood pressure. The patient also should be assessed for carotid bruits, heart murmurs, gallops, asymmetrical pulses or unequal blood pressures (coarctation vs aortic dissection), pulsatile abdominal masses, and pulmonary rales. Hypertensive encephalopathy is characterized by altered mental status in the setting of acute hypertension, and may be accompanied by headache, vomiting, seizures, visual disturbances, papilledema, or hematuria. In the pregnant (or postpartum) patient, the clinician should look for hyperreflexia and peripheral edema, suggesting preeclampsia.

## **DIAGNOSIS AND DIFFERENTIAL**

Testing should be guided by presenting symptoms, the most cost effective test is urinalysis. Renal impairment may present as hematuria, proteinuria, red cell casts, or elevations in blood urea nitrogen, creatinine, and potassium levels. An electrocardiogram may show ST- and T-wave changes consistent with coronary ischemia (see [Chapter 18](#)), electrolyte abnormalities, or left ventricular hypertrophy. A chest x-ray may help identify congestive heart failure (see [Chapter 22](#)), or aortic dissection (see [Chapter 27](#)). In patients with neurologic compromise, computed tomography of the head may show ischemic changes, edema, or blood (see [Chapter 141](#)). A urine or serum drug screen may identify illicit drug use. A pregnancy test should be done on all hypertensive women of childbearing potential.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Patients with hypertensive emergencies require O<sub>2</sub> supplementation, cardiac monitoring, and intravenous access. After attention to the ABCs of resuscitation, the treatment goal is to reduce arterial pressure gradually in the following clinical situations, with attention to the therapeutic goal.

1. **Aortic dissection:** reduce force of contraction and shear forces first with  $\beta$ -blockers, reduce heart rate to approximately 60 beats/min, reduce BP below 140 mm Hg systolic, and ideally, below 120 systolic (range of 100 to 120 mm Hg) if tolerated by the patient. Recommended first agents include **esmolol** (300 micrograms/kilogram IV bolus followed by a 50 micrograms/kilogram/min infusion) or **labetalol** (20 milligram IV over 2 min followed by subsequent doses of 20 to 40 milligrams IV every 10 min as needed up to 300 milligrams maximum). If  $\beta$ -blockers are contraindicated, use **verapamil** 5 to 10 milligrams IV, or **diltiazem** 0.25 milligram/kilogram IV over 2 min, to reduce heart rate. Follow  $\beta$ -blockers with vasodilators as needed to achieve desired blood pressure reduction. **Nicardipine** can be started by IV infusion: start at a rate of 5 milligrams/h. If target BP is not achieved in 5 to 15 min, increase dose by 2.5 milligrams/h every 5 to 15 min until target pressure or the maximum dose of 15 milligrams/h is reached. Alternatively, use **nitroprusside** IV infusion: 0.3 to 0.5 microgram/kilogram initial infusion, increase by increments of 0.5 microgram/kilogram/min; titrate to desired effect.
2. **Acute hypertensive pulmonary edema:** reduce BP by no more than 20% to 30%. First agent of choice is **nitroglycerin**, sublingual 0.4 milligram, up to 3 doses, paste 1 to 2 inches, or IV infusion, start 5 micrograms/min, increase by 5 micrograms/min every 3 to 5 min to 20 micrograms/min; if no response at 20 micrograms/min, increase by 10 micrograms/min every 3 to 5 min, up to 200 micrograms/min. Alternatives include **enalaprilat** IV, 0.625 to 1.25 milligrams over 5 min every 4 to 6 hours, titrate at 30 min intervals to a maximum of 5 milligrams every 6 hours, **nicardipine** (see dose above, #1) or **nitroprusside** (see dose above, #1).
3. **Acute coronary syndrome:** if BP is above 160 mm Hg systolic, reduce BP no more than 20% acutely. Start with **nitroglycerin** (see dose above, #2) or **metoprolol** 50 to 100 milligrams PO every 12 hours, or 5

milligrams IV every 5 to 15 min up to 15 milligrams. Avoid IV  $\beta$ -blockers if patient at risk for cardiogenic shock.

4. **Acute sympathetic crisis:** treat to relieve symptoms, start with **benzodiazepines** first. Follow with **nitroglycerin** (see dose above, #2), or **phentolamine**, bolus load: 5 to 15 milligrams IV.
5. **Acute renal failure:** for BP above 180/110 mm Hg, reduce BP by no more than 20% acutely. Recommend agents include **labetalol** (see dose above, #1) **nicardipine** (see dose above, #1) or **fenoldopam**, start 0.1 microgram/kilogram/min, titrate to desired effect every 15 min, range 0.1 to 1.6 micrograms/kilogram/min.
6. **Preeclampsia:** For BP above 160/110 mm Hg, use **labetalol** (see dose above, #1). **Hydralazine**, 5 to 10 milligrams IV, is less predictable, but commonly used.
7. **Hypertensive encephalopathy:** For BP above 180/110 (in the setting of immunosuppressive drugs, symptomatic BP may be lower), reduce BP by no more than 20% acutely. Recommended agents include **nicardipine** (see dose above, #1) **labetalol** (see dose above, #1), **fenoldopam** (see dose above, #5), or **nitroprusside** (see dose above, #1).
8. **Subarachnoid hemorrhage:** reduce systolic pressure below 160 mm Hg or MAP below 130 mm Hg to prevent rebleeding. Recommended agents include **nicardipine** (see dose above, #1) **labetalol** (see dose above, #1), or **esmolol** (see dose above, #1).
9. **Intracranial hemorrhage:** to reduce hemorrhage growth, for patients with evidence of increased intracranial pressure (decreased level of consciousness, evidence of midline shift or hematoma volume > 30 mL on CT imaging.) reduce MAP to 130 mm Hg. In patients for whom there is no suspicion of increased intracranial pressure, treatment may be intensified to a MAP of 110 mm Hg, or a systolic pressure of 150 to 160 mm Hg. Recommended agents include **nicardipine** (see dose above, #1) **labetalol** (see dose above, #1), or **esmolol** (see dose above, #1).
10. **Acute ischemic stroke:** If fibrinolytic therapy is planned, reduce BP below 185/110 mm Hg; if no fibrinolytic therapy is planned and BP remains elevated on repeat measures, reduce BP below 220/120 mm Hg. More intensive lowering can be accomplished safely (however, pharmacologic reduction below 160/100 is not advised). Recommended

agents include **labetalol** (see dose above, #1), **nicardipine** (see dose above, #1), or **nitroglycerin** paste, 1 to 2 inches.

11. For hypertensive urgency, useful agents include oral **labetalol** 200 to 400 milligrams, repeated every 2 to 3 hours; oral **captopril** 25 milligrams every 4 to 6 hours; sublingual **nitroglycerin** spray or tablets (0.3 to 0.6 milligram); or **clonidine**, 0.2 milligram oral loading dose, followed by 0.1 milligram/h until the DBP is below 115 mm Hg, or a maximum of 0.7 milligram.
12. For asymptomatic patients with severe hypertension, with BP above the 180 to 200 systolic range, or above 110 to 120 diastolic range, starting an oral agent at discharge should be considered. The choice of the oral agent should be based on coexisting conditions, if any. Diuretics, such as **hydrochlorothiazide** 25 milligrams/d, should be used in most patients with uncomplicated HTN. For patients with angina, postmyocardial infarction, migraines, or supraventricular arrhythmias, a  $\beta$ -blocker should be considered, such as **metoprolol** 50 milligrams orally 2 times daily. Angiotensin-converting enzyme inhibitors such as **lisinopril**, start at 10 milligrams daily, can be used in those with heart failure, renal disease, recurrent strokes, or diabetes mellitus. Restarting a noncompliant patient on a previously established regimen is a recommended strategy.

## **CHILDHOOD EMERGENCIES**

## **HYPERTENSIVE**

Children often will have nonspecific complaints such as throbbing frontal headache or blurred vision. Physical findings associated with HTN are similar to those found in adults.

The most common etiologies in this age group are renovascular lesions and pheochromocytoma. The decision to treat a hypertensive emergency in a child is based on the BP and associated symptoms. Urgent treatment is required if the BP exceeds prior measurements by 30%. Alternatively, if prior measurements are not known, childhood hypertension is defined as diastolic or systolic blood pressure  $\geq$  95th percentile on a standardized table. The goal is to reduce the BP by 25% within 1 hour in acutely



symptomatic patients. Preferred medications for the control of hypertensive emergencies in children include **labetalol**, 0.2 to 1.0 milligram/kilogram per dose, up to 40 milligrams/dose, or an infusion of 0.25 to 3.0 milligrams/kilogram/h; **nicardipine**, 1 to 3 micrograms/kilogram/min; or, if prior drugs fail, **nitroprusside**, 0.5 to 10.0 micrograms/kilogram/min. The treatment of pheochromocytoma is surgical excision and managing the BP with  $\alpha$ -adrenergic blockers such as phentolamine. Pediatric HTN that requires intervention in the emergency department will likely require admission.

## **PULMONARY HYPERTENSION**

Although the diagnosis of pulmonary hypertension cannot be made in the ED, suspicion of primary or secondary pulmonary hypertension as a cause of dyspnea, chest pain, or syncope can affect ED evaluation, consultation, or disposition. Pulmonary hypertension is a pathologic condition characterized by elevation of the pulmonary vascular pressure, which compromises right ventricular function. There can be an isolated increase in pulmonary arterial pressure or an elevation of both arterial and venous pressure. The hemodynamic parameter that defines pulmonary hypertension is a median pulmonary artery pressure of  $> 25$  mm Hg at rest or  $> 30$  mm Hg during effort. The most common symptoms are dyspnea, fatigue, syncope, and chest pain. Typically, this disorder will be seen in association with other cardiovascular or pulmonary disorders such as chronic obstructive pulmonary disease, left ventricular dysfunction or disorders associated with hypoxemia. Treatment of the underlying disorder is the only management indicated in the emergency setting, for example, oxygen for conditions producing hypoxia. Patients may be on calcium channel blockers chronically, or for primary pulmonary hypertension, patients may have arrangements for home infusions of epoprostenol (prostacyclin).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 61, “Systemic and Pulmonary Hypertension,” by David M. Cline, and Alberto J. Machado.

## CHAPTER 27

# Aortic Dissection and Aneurysms

David E. Manthey

## ABDOMINAL AORTIC ANEURYSMS

Aortic dissection and abdominal aortic aneurysms (AAAs) are important causes of morbidity and death that require rapid diagnosis and frequently require prompt operative repair to offer the patient any chance of survival. Diagnosing these conditions can be challenging and carries a high risk of misdiagnosis.

### Clinical Features

Four clinical scenarios arise regarding AAAs: acute rupture, aortoenteric fistula, chronic contained rupture, and an incidental finding. Although there are several other nonaortic large artery aneurysms that often require surgical repair by a vascular surgeon, they are aptly covered in *Tintinalli's Emergency Medicine*, 7th edition.

Acute rupturing AAA is a true emergency that, if not rapidly identified and repaired, will lead to death. The classic presentation is of an older (> 60 years) male smoker with atherosclerosis who presents with sudden onset severe back or abdominal pain, hypotension, and a pulsatile abdominal mass. Patients may present with syncope or some variation of unilateral flank pain, groin pain, hip pain, or pain localizing to one quadrant of the abdomen.

Fifty percent of patients describe a ripping or tearing pain that is severe and abrupt in onset. Patients may have a tender pulsatile abdominal mass on physical examination, but the absence of pain does not imply an intact

aorta. Obesity may mask a pulsatile abdominal mass. Nausea and vomiting are commonly present.

Shock may persist through presentation or may transiently improve due to compensatory mechanisms. Femoral pulsations are typically normal. Retroperitoneal hemorrhage may be appreciated as periumbilical ecchymosis (Cullen sign), flank ecchymosis (Grey-Turner sign), or scrotal hematomas. If blood compresses the femoral nerve, a neuropathy of the lower extremity may be present.

Aortoenteric fistulas, although rare, present as gastrointestinal bleeding, either a small sentinel bleed or massive life-threatening hemorrhage. A history of previous aortic grafting (eg, AAA repair) increases the suspicion. Because the duodenum is the usual site of the fistula, the patient may present with hematemesis, melenemesis, melena, or hematochezia.

Chronic contained rupture of AAA is an uncommon presentation. If an AAA ruptures into the retroperitoneum, there may be significant fibrosis and a limiting of blood loss. The patient typically appears quite well and may complain of pain for an extended period.

Discovering a previously undiagnosed asymptomatic AAA on physical or radiologic examination can be lifesaving. Those aneurysms larger than 5 cm in diameter (outer wall to outer wall) are at a greater risk for rupture, but all should be referred to a vascular surgeon.

## **Diagnosis and Differential**

Although the diagnosis may be relatively straightforward in the setting of syncope, back pain, and shock with a tender pulsatile abdominal mass, the differential diagnosis varies depending on the presentation. Missed AAAs are most frequently misdiagnosed as renal colic. This life-threatening disease process should be considered in the differential diagnosis for any patient that presents with back pain, an intraabdominal process (pancreatitis, diverticulitis, mesenteric ischemia, etc), possible testicular torsion, or gastrointestinal bleeding disorders (eg, esophageal varices, tumors, or ulcers).

If the diagnosis of rupturing AAA is clear on clinical grounds, the operating vascular surgeon should immediately evaluate the patient.

However, when the diagnosis is not entirely clear, confirming studies may be required. In the unstable patient, technically adequate bedside abdominal ultrasound has a > 90% sensitivity for identifying AAA and can measure the diameter of the aneurysm (see [Fig. 27-1](#)). Be aware that aortic rupture or retroperitoneal bleed cannot be reliably identified with ultrasound. Obesity and bowel gas technically may limit the study. In the stable patient, computed tomography (CT) can identify the AAA and delineate the anatomic details of the aneurysm and any associated rupture. The role of plain radiography in the diagnosis of rupturing AAA is unclear; a calcified, bulging aortic contour is present in only 65% of patients with symptomatic AAA.



**FIGURE 27-1.** Bedside US image of an abdominal aortic aneurysm. This aneurysm measures 6.5 cm.

## **Emergency Department Care and Disposition**

The primary role of the emergency physician is in identifying AAA.

1. For suspected rupturing AAA or aortoenteric fistula, prompt surgical consultation in anticipation of emergency surgery is critical. No diagnostic testing should delay surgical repair.
2. The patient is stabilized with large-bore intravenous access, judicious fluid administration for hypotension, treatment of hypertension (see [Chapter 26](#)), and typing and cross-matching of several units of packed red blood cells, with transfusion as needed. Because patients may rapidly deteriorate, those who undergo diagnostic testing should not be left unattended in the radiology department.
3. Pain control should be initiated with narcotic medications as compared to nonsteroidals due to medication induced platelet dysfunction. Control of pain is a compassionate intervention that may aid in control of blood pressure, but beware of hypotension.
4. For chronic contained rupturing AAA, consultation with a vascular surgeon for urgent repair and intensive care unit admission should be sought.
5. For AAA identified as an incidental finding, the patient potentially can be discharged home, depending on the aneurysmal size and comorbid factors. Telephone consultation with a vascular surgeon for admission or close office follow-up is usually adequate.

## **AORTIC DISSECTION**

### **Clinical Features**

Aortic dissection typically presents (> 85% of patients) with acute onset of pain that is most severe at onset located in the chest and radiating to the back. The location of the pain may indicate the area of the aorta that is involved. Seventy percent of patients with ascending involvement have anterior chest pain, and 63% of patients with involvement of the descending aorta have back pain. The pain pattern may change as the dissection progresses from one anatomic area to another. The pain is described as ripping or tearing by 50% of patients. Accompanying nausea, vomiting, diaphoresis and the feeling of impending doom are common.

Most patients are male (66%), older than 50 years (mean age, 63), and have a history of hypertension (72%). Another group of patients are younger with identifiable risk factors such as connective tissue disorders, congenital heart disease, and pregnancy. Up to 30% of patients with Marfan syndrome will develop a dissection. Iatrogenic induced aortic dissection may occur after aortic catheterization or cardiac surgery.

To communicate more effectively with the surgeons, the emergency department physician should classify aortic dissections in 1 of 2 ways. The Stanford classification divides dissections into those that involve the ascending aorta (type A) and those that are restricted to the descending aorta (type B). The DeBakey classification divides dissections into 3 groups: involvement of the ascending and descending aortas (type I), involvement of only the ascending aorta (type II), or involvement of only the descending aorta (type III). Also look for an intramural hematoma, due to infarction of the aortic media, that may resolve or progress onto a true dissection.

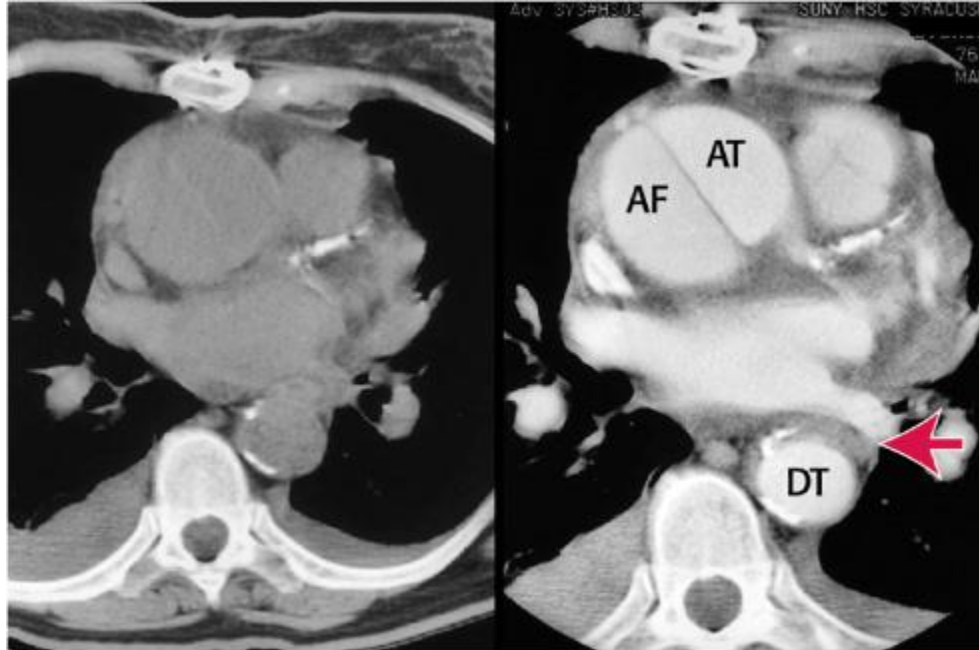
As the dissection progresses, seemingly unrelated symptom complexes may present themselves. Presentations include aortic valve insufficiency, coronary artery occlusion with myocardial infarction, carotid involvement with stroke symptoms, occlusion of vertebral blood supply with paraplegia, cardiac tamponade with shock and jugular venous distention, compression of the recurrent laryngeal nerve with hoarseness of the voice, and compression of the superior cervical sympathetic ganglion with Horner syndrome. The dissection may open back into the true aortic lumen with a marked decrease in symptoms, leading to a false sense of security.

The patient's physical examination findings will depend on the location and progression of the dissection. A diastolic murmur of aortic insufficiency may be heard. Hypertension and tachycardia are common, but hypotension also may be present. Fifty percent of patients have decreased pulsation in the radial, femoral, or carotid arteries. Although one might expect a difference in extremity blood pressures, no specific threshold values have been defined. Forty percent of patients have neurologic sequelae.

## **Diagnosis and Differential**

The differential diagnosis to be considered depends on the location and progression of the dissection. Other causes of aortic insufficiency, myocardial infarction, esophageal rupture, other causes of strokes, spinal injury or tumor, vocal cord tumors, and other causes of cardiac tamponade, including pericardial disease, may need to be considered. An electrocardiogram would help demonstrate disruption of a coronary artery, most commonly the right.

The diagnosis of aortic dissection depends on radiographic confirmation once the diagnosis is suspected. The chest x-ray is abnormal in 80% of patients with aortic dissection. The abnormality may be an abnormal aortic contour; widening of the mediastinum; deviation of the trachea, mainstem bronchi, or esophagus; apical capping; or pleural effusion. The “calcium sign” may be present, with intimal calcium deposits seen distant from the edge of the aortic contour. CT is 83% to 100% sensitive and 87% to 100% specific for the diagnosis of dissection. Spiral CT with rapid IV contrast boluses is the most sensitive (see [Fig. 27-2](#)). Angiography is rarely used anymore. It may better define the anatomy, extent, and complications of a dissection. Transesophageal echocardiograms, in experienced hands, are 97% to 100% sensitive and 97% to 99% specific. The use of these studies is institutionally dependent, and they should be ordered in conjunction with the consulting vascular or thoracic surgeon.



**FIGURE 27-2.** CT image of a type A aortic dissection. True and false lumens are present in the ascending aorta and descending aorta (descending false lumen at arrow) on noncontrast (left) and contrast (right) images.

*Key:* AF = ascending false lumen; AT = ascending true lumen; DT = descending true lumen.

## **Emergency Department Care and Disposition**

All patients with aortic dissection or strongly suspected aortic dissection require emergent vascular or thoracic surgical consultation and prompt radiographic confirmation of the diagnosis, which is best directed by the operating surgeon. In general, patients with dissection of the ascending aorta require prompt surgical intervention. The operative care of dissection of only the descending aorta is controversial and should be evaluated on a case-by-case basis.

1. Stabilization of the patient typically requires large-bore intravenous access with availability of type and cross-matched blood in case of free rupture.



2. Management of hypertension is best done with  $\beta$ -blockers because these decrease the blood pressure and the shear force (see [Chapter 26](#)). Agents such as **esmolol** (300 micrograms/kilogram IV bolus followed by a 50 micrograms/kilogram/min infusion) or **labetolol** (20 milligrams IV for 2 min followed by subsequent doses of 20 to 40 milligrams IV for 10 min) are typically used. The goal is to reduce heart rate to between 60 and 70.
3. Vasodilators, such as **nitroprusside** (starting at 0.3 micrograms/kilogram/min IV), should be used only after adequate inotropic blockade has been made with  $\beta$ -receptor or calcium-channel blockers (see [Chapter 26](#)). Intravenous antihypertensive therapy in an acute aortic dissection is traditionally titrated according to pain relief and BP with a final goal of achieving a systolic BP of 100 to 120 mm Hg. Even lower levels of systolic BP may be required as long as vital organs (brain, heart, kidneys) maintain adequate perfusion.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 62, “Aortic Dissection and Related Aortic Disorders,” by Gary A. Johnson and Louise A. Prince; and Chapter 63, “Aneurysms of the Aorta and Major Arteries” by Louise A. Prince and Gary A. Johnson.

# CHAPTER 28

## Occlusive Arterial Disease

Carolyn K. Synovitz

*Peripheral arterial disease* is defined as an ankle-brachial index (ABI) of  $<0.9$  (see ABI definition below). The disease prevalence is 4.3% in Americans under age 40 years; prevalence climbs to 15.5% in those over 70 years of age. High-risk individuals (such as those over 70 years, or those over 50 years with risk factors such as diabetes or tobacco use), should be evaluated carefully when complaints are indicative of possible occlusive arterial disease. This time-sensitive condition can lead to irreversible changes in peripheral nerves and skeletal muscle tissue in 4 to 6 hours. The most frequently involved arteries, in descending order, are the femoropopliteal, tibial, aortoiliac, and brachiocephalic.

### Clinical Features

Patients with acute arterial limb ischemia typically present with one of the “six Ps”: pain, pallor, poikilothermia (coldness), pulselessness, paresthesias, and paralysis. Pain is the earliest symptom and may increase with elevation of the limb. Changes in skin color with mottling, splotchiness, and cool temperature are common. One of the early signs of ischemic limb pain may present as muscle weakness. Limb viability may be in question when there is acute anesthesia progressing to paralysis. A decreased pulse distal to the obstruction is an unreliable finding for early ischemia, especially in patients with peripheral vascular disease and well-developed collateral circulation. *Claudication* refers to a cramplike pain, ache, or tiredness that is brought on by exercise and relieved by rest. It is reproducible, resolves within 2 to 5 min of rest, and reoccurs at consistent walking distances. The pain of acute limb ischemia is not well localized, is not relieved by rest or gravity, and can be a worsening of chronic pain (if it is caused by a thrombotic event).

## Diagnosis and Differential

Although thromboembolic disease is the most common cause of acute arterial occlusion, the differential diagnosis may include: catheterization complications, vasculitis, Raynaud disease, thromboangiitis obliterans, blunt or penetrating trauma, or low-flow shock states such as sepsis. Most commonly, a history of an abruptly ischemic limb in a patient with atrial fibrillation or recent myocardial infarction is strongly suggestive of an embolus. A history of claudication suggests a thrombosis.

For more objective testing, a handheld Doppler can document blood flow or its absence in the affected limb. Duplex ultrasonography can detect an obstruction to flow with sensitivity greater than 85%. In addition, the ABI can be easily measured in the emergency department. Using a blood pressure cuff, place a Doppler US at the brachial artery and record the pressure of occlusion. Repeat the procedure on the leg, measuring the occlusion pressure of the posterior tibial and dorsalis pedis arteries. The ABI is the leg occlusion pressure divided by the arm occlusion pressure; normal ABI is  $> 0.9$ . With arterial occlusion with a blood pressure cuff, the ABI usually is markedly diminished with a ratio between 0.9 and 0.41. A ratio lower than 0.41:1 is usually found in limbs with critical ischemia. A pressure difference greater than 30 mm Hg between any two adjacent levels of the limb can localize the site of obstruction. The diagnostic gold standard is the arteriogram, which can define the anatomy of the obstruction and direct treatment of the limb.

## Emergency Department Care and Disposition

1. Patients with acute arterial occlusion should be stabilized. Fluid resuscitation and pain medications should be administered as needed. Dependent positioning can increase perfusion pressure. Obtain an ECG and consider echocardiography to assess for conditions associated with embolism.
2. It is standard procedure to initiate anticoagulation with **unfractionated heparin**. Dosing is weight-based: 80 units/kilogram intravenous bolus

followed by infusion of 18 units/kilogram/h. The activated partial thromboplastin time should be maintained between 55 and 80 seconds (1.5 to 2.5 times normal). Aspirin should also be administered. Use of thrombolytics is controversial, with no clear benefit.

3. Definitive treatment should be performed in consultation with a vascular surgeon and an interventional radiologist. Catheter-directed embolectomy is the preferred approach for occlusion caused by an embolus. Other options include thrombolysis and surgery.
4. All patients with an acute arterial occlusion should be admitted to a telemetry bed or to the intensive care unit, depending on the stability of the patient and the planned course of therapy.
5. Reperfusion injury after revascularization of the injury can result in myoglobinemia, renal failure, hyperkalemia, and metabolic acidosis, these complications account for one-third of deaths from occlusive arterial disease.
6. Chronic peripheral arterial disease patients who lack comorbidities and have no immediate limb threat, can be discharged on aspirin (75 milligrams daily), with close vascular surgical follow-up for reassessment and further care.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 64, "Occlusive Arterial Disease," by Anil Chopra and David Carr.

## **SECTION 5 Pulmonary Emergencies**

# **CHAPTER 29**

## **Respiratory Distress**

Joshua T. Hargraves

Causes of respiratory distress are multifactorial and include the findings of dyspnea, hypoxia, hypercapnia, and cyanosis. Despite the increasing reliance on ancillary studies and technology, the evaluation of respiratory distress depends on a careful history and physical examination.

### **DYSPNEA**

Dyspnea is the subjective feeling of difficult, labored, or uncomfortable breathing. There is no single pathophysiologic mechanism that causes dyspnea. However, most patients with dyspnea have a cardiac or a pulmonary cause.

#### **Clinical Features**

The initial assessment of any patient with dyspnea should be directed toward identifying respiratory failure. Dyspnea is a subjective complaint difficult to quantify. Vital signs (including pulse oximetry) and general impression will identify those in significant distress. Tachycardia, tachypnea, stridor, and the use of accessory respiratory muscles point to significant respiratory distress. Other significant signs include lethargy, agitation, altered mental status, and inability to speak due to breathlessness. In patients with any of these signs or symptoms, oxygen should be administered immediately. When there is no improvement, the need for aggressive airway management and mechanical ventilation should be anticipated. Lack of these significant signs and symptoms indicates a lesser degree of distress, thereby allowing for a detailed history and physical examination that often may help identify the etiology of dyspnea.

## Diagnosis and Differential

The history and physical examination should be the primary aids in identifying the etiology of dyspnea. However, ancillary testing may aid in determining the severity and specific cause ([Table 29-1](#)). Overall clinical gestalt is important, as are specific findings of an S<sub>3</sub> gallop and jugular venous distention. Pulse oximetry is a rapid but insensitive screen for disorders of gas exchange. Arterial blood gas (ABG) analysis has improved sensitivity but does not take into account work of breathing. ABG analysis may also demonstrate a metabolic acidosis, which can be a common cause of hyperpnea. A chest radiograph may identify pulmonary and cardiac causes of dyspnea. In addition, an abnormal electrocardiogram or elevated cardiac enzymes may point toward a cardiac cause of dyspnea. A peak expiratory flow rate may indicate reactive airway disease. Additional laboratory tests that may prove helpful include a complete blood count, B-type natriuretic peptide, and D-dimer assay. Uncommonly, the cause of dyspnea may not be identified. Specialized testing that may be indicated include computed tomography of the chest, echocardiography, pulmonary function testing, cardiac stress testing, nuclear medicine scans, or combined cardiopulmonary exercise testing.

Most Common Causes	Most Immediately Life-Threatening
Obstructive airway disease; asthma, COPD	Upper airway obstruction: foreign body, angioedema, hemorrhage
Congestive heart failure/cardiogenic pulmonary edema	Tension pneumothorax
Ischemic heart disease: unstable angina and myocardial infarction	Pulmonary embolism
Pneumonia	Neuromuscular weakness: myasthenia gravis, Guillain-Barré syndrome, botulism

Key: COPD = chronic obstructive pulmonary disease.

**TABLE 29-1** Causes of Dyspnea

## Emergency Department Care and Disposition

Just as there is no single cause of dyspnea, there is no single treatment. The following are general treatment guidelines for dyspnea.

1. Patients identified as having impending respiratory failure will need aggressive airway management and mechanical ventilation. Noninvasive ventilation techniques, such as continuous positive airway pressure and biphasic positive airway pressure, should be considered.
2. The goal of therapy is to maintain the PaO<sub>2</sub> above 60 mm Hg or the oxygen saturation above 90%. Lower goals are appropriate in those with long-standing lung disease such as chronic obstructive pulmonary disease (COPD).
3. After oxygenation has been insured, disorder-specific treatment and evaluation can be pursued.
4. The disposition of patients with dyspnea depends on its etiology. Any patient with hypoxia and an unclear cause of dyspnea requires hospital admission.

## **HYPOXEMIA**

Hypoxia is the inadequate delivery of oxygen to the tissues. Oxygen delivery is a function of cardiac output, hemoglobin concentration, and oxygen saturation. Hypoxemia is arbitrarily defined as a PaO<sub>2</sub> below 60 mm Hg, where oxygen saturation and blood oxygen content drop quickly. Hypoxemia results from a combination of five distinct mechanisms: (a) hypoventilation hypoxia in which lack of ventilation increases PaCO<sub>2</sub>, thereby displacing oxygen from the alveolus and lowering the amount delivered to the alveolar capillaries; (b) right-to-left shunt in which blood bypasses the lungs, thereby increasing the amount of unoxygenated blood entering the systemic circulation; (c) ventilation/perfusion mismatch in which areas of the lung are perfused but not ventilated; (d) diffusion impairment in which alveolar-blood barrier abnormality causes impairment of oxygenation; and (e) low inspired oxygen, such as occurs at high altitude.



## **Clinical Features**

Signs and symptoms of hypoxemia are nonspecific. Acute physiologic responses to hypoxemia include pulmonary arterial vasoconstriction and increases in minute ventilation and sympathetic tone manifesting as tachypnea, tachycardia, and an initial hyperdynamic cardiac state. The predominant features can be neurologic, and may include headache, somnolence, lethargy, anxiety, agitation, coma, or seizures. Chronic hypoxemia may result in polycythemia, digital clubbing, cor pulmonale, and changes in body habitus (eg, the “pink puffer” or “blue bloater” presentations of COPD). Cyanosis may be present but is not a sensitive or specific indicator of hypoxemia.

## **Diagnosis and Differential**

The diagnosis of hypoxemia requires clinical suspicion and objective measurement. Formal diagnosis requires ABG analysis, but pulse oximetry may be useful for gross abnormalities or trends. As is the case with dyspnea, the etiology of hypoxemia can be multifactorial. Determination of the exact cause is facilitated by thorough, careful history and physical examination. Hypoxemia may be quantified, and clues to its etiology may be obtained, by calculation of the Alveolar-arterial oxygen gradient (“A-a gradient,” where the capital “A” represents alveolar oxygen tension and “a” indicates arterial oxygen level). The formula for calculating A-a gradient while breathing room air at sea level is:

$$P(A-a)O_2 = 147 - (PaCO_2 \times 1.25) - PaCO_2$$

The A-a gradient is increased in cases of right-to-left shunts, ventilation-perfusion mismatch, and diffusion impairment. The normal value for a 20 year old nonsmoker is 5 to 10; the upper limit of normal increases by 1 for each decade of life.

## **Emergency Department Care and Disposition**

Regardless of the specific cause of hypoxemia, the initial approach remains the same. The following are general treatment guidelines for hypoxia:

1. Supplemental oxygen is administered to achieve an O<sub>2</sub> saturation greater than 90%.
2. The airway is managed aggressively as needed (see [Chapter 1](#)).
3. Cause-specific treatment and evaluation should be pursued.
4. All patients with new hypoxemia should be admitted and monitored until their condition is stabilized.

## **HYPERCAPNIA**

Hypercapnia is arbitrarily defined as a PaCO<sub>2</sub> above 45 mm Hg and is exclusively due to alveolar hypoventilation. Factors that affect alveolar ventilation include respiratory rate, tidal volume, and dead space volume. Alveolar ventilation is tightly controlled by the body to maintain PaCO<sub>2</sub> in a narrow range. Hypercapnia is almost never caused by increased CO<sub>2</sub> production.

### **Clinical Features**

The signs and symptoms of hypercapnia depend on PaCO<sub>2</sub>'s absolute value and rate of change. Acute elevations result in increased intracranial pressure, prompting patient complaints of headache, confusion, and lethargy. Coma, encephalopathy, and seizures may be present in cases in which the PaCO<sub>2</sub> acutely rises above 80 mm Hg; similar PaCO<sub>2</sub> levels may be well tolerated if elevations are chronic.

### **Diagnosis and Differential**

The diagnosis requires clinical suspicion and ABG analysis, pulse oximetry may be completely normal. In acute cases, the ABG will demonstrate an elevation in PaCO<sub>2</sub> with a respiratory acidosis and minimal metabolic compensation. Common causes of hypercapnia include COPD, respiratory center depression from drugs (eg, opiates, sedatives, anesthetics), neuromuscular impairment from disease (Guillain-Barré syndrome) or toxin (botulism), and finally thoracic cage disorders (morbid obesity, kyphoscoliosis).

## **Emergency Department Care and Disposition**

Treatment of acute hypercapnia requires aggressive measures to increase minute ventilation. The following are general treatment guidelines for hypercapnia:

1. Airway maintenance is crucial and mechanical ventilation may be indicated (see [Chapter 1](#)).
2. A trial of biphasic positive airway pressure or continuous positive airway pressure may prove helpful and improve minute ventilation. In some cases treatment should include condition-specific therapies such as bronchodilators for COPD, or reversal agents for opiate overdose.
3. Disposition depends on etiology, but many patients with hypercapnia require hospital admission and monitoring.

## **CYANOSIS**

Cyanosis is a bluish color of the skin or mucous membranes, resulting from an increased amount of deoxyhemoglobin. The detection of cyanosis is highly subjective and not a sensitive indicator of arterial oxygenation. Traditional teaching purports that cyanosis develops when the deoxyhemoglobin level exceeds 5 milligrams/dL, but this is greatly variable.

### **Clinical Features**

The presence of cyanosis suggests tissue hypoxia. The presence of cyanosis with a normal PaO<sub>2</sub> suggests an abnormal hemoglobin such as methemoglobin. Cyanosis is divided into central and peripheral. Central cyanosis, which is most reliably observed under the tongue or on the buccal mucosa, is due to inadequate pulmonary oxygenation or abnormal hemoglobins. Peripheral cyanosis is cyanosis of the distal extremities due to diminished peripheral blood flow.

## Diagnosis and Differential

The causes of cyanosis may be multifactorial ([Table 29-2](#)). In some cases, diagnosis is confounded by coexistence of central and peripheral cyanosis. Pulse oximetry is easily available for continuous monitoring, but it may overestimate oxygen saturation when there is dyshemoglobinemia. ABG analysis with cooximetry is the gold standard for diagnosis of cyanosis, since ABG alone may be misleading in the presence of abnormal hemoglobin. Methemoglobinemia and carboxyhemoglobinemia may cause cyanosis with a normal PaO<sub>2</sub>. Methemoglobinemia is associated with blood that has been described as chocolate brown, and which does not change color with exposure to room air. Classically, carboxyhemoglobin produces a cherry-red mucous membrane discoloration. A hematocrit may demonstrate polycythemia vera or severe anemia, both of which may contribute to cyanosis.

Central Cyanosis	Peripheral Cyanosis
Hypoxemia	Reduced cardiac output
Decreased FiO <sub>2</sub> : high altitude	Cold extremities
Hypoventilation	Maldistribution of blood flow:
Ventilation-perfusion mismatch	distributive forms of shock
Right-to-left shunt: congenital heart disease, pulmonary arteriovenous fistulas, multiple intrapulmonary shunts	Arterial or venous obstruction
Hemoglobin abnormalities	
Methemoglobinemia: hereditary, acquired	
Sulfhemoglobinemia: acquired	
Carboxyhemoglobinemia (not true cyanosis)	

Key: FiO<sub>2</sub> = fraction of inspired oxygen concentration.

## **TABLE 29-2** Causes of Cyanosis

### **Emergency Department Care and Disposition**

Patients with cyanosis require aggressive treatment and rapid identification of the underlying etiology. The following are general treatment guidelines for cyanosis:

1. Patients should be started on supplemental oxygen to achieve an oxygen saturation greater than 90%. Those with central cyanosis should improve rapidly. If there is no improvement, suspect impaired cardiac circulation, abnormal hemoglobin or pseudocyanosis.
2. Peripheral cyanosis should respond to therapy directed at the specific condition causing the cyanosis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 65, “Respiratory Distress,” by John Sarko, and J. Stephen Stapczynski.

## **CHAPTER 30**

# **Bronchitis, Pneumonia, and SARS**

Jeffrey M. Goodloe

## **BRONCHITIS**

Acute bronchitis is a commonly encountered, self-limited, viral infection producing inflammatory changes within the larger airways of the lung. Sharing the viral pathogens of upper respiratory infections, including those of the common cold, acute bronchitis is often caused by one of the following: influenza A or B, parainfluenza, respiratory syncytial virus, or coronavirus.

### **Clinical Features**

The predominant cough of acute bronchitis may be productive and can easily last up to 3 weeks. Sputum purulence is usually indicative of sloughed inflammatory airway cells and, taken alone, does not indicate a bacterial etiology. Bronchitis lacks the suggestive symptoms and signs of pneumonia, specifically fever  $> 38^{\circ}\text{C}$  ( $100.4^{\circ}\text{F}$ ), adult heart rate  $> 100$  beats/min, and/or adult respiratory rate  $> 24$  breaths/min. Wheezing may be present.

### **Diagnosis and Differential**

The diagnosis of acute bronchitis is made clinically with the following criteria: (a) acute onset cough (shorter than 3 weeks' duration), (b) absence of chronic lung disease history, (c) normal vital signs, and (d) no auscultatory abnormalities that suggest pneumonia. Pulse oximetry is

indicated if the patient describes dyspnea or appears short of breath. Bedside peak flow testing may prove illustrative of reductions in forced expiratory volume in 1 second in over half of patients and is best indicated if wheezing is heard on examination. A chest radiograph is not required in non-elderly patients who appear nontoxic. Among the differential etiologies of cough prolonged beyond 3 weeks, consider pertussis in adolescents and young adults, particularly if eliciting a known contact with a confirmed pertussis case or coughing paroxysms with prominent posttussive emesis.

## **Emergency Department Care and Disposition**

1. The use of antibiotics for acute bronchitis, while commonly requested by patients and prescribed by practitioners, does NOT confer clinically relevant benefits in a viral illness, but produces side effects such as gastrointestinal distress, vaginitis, and future pathogen resistance.
2. If pertussis is strongly suspected, **azithromycin** is indicated, in adults, day 1 with 500 milligrams orally, followed by days 2 to 5 with 250 milligrams orally. This treatment does not shorten the illness, but decreases coughing paroxysms and limits disease transmission.
3. Patients with evidence of airflow obstruction should be treated with bronchodilators. **Albuterol** by metered dose inhaler using a spacer, adult dosage of 2 puffs every 4 to 6 hours, is usually effective in symptomatic relief of dyspnea and cough reduction.
4. Additional agents for cough suppression, mucolysis, and symptomatic relief may be considered on an individual basis factoring comorbidities, drug interactions, and potential side effects.
5. Discharge instructions should encourage timely follow-up with a primary care physician, smoking cessation when applicable, and when to return to the emergency department based upon clinical symptoms.

## **PNEUMONIA**

Pneumonia, most commonly a bacterial infection of the alveolar lung, afflicts millions in the United States yearly, remaining a leading cause of

morbidity and mortality. Pneumococcus (*Streptococcus pneumoniae*) is the classic bacterial etiology, though incidence from atypical and opportunistic agents, particularly if pneumonia is acquired in health care settings, is increasing. *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Haemophilus influenzae* are additional causative bacterial agents. *Legionella pneumophila*, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and a spectrum of respiratory viruses account for the bulk of atypical pneumonias. Anaerobes are less frequently encountered, but must be highly suspected in circumstances of likely aspiration. Risk factors for pneumonia are multiple, and include diseases of the respiratory tract (eg, chronic obstructive pulmonary disease or COPD) and immune system (eg, cancer, AIDS), as well as chronic conditions associated with aspiration, bacteremia, and debilitation.

## Clinical Features

Patients with undifferentiated bacterial pneumonia typically present with some combination of cough, fatigue, fever, dyspnea, sputum production, and pleuritic chest pain. Physical examination often reveals tachypnea, tachycardia, low pulse oximetry, and the auscultatory findings of bronchial breath sounds and rhonchi suggestive of consolidation. Impaired air passage may be indicated by wheezing. While historical features and associated symptoms and signs can prove helpful in predicting a likely causative organism, the treatment of pneumonia has shifted to empiric treatment based on the patient's environment. The clinician should differentiate between community acquired versus healthcare-associated pneumonia with its risk for organisms that require specific and/or broadened antibiotic coverage, such as *Pseudomonas aeruginosa* and/or methicillin-resistant *Staphylococcus aureus*. Patients meeting criteria for health care associated pneumonia include patients hospitalized over 48 hours within the prior 90 days, those receiving routine outpatient treatments of dialysis, chemotherapy, wound care, or home IV antibiotic therapy, and residents in a nursing facility.

Clinical features of aspiration pneumonitis depend on the volume and pH of the aspirate, the presence of particulate matter in the aspirate, and



bacterial contamination. Although aspiration of acidic, larger volumes result in a relatively rapid onset of tachypnea, tachycardia, and respiratory distress that may progress to pulmonary failure, most cases of aspiration pneumonia progress insidiously. While aspiration pneumonias may occur anywhere in the lung, aspirated material has a predilection for the right lower lobe (due to gravity and tracheobronchial tree anatomy). Untreated or partially treated aspiration pneumonia may progress to empyema, defined as pus in the pleural space, or a lung abscess.

## **Diagnosis and Differential**

Uncomplicated presentations in otherwise healthy patients may not require use of radiology, laboratory, or pathology resources, however, chest radiography is most commonly used for diagnosis (see [Fig. 30-1](#)). Depending upon the anticipated etiology and disposition course, assessing white blood count with differential analysis, serum electrolytes, blood urea nitrogen, creatinine, glucose, blood gases, sputum gram staining, and cultures of sputum and blood provide benefit, particularly in patients requiring intensive care unit admission. Most patients do not require identification of a specific organism to make a diagnosis and begin treatment. Due to the numerous special patient populations, the parent text should often be consulted for additional guidance in complicated cases.



**FIGURE 30-1.** Pneumonia suggesting *Legionella*.

The differential diagnosis of nontrauma patients with respiratory complaints and radiographic abnormality is lengthy and partially includes: noninfectious atelectasis; chronic pulmonary fibrosis; pleural effusion; chemical pneumonitis; inflammatory disorders, such as sarcoidosis; neoplasm; postsurgical changes; tuberculosis; bronchiolitis obliterans; pulmonary embolus; congestive heart failure; and pulmonary vasculitides, such as Goodpasture disease or Wegener granulomatosis. Further differential diagnosis details can be found in the parent text.

### **Emergency Department Care and Disposition**

1. Vital respiratory function (oxygenation, ventilation) should be supported as indicated, with rapidly impending or unresponsive respiratory failure managed via intubation and mechanical ventilation. Noninvasive ventilation may prevent the need for intubation.
2. The complexity of pneumonia severity scoring as a means to determine discharge or admission prevents inclusion in this manual. In general,

progressive degrees of abnormal vital signs, comorbidities, and advancing age confer increased need for inpatient management.

3. Antibiotic treatment should be initiated in all cases of suspected bacterial pneumonia, with the specific choice(s) made considering the patient's recent environment, differentiating community acquired pneumonia (CAP) from health care associated pneumonia, comorbidities, drug allergies, drug-drug interactions, and local resistance patterns.
4. Specialty society guidelines and infectious disease consultation advice change with advent of antimicrobials and resistance patterns. The antimicrobials listed here represent a summary of current and generally accepted antibiotic regimens for adults with the indicated clinical situations. Dosages may require adjustment for renal insufficiency. (Refer to [Chapter 68](#) in the parent text for more details.)
5. Outpatient management of uncomplicated CAP in otherwise healthy patients: **azithromycin** day 1 with 500 milligrams orally, followed by days 2 to 5 with 250 milligrams orally or **doxycycline** 100 milligrams orally twice daily for 10 days (this is a low-cost alternative). The Centers for Disease Control and Prevention (CDC) recommends reserving oral fluoroquinolones for those failing macrolide or tetracycline class therapy to minimize resistance.
6. Outpatient management of CAP in patients with significant comorbidities (and without healthcare-associated pneumonia suspected): **levofloxacin** 750 milligrams orally daily for 5 days or **amoxicillin-clavulanate** 875/125 milligrams orally twice daily for 10 days plus **azithromycin** day 1 with 500 milligrams orally, followed by days 2 to 5 with 250 milligrams orally.
7. Inpatient management of CAP in patients not requiring ICU admission: initiate **levofloxacin** 750 milligrams IV or **ceftriaxone** 1 gram IV plus **azithromycin** 500 milligrams IV. Utilize antibiotics early in the course of any pneumonia requiring admission.
8. Inpatient management of CAP in patients requiring ICU admission: initiate **ceftriaxone** 1 gram IV plus **levofloxacin** 750 milligrams IV. If methicillin-resistant *S aureus* (MRSA) suspected, add **vancomycin** 10 to 15 milligrams/kilogram IV to the regimen.
9. Inpatient management of suspected healthcare-associated pneumonia: initiate double coverage against *Pseudomonas* with **levofloxacin** 750

milligrams IV plus **cefepime** 1 to 2 grams IV every 8 to 12 hours or **piperacillin/tazobactam** 4.5 grams IV every 6 hours. Also, initiate coverage against MRSA with **vancomycin** 10 to 15 milligrams/kilogram IV or **linezolid** 600 milligrams IV every 12 hours.

10. Aspiration: In aspiration-induced pneumonitis, prophylactic antibiotics are not recommended and their indiscriminate use may contribute to organism resistance. For witnessed aspirations, immediate tracheal suction followed by bronchoscopy (if needed to remove large particles) is indicated. In pneumonitis that has already progressed to pneumonia prior to or shortly after emergency department presentation, initiate **levofloxacin** 750 milligrams IV plus **clindamycin** 600 milligrams IV every 6 hours.
11. Empyema: initiate **piperacillin/tazobactam** 4.5 grams IV every 6 hours. If MRSA suspected, add **vancomycin** 10 to 15 milligrams/kilogram IV to the regimen. The patient should be admitted with early consultation with a pulmonologist or thoracic surgeon for further consideration of definitive diagnostic measures and treatment options to promote drainage.
12. Lung abscess: initiate **clindamycin** 600 milligrams IV every 6 hours for anaerobic coverage plus **ceftriaxone** 1 gram IV every 12 hours. Inpatient medical management successfully treats a significant majority of lung abscesses; surgical consultation is required in only a minority of cases.
13. Discharge instructions should, at a minimum, include: timely follow-up with a primary care physician, smoking cessation (when applicable), and delineation of symptoms that should prompt a return visit to the emergency department.

## **SEVERE ACUTE RESPIRATORY SYNDROME**

Severe acute respiratory syndrome (SARS) came to worldwide attention in the winter of 2003. Numerous deaths were reported in Asia, North America, and Europe. The etiologic agent is a coronavirus, SARS-CoV, spread by “droplet infection”. In the event of SARS outbreak, up-to-date information

can be found at the CDC Web site (<http://www.cdc.gov/ncidod/sars/>) as well as at the WHO Web site (<http://www.who.int/topics/sars/en/>).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 67, “Acute Bronchitis and Upper Respiratory Tract Infections,” by Thomas A. Tallman; Chapter 68, “Community-Acquired Pneumonia, Aspiration Pneumonia, and Noninfectious Pulmonary Infiltrates,” by Charles L. Emerman, Eric Anderson, and David M. Cline; and Chapter 69, “Empyema and Lung Abscess,” by Eric Anderson and Sharon E. Mace.

# **CHAPTER 31**

## **Tuberculosis**

Amy J. Behrman

Tuberculosis (TB) is a major global health problem, infecting one-third of the world's population and causing approximately 2 million deaths annually. In the United States, TB is an important public health problem, particularly among immigrants whose active TB case rate is 11 times higher than that of nonimmigrants. Other risk factors include HIV infection; living or working in prison, shelters, and long-term care facilities; caring for TB patients; and alcohol/drug abuse.

## **CLINICAL FEATURES**

### **Primary TB**

Primary TB infection is usually asymptomatic in immune-competent adults, generally presenting with only a new positive reaction to TB skin testing. When present, symptoms often include fever, cough, weight loss, malaise and chest pain. Some patients may present with active pneumonitis (which may be mistaken for community-acquired pneumonia) or extrapulmonary disease.

Children are more likely to present with active early disease, although the presenting symptoms may be subtle even when chest radiographs (CXR) are abnormal. Presenting symptoms may include fever, cough, wheezing, poor feeding, and fatigue. TB meningitis and military TB (see descriptions below) are more common in children than adults.

Immunocompromised patients are much more likely to develop rapidly progressive primary infections. (All patients with active TB should be evaluated for immune-compromising conditions.) Symptoms may be pulmonary (fever, cough, dyspnea, hemoptysis) or may be extrapulmonary,

reflecting early hematogenous spread to the central nervous system or other sites.

## **Reactivation TB**

Latent tuberculosis infections are asymptomatic with positive tuberculin skin tests (TSTs) and/or positive interferon-gamma release assays (IGRAs). Latent tuberculosis infections will progress to active disease (ie, reactivation TB) in 5% of cases within 2 years of primary infection; an additional 5% will reactivate over their lifetimes. Reactivation rates are much higher in the very young, the elderly, persons with recent primary infection, those with immune compromise (in particular, HIV), and those with chronic diseases such as diabetes and renal failure.

Most patients with reactivation TB present sub-acutely with fever, malaise, weight loss, fatigue, and night sweats. Most patients with active TB will have pulmonary involvement characterized by subsequent development of productive cough. Hemoptysis, pleuritic chest pain, and dyspnea may develop. Rales and rhonchi may be found, but the pulmonary examination is not usually diagnostic. TB should be considered in any HIV patient with respiratory symptoms, even if chest radiographs are normal (see [Chapter 92](#) HIV and AIDS).

Extrapulmonary TB develops in up to 20% of cases. Lymphadenitis, with painless enlargement and possible draining sinuses, is the most common presentation. Patients may also present with symptomatic pleural effusion, pericarditis, peritonitis, or meningitis. Additional sites of reactivation TB after hematogenous spread include bones, joints, adrenals, GI tract and GU tract. Extrapulmonary reactivation TB is more common and often more severe in young children and immune-compromised patients as noted for primary TB infection above.

Miliary TB is a multisystem disease caused by massive hematogenous dissemination. It is also more common in immune-compromised patients and young children. Symptoms are systemic with fever, weight loss, adenopathy and malaise. Patients may present with multiorgan failure or ARDS.

# DIAGNOSIS AND DIFFERENTIAL

Variable presentations and limited testing options make TB diagnosis particularly challenging in the ED. Consider the diagnosis of active TB in any patient with respiratory or systemic complaints, in order to facilitate early diagnosis and treatment and to reduce exposure risks. Differential diagnoses may include other infectious causes of pulmonary and extrapulmonary lesions as well as malignancy.

## Imaging

CXR is still the most useful ED diagnostic tool for TB. Active primary pulmonary TB usually presents with parenchymal infiltrates in any lung area. Hilar and/or mediastinal adenopathy may occur with or without infiltrates. Effusions, usually unilateral, may be seen with or without infiltrates.

Reactivation TB classically presents with lesions in the upper lobes or superior segments of the lower lobes ([Fig. 31-1](#)). Cavitation, calcification, scarring, atelectasis, hilar adenopathy, and effusions may be seen. Cavitation is associated with increased infectivity. Miliary TB may cause diffuse, small (1 to 3 mm) nodular infiltrates. Patients coinfecting with HIV and TB are particularly likely to present with atypical chest radiographs.





**FIGURE 31-1.** Cavitary tuberculosis of the right upper lobe.

Scarring, volume loss, and calcified or noncalcified nodules may be identified (often as incidental findings) in patients with asymptomatic latent TB infection; these patients do not require urgent treatment or isolation. Comparison with prior films may be very useful in determining the likelihood of active TB infection.

### **Laboratory Studies**

Acid-fast staining of sputum can detect mycobacteria in 60% of patients with pulmonary TB, although the yield is lower in HIV patients. Results may be available within several hours, which increases potential ED utility, but there are serious limitations. Many patients will have false negatives on a single sputum sample. Microscopy of nonsputum samples (eg, pleural fluid, cerebrospinal fluid) is even less sensitive. Microscopy cannot differentiate between TB and nontuberculous mycobacteria.

Culture of sputum (or other specimens) is the gold standard for diagnosing active TB. Unfortunately, definitive culture results generally

take 4 to 6 weeks. When available, newer technologies such as TB-specific nucleic acid amplification can produce results within 24 hours (thus rendering these tests potentially applicable to ED management). These tests have better positive predictive value than acid-fast staining and may also enable more cost-effective utilization of isolation, treatment, and contact-tracing resources. Tuberculin skin tests (TST), identify most patients with latent, prior, or active TB. Results are read 48 to 72 hours after placement, limiting the ED utility of this approach. Patients with HIV or other immunosuppressive conditions, and patients with disseminated TB, may have false negative TSTs. Immigrants who received BCG vaccine in childhood may have false positive TSTs. Finally, the TST may become paradoxically negative during active TB infection.

Interferon-gamma release assays (IGRA) of whole blood may become more useful than TST for ED evaluation of suspected TB, since these tests may produce results within hours. IGRAs appear to be equally sensitive to, and more specific than, TST. The fact that prior BCG vaccination should not cause false positive results makes IGRA particularly useful when evaluating immigrants from high-prevalence countries.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. ED and prehospital staff should be trained to identify patients at risk for active TB early in their evaluation and isolation protocols should be enacted (see parent chapter).
2. Therapy should include at least 4 drugs until susceptibility profiles are available for a patient. The initial regimen usually includes: **isoniazid**, INH (5 milligrams/kilogram up to 300 milligrams PO daily in adults, 10 to 15 milligrams/kilogram up to 300 milligrams PO daily in children), rifampin (10 milligrams/kilogram up to 600 milligrams PO daily in adults, 10 to 20 milligrams/kilogram up to 600 milligrams PO daily in children), pyrazinamide (20 to 25 milligrams/kilogram up to 2 grams PO daily in adults, 15 to 30 milligrams/kilogram up to 2 grams PO daily in

children), and ethambutol (15 to 20 milligrams/kilogram up to 1.6 grams PO daily in adult, 15 to 20 milligrams/kilogram up to 1 gram PO daily in children) for 2 months. Give pyridoxine 10 to 50 milligrams/day with INH. At least 2 drugs (usually INH and rifampin) are continued for 18 to 31 months. An alternative to rifampin in adults is rifabutin (5 milligrams/kilogram up to 300 milligrams PO daily). Directly observed therapy (DOT) may improve outpatient compliance with these complex regimens (see parent chapter for DOT recommendations and more treatment options, or see the Centers for Disease Control and Prevention website <http://www.cdc.gov/tb/>). Patients with immune compromise or multidrug resistant (MDR) TB may require more drugs for longer periods. When available, culture and sensitivity results are used to tailor the antimicrobial regimen.

3. Concern for MDR TB should be high in patients with history of birth in or travel to endemic areas. MDR TB is also more likely in patients who have previous TB treatment, HIV infection, cavitary disease, or known acid-fast positive sputum smears. Treatment of known or suspected MDR TB begins with at least 4 oral drugs plus an injectable agent (eg, spectinomycin, amikacin, capreomycin). ID consultation is appropriate for MDR TB.
4. Admission is indicated for clinical instability, hypoxia, dyspnea, diagnostic uncertainty, unreliable outpatient follow-up or compliance, and suspected or known MDR TB. ED physicians should know local laws and resources regarding involuntary hospitalization and treatment (including DOT). Patients with suspected TB should wear masks during all transport, and these cases should be admitted to airborne-isolation single rooms.
5. Patients with active TB who are discharged from the ED must have documented immediate referral to a physician or local public health department for long-term treatment and contact tracing. Most TB can be treated in the outpatient setting with closely monitored daily or intermittent regimens. TB treatment should usually be initiated by the ambulatory providers who will be monitoring compliance and adverse events, rather than by the ED physician. Persons with latent TB infection should be referred to primary care or public health clinics for INH treatment (to prophylax against reactivation TB).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 70, “Tuberculosis,” by Vu D. Phan and Janet M. Poponick.

## **CHAPTER 32**

# **Spontaneous and Iatrogenic Pneumothorax**

Rodney L. McCaskill

Pneumothorax occurs when air enters the potential space between the parietal and visceral pleura, leading to partial lung collapse. Smoking is the most common risk factor for spontaneous pneumothorax, which most likely results from subpleural bulla rupture. Primary pneumothorax occurs in patients without known lung disease and secondary pneumothorax occurs most often in patients with chronic obstructive pulmonary disease, but other underlying diseases such as asthma, cystic fibrosis, interstitial lung disease, cancer, and *Pneumocystis carinii* pneumonia have been implicated. Iatrogenic pneumothorax occurs secondary to invasive procedures such as needle biopsy of the lung, placement of a subclavian line, nasogastric tube placement, or positive pressure ventilation. Tension pneumothorax results from positive pressure in the pleural space leading to decreased venous return, hypotension, and hypoxia. Hemopneumothorax occurs in 2% to 7% of patients with spontaneous pneumothorax.

## **CLINICAL FEATURES**

Symptoms resulting from a pneumothorax are directly related to its size, rate of development, and the health of the underlying lung. Acute onset of pleuritic pain is found in most patients, whereas a large volume pneumothorax may cause dyspnea, tachycardia, hypotension, and hypoxia. Decreased breath sounds on the affected side have a positive predictive value between 86% and 97%. Hypotension, tracheal deviation, and hyperresonance of the affected side are the hallmarks of tension pneumothorax.

## **DIAGNOSIS AND DIFFERENTIAL**

Pneumothorax is usually diagnosed by posteroanterior chest x-ray which has a sensitivity of 83%. Expiratory films are no more helpful than inspiratory films. Chest CT is more sensitive and may be useful in patients with bullous changes on x-ray. Ultrasound can be used and has a sensitivity approaching 100%. Ultrasound signs include absence of lung sliding in real-time (100% sensitive but not specific), the demonstration of a “lung point” (66% sensitive and nearly 100% specific), and absence of normal vertical comet-tail artifacts. The clinician should be aware that a pneumothorax can be associated with ST changes and T wave inversion on EKG.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. In patients with unstable vital signs and clinical features suggestive of tension pneumothorax, immediate needle thoracostomy followed by tube thoracostomy is indicated. X-rays should not be obtained before treatment.
2. In stable patients, oxygen 2 to 4 L/min by nasal canula helps increase resorption of intrapleural air.
3. Patients with small primary pneumothoraces may be observed for 6 hours and discharged with surgical follow-up if there is no enlargement on repeat x-ray. However, many eventually require tube thoracotomy.
4. A catheter or needle may be used to simply aspirate a small pneumothorax with success rates from 37% to 75%. The patient can be discharged with surgical follow-up at 6 hours postaspiration if there is no pneumothorax recurrence.
5. A small catheter can be used to aspirate the pleural space and then attached to a Heimlich valve and left secured in place. The patient may be discharged at 6 hours postaspiration with surgical follow-up if there is no pneumothorax recurrence.
6. Tube thoracostomy and admission are indicated for failed aspiration, large pneumothorax, recurrent pneumothorax, bilateral pneumothoraces,

hemothorax, abnormal vital signs, or severe dyspnea. The success rate for tube thoracostomy is 95%. Tube thoracostomy also should be considered in patients undergoing general anesthesia, positive pressure ventilation, or helicopter transport.

Complications of treatment include development of tension pneumothorax, failure of lung reexpansion, persistent air leak, infection, and reexpansion pulmonary edema. Reexpansion pulmonary edema is rare, and tends to occur in younger patients with large volume pneumothorax; treatment is supportive.

## **Iatrogenic Pneumothorax**

Iatrogenic pneumothorax results from an invasive procedure. Transthoracic needle procedures account for 50% of iatrogenic pneumothoraces and subclavian vein catheterization accounts for an additional 25%. A postprocedure chest x-ray is indicated, although the clinician should remember that the presentation can be delayed after subclavian line placement. Treatment is generally the same as for spontaneous pneumothorax.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 71, "Spontaneous and Iatrogenic Pneumothorax," by Roger L. Humphries and William Franklin Young, Jr.

## CHAPTER 33

# Hemoptysis

Jeffrey Dixon

Hemoptysis is the expectoration of blood from the lower respiratory tract. *Massive hemoptysis*, defined somewhat arbitrarily as a bleeding rate exceeding 600 mL per 24 hours, constitutes an emergency and requires prompt intervention to prevent asphyxiation from impaired gas exchange. Minor hemoptysis, the production of smaller quantities of blood (often mixed with mucus), is rarely life threatening but requires careful ED management.

## CLINICAL FEATURES

Hemoptysis may be the presenting symptom for many different diseases. A careful history should focus on the presence of underlying lung disease or history of tobacco use. The acute onset of fever, cough, and bloody sputum may indicate pneumonia or bronchitis. An indolent productive cough can indicate bronchitis or bronchiectasis. Dyspnea and pleuritic chest pain are potential indicators of pulmonary embolism. Tuberculosis should be considered in the setting of fevers or night sweats. Bronchogenic carcinoma may present with chronic weight loss and a change in cough. Chronic dyspnea and minor hemoptysis may indicate mitral stenosis or alveolar hemorrhage syndromes (most commonly seen in patients with renal disease).

The physical examination, which is usually not helpful in localizing the site of bleeding, is aimed at assessing the severity of the hemoptysis. The examination may also provide clues to the underlying disease process. Common signs include fever and tachypnea. Tachypnea may be a sign of respiratory compromise with hypoxemia. Hypotension is an ominous sign, usually seen only in massive hemoptysis. The cardiac examination may reveal signs of valvular heart disease (eg, the diastolic murmur of mitral



stenosis). The nasal and oral cavities should be inspected carefully to help rule out an extrapulmonary source of bleeding (pseudohemoptysis).

## **DIAGNOSIS AND DIFFERENTIAL**

A careful history and physical examination may suggest a diagnosis. Pulse oximetry and a chest x-ray (PA and lateral, if the patient's condition allows) are always indicated. Other tests that may be helpful include arterial blood gas, hemoglobin and hematocrit levels, platelet count, coagulation studies, urinalysis, and electrocardiogram. Chest CT should be considered if there is hemoptysis with an abnormal chest radiograph. The long differential diagnosis list includes infectious, neoplastic, and cardiac etiologies. Infectious etiologies include bronchitis, bronchiectasis, bacterial pneumonia, tuberculosis, fungal pneumonia, and lung abscess. Neoplastic etiologies include bronchogenic carcinoma and bronchial adenoma. Cardiogenic etiologies include mitral stenosis and left ventricular failure. Trauma, foreign body aspiration, pulmonary embolism (hemoptysis is one of the Wells criteria), primary pulmonary hypertension, pulmonary vasculitis, and bleeding diathesis are other potential causes.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Supplemental oxygen should be administered as needed, to maintain adequate oxygenation.
2. Normal saline or lactated Ringer's solution should be administered initially for hypotension.
3. Blood should be typed and cross-matched if transfusion is necessary. Packed red blood cells should be transfused as needed.
4. Fresh frozen plasma (2 units) should be administered to patients with coagulopathies, including those taking warfarin; platelets should be given for thrombocytopenia (see [Chapter 137](#) "Transfusion Therapy").

5. Patients with ongoing massive hemoptysis may benefit from being placed in the decubitus position, with the bleeding lung in dependent position.
6. **Hydrocodone** (5 to 15 milligrams every 4 to 6 hours) or other opioids may provide some cough suppression.
7. Endotracheal intubation with a large diameter (8.0 mm) tube, which facilitates bronchoscopy, should be performed if there is respiratory failure or if the patient cannot clear blood or secretions from the airway.
8. Any patient with moderate to severe hemoptysis requires admission to the hospital, and strong consideration should be given to placement in the intensive care unit. Patients with mild hemoptysis who have conditions that predispose them to severe bleeding also should be considered for admission to an intensive care unit. The advice of a pulmonologist or thoracic surgeon is required for decisions as to whether bronchoscopy, computed tomography, or angiography for bronchial artery embolization might be needed. If the appropriate specialists are not available, the patient should be stabilized and then transferred to another facility.
9. Patients who are discharged home should be treated for several days with cough suppressants inhaled  $\beta$ -agonist bronchodilators as needed, and antibiotics if bacterial infection is thought to be the cause. Close follow-up is essential, particularly in those at high risk for neoplasm.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 66, "Hemoptysis," by William Franklin Young, Jr.

## CHAPTER 34

# Asthma and Chronic Obstructive Pulmonary Disease

Joshua Gentges

Although most asthmatic attacks are mild and reversible, severe attacks can be fatal. COPD is the fourth leading cause of death in the world and is the only major cause of death that is increasing in frequency.

## CLINICAL FEATURES

Asthma is reversible airway obstruction associated with hyperresponsiveness of the tracheobronchial tree. COPD has 2 dominant forms: (*a*) pulmonary emphysema, characterized by abnormal, permanent enlargement and destruction of the air spaces distal to the terminal bronchioles; and (*b*) chronic bronchitis, a condition of excess mucous secretion in the bronchial tree, with a chronic productive cough occurring on most days for at least 3 months in the year for at least 2 consecutive years. Elements of both forms are often present, although one predominates.

Acute exacerbations of asthma and COPD are usually triggered by smoking, exposure to noxious stimuli (eg, pollutants, cold, stress, antigens, or exercise), adverse response to medications (eg, decongestants,  $\beta$ -blockers, nonsteroidal anti-inflammatory drugs), allergic reactions, and noncompliance with prescribed therapies. Triggers and complications of asthma and COPD include respiratory infection, pneumothorax, myocardial infarction, dysrhythmias, pulmonary edema, chest trauma, metabolic disorders, and abdominal processes.

Classically, patients with exacerbations of asthma or COPD present with complaints of dyspnea, chest tightness, wheezing, and cough. Physical examination shows wheezing with prolonged expiration. Wheezing does not correlate with the degree of airflow obstruction; a “quiet chest” may indicate severe airflow restriction. Patients with severe attacks may be

sitting upright with forward posturing, with pursed-lip exhalation, accessory muscle use, paradoxical respirations, and diaphoresis. Pulsus paradoxus of 20 mm Hg or higher may be seen. Severe airflow obstruction and ventilation/ perfusion imbalance can cause hypoxia and hypercapnia. Hypoxia is characterized by tachypnea, cyanosis, agitation, apprehension, tachycardia, and hypertension. Signs of hypercapnia include confusion, tremor, plethora, stupor, hypopnea, and apnea. Impending respiratory failure may be signaled by alteration in mental status, lethargy, quiet chest, acidosis, worsening hypoxia, and hypercapnia.

## **DIAGNOSIS AND DIFFERENTIAL**

Emergency department diagnosis of asthma or COPD usually is made clinically. Severity can be gauged by clinical judgment and more objectively by peak expiratory flow (PEF) rate in cooperative patients. A PEF <40% of predicted in asthmatics or <100 L/min in COPD patients indicates a severe exacerbation. Pulse oximetry is a fast, easy, and noninvasive means for assessing and monitoring oxygen saturation during treatment, but it does not aid in predicting clinical outcomes and cannot predict hypercapnia or acidosis. Arterial blood gas (ABG) serves primarily to evaluate hypercapnia and acidosis in moderate to severe attacks. Compensated hypercapnia and hypoxia is common in COPD patients; comparison with previous ABG values is helpful. Normal  $PCO_2$  in the setting of an acute asthmatic attack is an ominous finding (indicating respiratory fatigue) if the patient's clinical appearance is poor. An arterial pH lower than that which is consistent with renal compensation implies acute hypercarbia or metabolic acidosis. Asthma and COPD can coexist or be mistaken for one another, but the larger diagnostic challenge is separating these diagnoses from other serious respiratory emergencies. Congestive heart failure (CHF) commonly coexists or mimics COPD and can also cause wheezing. Chest x-ray, BNP, and signs of fluid overload (jugular venous distention or hepatojugular reflux) help differentiate COPD from CHF. Chest x-ray is used to diagnose complications such as pneumonia and pneumothorax. Electrocardiograms are useful to identify

dysrhythmias or suspected ischemic injury. A high index of suspicion is necessary to rule out pulmonary embolism (PE). Sudden onset of symptoms, syncope or near syncope, or classical PE presentation (pleuritic chest pain, dyspnea, tachycardia, hypoxia) warrant further testing.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Although COPD patients often have more comorbidity than do those with asthma, the therapy for acute bronchospasm is similar to that for inflammation. Treatment should precede history-taking in acutely dyspneic patients, because patients may decompensate rapidly. Patients who appear acute should be placed on a cardiac monitor and undergo noninvasive blood pressure and continuous pulse oximetry monitoring. An intravenous line should be started in patients with moderate and severe attacks. The primary goal of therapy is to correct tissue oxygenation.

1. Empiric supplemental oxygen should be administered to correct SaO<sub>2</sub> above 90%. Oxygen may exacerbate hypercapnia in the setting of COPD. Perform an ABG measurement if there is concern for symptomatic hypercapnia.
2.  $\beta$ -Adrenergic agonists are first-line agents used to treat acute bronchospasm in COPD and asthma. Aerosolized forms (by nebulizer or metered-dose inhaler with a spacer) minimize systemic toxicity and are preferred. **Albuterol sulfate** 2.5 to 5 milligrams is the most common agent. Deliver doses every 20 min or as continuous nebulization (10 to 15 milligrams/h), titrating treatment to clinical response and signs of toxicity (tachycardia, hypertension, and palpitations). **Levalbuterol** can be given at half of the dose of albuterol, but administration by continuous nebulization has not been studied. **Terbutaline** (0.25 to 0.5 mL) or **epinephrine** 1:1000 (0.1 to 0.3 mL) SC may be administered to patients not tolerating aerosolized therapy. Epinephrine should be used with caution in the presence of underlying cardiovascular disease.

3. Steroids should be given in the ED, to patients with exacerbations of asthma and COPD. The initial dose is the equivalent of 40 to 60 milligrams of prednisone. Neither the choice of steroid nor the route of administration is critical. If the patient is unable to take oral medication, intravenous **methylprednisolone** 60 to 125 milligrams may be used. Additional doses may be given every 4 to 6 hours. Inhaled steroids are not indicated for the treatment of acute symptoms. A 5- to 10-day course of oral steroids (**prednisone** 40 to 60 milligrams/dose) is beneficial for discharged patients with a significant exacerbation of asthma or COPD.
4. Anticholinergics are useful adjuvants when given with other therapies. Nebulized **ipratropium** (500 milligrams = 2.5 mL) may be administered alone or mixed with albuterol. The effects of ipratropium peak in 1 to 2 hours and last 3 to 4 hours. Dosages may be repeated every 1 to 4 hours.
5. COPD patients with change in sputum color and volume may benefit from antibiotic therapy directed at *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. Commonly used agents include doxycycline, the macrolides, cephalosporins, and fluoroquinolones. Antibiotic use in asthma should be reserved for concurrent bacterial infections such as pneumonia.
6. Intravenous **magnesium sulfate** (1 to 2 grams over 30 min) is used when asthma exacerbations are severe ( $FEV_1 < 25\%$  of predicted). However, it is not currently recommended for mild or moderate asthma exacerbations and should not be substituted for standard regimens.
7. Several studies have suggested that an 80%:20% mixture of helium and oxygen (Heliox) can lower airway resistance and aid in drug delivery in the patient with very severe asthma exacerbation. Care must be taken with use of this therapy in the oxygen-dependent patient since Heliox administration necessarily entails an  $FIO_2$  less than 1.0.
8. Mechanical ventilation is necessary in patients with respiratory muscle fatigue, respiratory acidosis, altered mental status, or hypoxia refractory to standard therapies. Noninvasive partial pressure ventilation (NPPV) has become a useful alternative to intubation and invasive ventilation. NPPV lowers intubation rates, short-term mortality, and length of hospitalization in COPD. NPPV can be given by continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP).

BiPAP has the advantage of reducing the work of breathing. CPAP is titrated up to 15 cm H<sub>2</sub>O, while BiPAP settings are between 8 and 20 cm H<sub>2</sub>O for inspiration and 4 and 15 cm H<sub>2</sub>O for expiration.

9. Patients in whom NPPV is likely to fail include those in whom there is poor mask fit or inability to cooperate with (or tolerate) NPPV, those who are obtunded, cannot clear airway secretions, or are hemodynamically unstable. NPPV is also not viable in extremely obese patients, or those who are status-post recent facial or gastroesophageal surgery or facial burns. When NPPV is not a viable option, oral intubation is indicated. Using rapid inspiratory flow rates at a reduced respiratory frequency (12 to 14 breaths/min), while allowing for an adequate expiratory phase, may help reduce air-trapping and subsequent barotrauma. Therapy should be guided by pulse oximetry and ABG results. Sedation and continued therapy for bronchospasm should continue after the patient has been placed on mechanical ventilation.
10. Admission criteria for patients with asthma include failure of outpatient treatment, persistent and worsening dyspnea, PEF or forced expiratory volume in 1 second (FEV<sub>1</sub>) less than 40% of predicted, hypoxia, hypercarbia, and altered mental status; presence of comorbidities increases likelihood of need for admission. As compared to asthmatics, patients with acute COPD exacerbations are more likely to require admission. Indications for COPD admission include therapeutic failure, severe dyspnea, significant comorbidities, arrhythmias, older age, insufficient home support, worsening hypoxia and hypercapnia with acidosis, and impaired mental status.

In the absence of intubation, sedatives, hypnotics, and other medications that depress respiratory drive are generally contraindicated. Methylxanthines do not improve lung function in acute asthma or COPD.  $\beta$ -Blockers may exacerbate bronchospasm. Antihistamines and decongestants should be avoided as they diminish clearance of respiratory secretions. Mucolytics should also be withheld; they may exacerbate bronchospasm. Mast cell and leukotriene modifiers have no role in the treatment of acute exacerbations of asthma or COPD. Ketamine and halothane have been reported as therapy for refractory asthma, but controlled studies are lacking.

Close follow-up care must be arranged for discharged patients to ensure resolution of the exacerbation and review the management plan. Despite appropriate therapy, these patients have high relapse rates. Education of the asthma and COPD patients before discharge (ie, review of medications, inhaler techniques, use of PEF measurements, avoidance of noxious stimuli, and need to follow-up) should be an integral part of ED care.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 72, “Acute Asthma in Adults,” by Rita K. Cydulka; and Chapter 73, “Chronic Obstructive Pulmonary Disease,” by Rita K. Cydulka and Craig G. Bates.



## **SECTION 6 Gastrointestinal Emergencies**

# **CHAPTER 35**

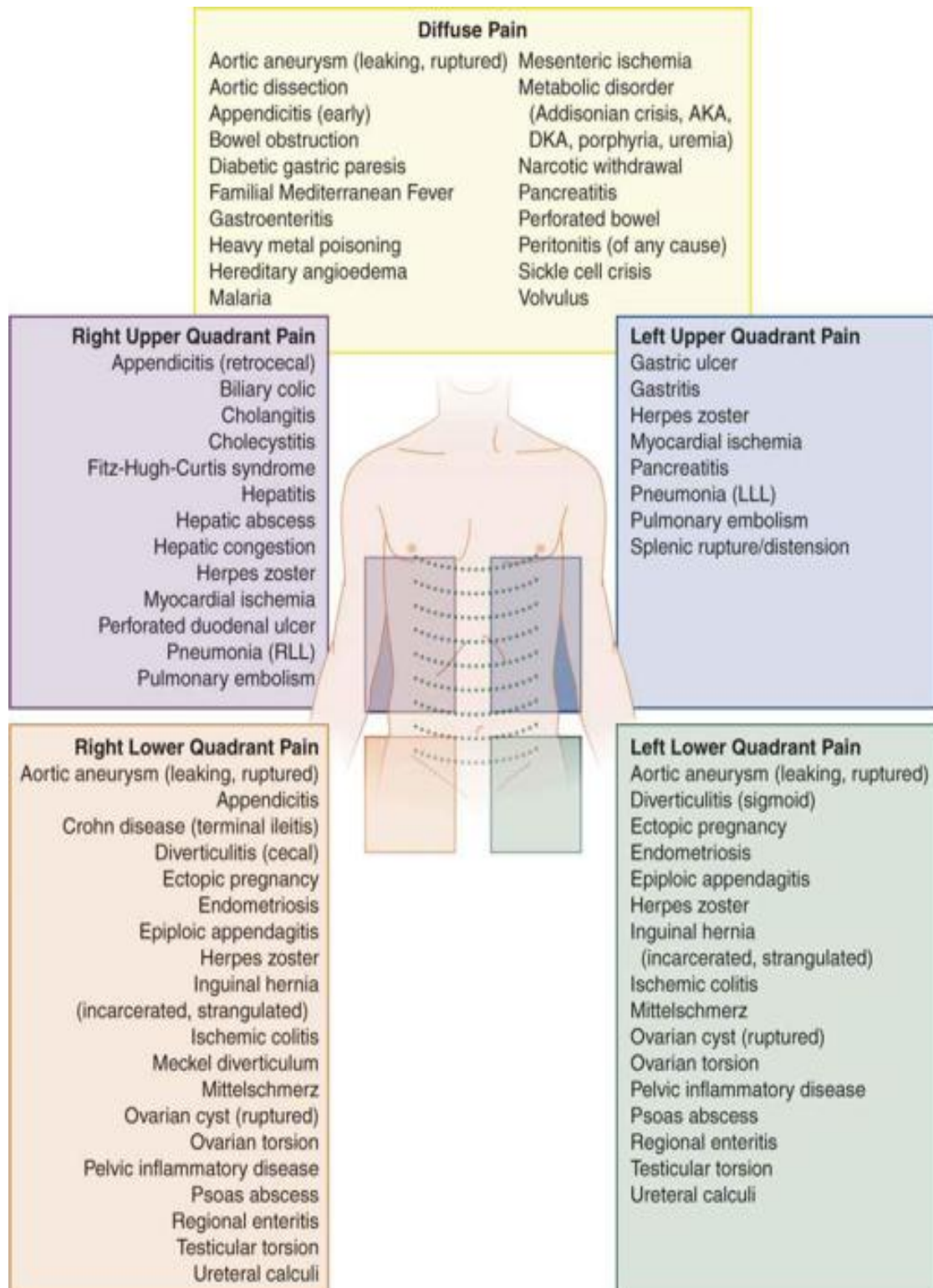
## **Acute Abdominal Pain**

**David M. Cline**

Acute abdominal pain may be due to numerous etiologies including gastrointestinal, genitourinary, cardiovascular, pulmonary, musculoskeletal, dermatologic, neurogenic, and other sources.

### **CLINICAL FEATURES**

Consider immediate life threats that might require emergency intervention. Elicit time of pain onset; character, severity, location of pain and its referral ([Fig. 35-1](#)); aggravating and alleviating factors; and similar prior episodes. Cardiorespiratory symptoms, such as chest pain, dyspnea, and cough; genitourinary symptoms, such as urgency, dysuria, and vaginal discharge; and any history of trauma should be elicited. In older patients it is also important to obtain a history of myocardial infarction, dysrhythmias, coagulopathies, and vasculopathies. Past medical and surgical histories should be elicited, and a list of medications, particularly steroids, antibiotics, or nonsteroidal anti-inflammatory drugs (NSAIDs), should be noted. A thorough gynecologic history is indicated in female patients.



**FIGURE 35-1.** Differential diagnosis of acute abdominal pain by location.  
 Key: AKA = alcoholic ketoacidosis; DKA = diabetic ketoacidosis; LLL = lower left lobe; RLL = right lower lobe.

The physical examination should include the patient's general appearance. Patients with peritonitis tend to lie still. The skin should be evaluated for pallor or jaundice. The vital signs should be inspected for signs of hypovolemia due to blood loss or volume depletion. Due to medications or the physiology of aging, tachycardia may not always occur in the face of hypovolemia. A core temperature should be obtained; however, absence of fever does not rule out infection, particularly in the elderly. The abdomen should be inspected for contour, scars, peristalsis, masses, distention, and pulsation. The presence of hyperactive or high-pitched or tinkling bowel sounds increases the likelihood of small bowel obstruction.

Palpation is the most important aspect of the physical examination. The abdomen and genitals should be assessed for tenderness, guarding, masses, organomegaly, and hernias. Rebound tenderness, often regarded as the clinical criterion standard of peritonitis, has several important limitations. In patients with peritonitis, the combination of rigidity, referred tenderness, and, especially, cough pain usually provides sufficient diagnostic confirmation; false-positive rebound tenderness occurs in about 1 patient in 4 without peritonitis. This has led some investigators to conclude that rebound tenderness, in contrast to cough pain, is of no predictive value. A useful and underused test to diagnose abdominal wall pain is the sit-up test, also known as the *Carnett sign*. After identification of the site of maximum abdominal tenderness, the patient is asked to fold his or her arms across the chest and sit up halfway. The examiner maintains a finger on the tender area, and if palpation in the semisitting position produces the same or increased tenderness, the test is said to be positive for an abdominal wall syndrome.

Perform a pelvic examination in all postpubertal females. During the rectal examination, the lower pelvis should be assessed for tenderness, bleeding, and masses.

Elderly patients often fail to manifest the same signs and symptoms as younger patients, with decreased pain perception and decreased febrile or muscular response to infection or inflammation. Biliary disease, bowel obstruction, diverticulitis, cancer, and hernia are more common causes of abdominal pain in patients over 50 years old. Hypotension from volume contraction, hemorrhage, or sepsis can be missed if a normally hypertensive patient appears normotensive. Conditions, somewhat less frequent but proportionately higher in occurrence, among the elderly include sigmoid volvulus, diverticulitis, acute mesenteric ischemia, and abdominal aortic aneurysm. Mesenteric ischemia should be considered in any patient older than 50 years with abdominal pain out of proportion to physical findings.

## **DIAGNOSIS AND DIFFERENTIAL**

Suggested laboratory studies for goal-directed clinical testing are listed in [Table 35-1](#). All women of child-bearing age with abdominal pain or abnormal vaginal bleeding should receive a qualitative screening pregnancy test. A complete blood count is neither sensitive nor specific to identify abdominal pathology, however, it remains the most commonly ordered test for ED patients with abdominal pain.

Laboratory Test	Clinical Suspicion
β-Human chorionic gonadotrophin	Pregnancy Ectopic or molar pregnancy
Coagulation studies (PT, PTT)	GI bleeding End-stage liver disease Coagulopathy
Electrocardiogram	Cardiac Ischemia
Electrolytes	Dehydration Endocrine or metabolic disorder
Glucose	Diabetic ketoacidosis Pancreatitis
Gonococcal/chlamydia testing	Cervicitis/urethritis Pelvic inflammatory disease
Hemoglobin	GI bleeding

Lactate	Mesenteric ischemia
Lipase	Pancreatitis
Liver function tests	Cholecystitis Cholelithiasis Hepatitis
Platelets	GI bleeding
Renal function tests	Acute renal failure Renal insufficiency Dehydration
Urinalysis	Urinary tract infection Pyelonephritis Nephrolithiasis

Key: PT = Prothrombin time, PTT = partial thromboplastin time.

(Reproduced with permission from Fitch M: Utility and limitations of laboratory studies, in Cline DM, Stead LG (eds): Abdominal Emergencies. New York, McGraw-Hill Medical, 2008, p. 19.)

**TABLE 35-1** Suggested Laboratory Studies for Goal-Directed Clinical Testing in

Plain abdominal radiographs are helpful in patients with suspected obstruction, perforation (look for free air), or to follow previously identified stones in renal colic patients. Ultrasonography is useful for the diagnosis of cholelithiasis, choledocholithiasis, cholecystitis, biliary duct dilatation, pancreatic masses, hydronephrosis, intrauterine or ectopic pregnancies, ovarian and tubal pathologies, free intraperitoneal fluid, suspected appendicitis (institution specific), and abdominal aortic aneurysm. Computed tomography (CT) is the preferred imaging method for mesenteric ischemia, pancreatitis, biliary obstruction, aortic aneurysm, appendicitis, and urolithiasis and is superior for identifying virtually any abnormality that can be seen on plain films. Intravenous contrast is essential to identify vascular lesions, is helpful to identify inflammatory conditions (ie, appendicitis), but is not needed for

urolithiasis. Oral contrast aids in the diagnosis of bowel obstruction, but otherwise is less useful.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Unstable patients should be resuscitated immediately, then diagnosed clinically with emergent surgical consultation.

1. The most common resuscitation need for abdominal pain patients is intravenous hydration with normal saline or lactated Ringer's solution. During the initial evaluation, the patient should have nothing by mouth.
2. The judicious use of analgesics is appropriate and may facilitate the ability to obtain a better history and more accurate physical examination. Consider **morphine** 0.1 milligram/kilogram IV, which can be reversed by **naloxone** (0.4 to 2 milligrams SC/IV) if necessary. NSAIDs are useful in patients with renal colic, but their use in other conditions is controversial and they can mask peritoneal inflammation.
3. Antiemetics, such as **ondansetron** 4 milligrams IM/IV, or **metoclopramide** 10 milligrams IM or slow IV, also increase the patient's comfort and facilitate assessment of the patient's signs and symptoms.
4. When appropriate, antibiotic treatment (ie, **gentamicin** 1.5 milligrams/ kilogram IV plus **metronidazole** 1 gram IV; or **piperacillin-tazobactam**, 3.375 grams IV) should be initiated, depending on the suspected source of infection. See specific chapters that follow in this section for additional guidelines.
5. Surgical or obstetric and gynecologic consultation should be obtained for patients with suspected acute abdominal or pelvic pathology requiring immediate intervention, including, but not limited to, abdominal aortic aneurysm, intraabdominal hemorrhage, perforated viscus, intestinal obstruction or infarction, and ectopic pregnancy. Historically, the "acute abdomen" or "surgical abdomen" has been identified by the presence of pain, guarding, and rebound as indicating a likely need for emergent surgery.
6. Indications for admission include toxic appearance, unclear diagnosis in elderly or immunocompromised patients, inability to reasonably exclude serious etiologies, intractable pain or vomiting, altered mental status, and inability to follow discharge or follow-up instructions. Continued observation with serial examinations is an alternative. Many patients with nonspecific abdominal pain can be discharged safely with 24 hours of follow-up and instructions to return immediately for increased pain, vomiting, fever, or failure of symptoms to resolve.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 74, “Acute Abdominal Pain,” by Mary Claire O’Brien.



# **CHAPTER 36**

## **Nausea and Vomiting**

Jonathan A. Maisel

Although nausea and vomiting are typically caused by gastrointestinal disorders, the clinician must consider systemic causes as well. Neurologic, infectious, cardiac, endocrine, renal, obstetric, pharmacologic, toxicologic, and psychiatric disorders may all be the cause of nausea and vomiting. A comprehensive history and physical examination, as well as the use of various diagnostic modalities, are needed to determine the cause and its complications.

### **CLINICAL FEATURES**

History is essential in determining the cause of vomiting. Important features to elicit include the onset and duration of symptoms, the frequency and timing of episodes, the content of the vomitus (eg, undigested food, bile-tinged, feculent), associated symptoms (eg, fever, abdominal pain, diarrhea), exposure to foodborne pathogens, and the presence of sick contacts. A thorough past medical and surgical history (eg, prior abdominal surgery) will also be valuable. The physical examination should initially focus on determining the presence or absence of a critical, life-threatening condition. Hypotension, tachycardia, lethargy, poor skin turgor, dry mucous membranes, and delayed capillary refill suggest significant dehydration. A careful abdominal examination will help clarify the presence or absence of a primary GI etiology. The extent to which the balance of the physical examination will be of value will be dictated by the history. In the event that a reliable history is not available (eg, drug overdose, cognitive impairment), a comprehensive physical examination is warranted.

### **DIAGNOSIS AND DIFFERENTIAL**

Vomiting with blood could represent gastritis, peptic ulcer disease, or carcinoma. However, aggressive nonbloody vomiting followed by hematemesis is more consistent with a Mallory-Weiss tear. The presence of bile rules out gastric outlet obstruction, such as from pyloric stenosis or strictures. The presence of abdominal distension, surgical scars, or an incarcerated hernia suggests a small bowel obstruction. The presence of fever would suggest an infectious (eg, gastroenteritis, appendicitis, cholecystitis) or inflammatory cause. Vomiting with chest pain suggests myocardial infarction. Posttussive vomiting suggests pneumonia. Vomiting with back or flank pain can be seen with aortic aneurysm or dissection, pancreatitis, pyelonephritis or renal colic. Headache with vomiting suggests increased intracranial pressure, such as with subarachnoid hemorrhage, tumor, or head injury. The presence of vertigo and nystagmus suggests either vestibular or CNS pathology. Vomiting in a pregnant patient is consistent with hyperemesis gravidarum in the first trimester; but in the third trimester, could represent preeclampsia if accompanied by hypertension. Associated medical conditions are also useful in discerning the cause of vomiting: diabetes mellitus suggests ketoacidosis, peripheral vascular disease suggests mesenteric ischemia, and medication use or overdose (eg, lithium or digoxin) suggests toxicity.

All women of childbearing age warrant a pregnancy test. In vomiting associated with abdominal pain, liver function tests, urinalysis, and lipase or amylase determinations may be useful. Electrolyte determinations and renal function tests are usually of benefit only in patients with severe dehydration or prolonged vomiting. In addition, they may confirm the presence of Addisonian crisis, with hyperkalemia and hyponatremia. Obtain specific drug levels for acetaminophen, salicylates, and digoxin when toxicity is suspected, and urine and/or serum toxicology screens when ethanol or illicit drug use is suspected. The electrocardiogram and chest radiograph can be reserved for patients with suspected cardiac ischemia or pulmonary infection. Abdominal x-rays can be used to confirm the presence of intestinal obstruction. If plain x-rays are unrevealing, CT scan of the abdomen and pelvis with IV and PO contrast is not only helpful for revealing the location of a mechanical obstruction, but may also clarify alternative explanations for the patient's symptoms. CT scan of the brain

will be helpful if a CNS lesion is suspected. Measuring intraocular pressure with a Tono-Pen<sup>®</sup> (Reichert, Inc., Depew, NY) is useful if glaucoma is suspected.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The treatment of nausea and vomiting consists of correcting fluid and electrolyte problems. In addition, one must initiate specific therapy for any life-threatening cause identified in the initial workup.

1. Resuscitation of seriously ill patients requires intravenous boluses of normal saline 20 mL/kilogram. Boluses may be repeated as necessary, targeting euvolemia. Caution should be used in the elderly, and those with compromised left ventricular function. Mildly dehydrated patients may tolerate an oral rehydration solution containing sodium as well as glucose to enhance fluid absorption. Many commercial products (eg, **Pedialyte**<sup>®</sup>) are available. The World Health Organization advocates a mixture of 4 oz orange juice, 8 tsp sugar, and 1 tsp salt in 1 L boiled water.
2. Nutritional supplementation should be started as soon as nausea and vomiting subside. Patients can quickly advance from clear liquids to solids, such as rice and bread. Patients may benefit from avoiding raw fruit, caffeine, and lactose and sorbitol-containing products.
3. Antiemetic agents are useful in actively vomiting patients with dehydration. **Ondansetron** 4 to 8 milligrams IV or ODT (children 0.15 milligram/ kilogram) is very effective and well tolerated, and can be administered to pregnant women (category B). **Promethazine** 25 milligrams (0.25 to 1 milligram/kilogram in children over 2 years) IM or PR every 4 to 6 hours can be effective. **Prochlorperazine** 5 to 10 milligrams IM every 4 hours, or 25 milligrams PR every 12 hours is effective. **Metoclopramide** 10 milligrams (children 0.1 milligram/kilogram) IV/IM every 6 to 8 hours is useful and can be administered to pregnant women (category B). **Meclizine** 25 milligrams PO every 6 hours is effective for vomiting associated with vertigo.

Patients with a life-threatening cause of vomiting require admission. In addition, toxic or severely dehydrated patients, particularly infants and the elderly, or those still intolerant of oral fluids after hydration, warrant admission. Patients with an unclear diagnosis, but favorable examination findings after hydration, can be discharged home safely with antiemetics. Work excuses are indicated for patients in the food, day care, and health care industries.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 75, "Nausea and Vomiting," by Susan Bork, Jeffrey Ditkoff, and Bophal Sarha Hang.

## CHAPTER 37

# Disorders Presenting Primarily With Diarrhea

Jonathan A. Maisel

*Diarrhea* is defined as three or more watery stools per day. There are four basic mechanisms: increased intestinal secretion (eg, cholera), decreased intestinal absorption (eg, enterotoxins, inflammation, or ischemia), increased osmotic load (eg, laxatives, lactose intolerance), and abnormal intestinal motility (eg, irritable bowel syndrome). Approximately 85% of cases are infectious in etiology.

## DIARRHEA

### Clinical Features

Determine if the diarrhea is acute (<3 weeks duration) or chronic (> 3 weeks). Acute diarrhea is more likely to represent a serious problem, such as infection, ischemia, intoxication, or inflammation. Inquire about associated symptoms. Features such as fever, pain, presence of blood, or type of food ingested, may help in the diagnosis of infectious gastroenteritis, food poisoning, diverticulitis, or inflammatory bowel disease. Neurological symptoms can be seen in certain diarrheal illnesses, such as seizure with shigellosis or hyponatremia, or paresthesias and reverse temperature sensation with ciguatoxin.

Details about the host can also better define the diagnosis. Malabsorption from pancreatic insufficiency or HIV-related bowel disorders need not be considered in a healthy host. Dietary practices, including frequent restaurant meals, exposure to day care centers, consumption of street vendor food or raw seafood, overseas travel, and camping with the ingestion of lake or stream water, may isolate the vector and narrow the differential diagnosis for infectious diarrhea (eg, lakes or streams—*Giardia*, oysters suggest *Vibrio*; rice suggests *Bacillus cereus*; eggs suggest *Salmonella*; and meat

suggests *Campylobacter*, *Staphylococcus*, *Yersinia*, *Escherichia coli*, or *Clostridium*). Certain medications, particularly antibiotics, colchicine, lithium, and laxatives, can all contribute to diarrhea. Travel may predispose the patient to enterotoxigenic *E coli* or *Giardia*. Social history, such as sexual preference, drug use, and occupation, may suggest diagnoses such as HIV-related illness or organophosphate poisoning.

The physical examination begins with assessment of hydration status. Abdominal examination can narrow the differential diagnosis as well as reveal the need for surgical intervention. Even appendicitis can present with diarrhea in up to 20% of cases. Rectal examination can rule out impaction or presence of blood, the latter suggesting inflammation, infection, or mesenteric ischemia.

## **Diagnosis and Differential**

The most specific tests in diarrheal illness all involve examination of the stool in the laboratory. Stool culture testing should be limited to severely dehydrated or toxic patients, those with blood or pus in their stool, immunocompromised patients, and those with diarrhea lasting longer than 3 days. Consider testing for *Salmonella*, *Shigella*, *Campylobacter*, Shiga toxin-producing *E coli*, or amoebic infection. Make the laboratory aware of which pathogens you suspect. In patients with diarrhea > 7 days, those who have traveled abroad, or consumed untreated water, an examination for ova and parasites may be useful to rule out *Giardia* or *Cryptosporidium*. Multiple samples may be required. Assay for *Clostridium difficile* toxin may be useful in ill patients with antibiotic-associated diarrhea or recent hospitalization.

Because most diarrheal illnesses are viral or self-limited, laboratory testing in routine cases is not indicated. However, in extremely dehydrated or toxic patients, electrolyte determinations and renal function tests may be useful. (Hemolytic-uremic syndrome, characterized by acute renal failure, thrombocytopenia, and hemolytic anemia, may complicate *E coli* 0157:H7 infections in children and the elderly). If toxicity is suspected, tests for levels for theophylline, lithium or heavy metals will aid in the diagnosis. Radiographs are reserved for ruling out intestinal obstruction or pneumonia,

particularly *Legionella*. In addition, CT scanning or angiography may be indicated in acute mesenteric ischemia.

## **Emergency Department Care and Disposition**

The treatment of diarrhea consists of correcting fluid and electrolyte problems. In addition, one must initiate specific therapy for any life-threatening cause identified in the initial workup.

1. Replacement of fluids can be intravenous (boluses of 500 mL IV in adults, 20 mL/kilogram in children) with normal saline solution in seriously ill patients. Mildly dehydrated patients who are not vomiting may tolerate an oral rehydrating solution containing sodium (eg, Pedialyte<sup>®</sup>) as well as glucose to enhance fluid absorption (glucose transport unaffected by enterotoxins). The World Health Organization advocates a mixture of 4 oz orange juice, 8 tsp sugar, and 1 tsp salt in 1 L boiled water. The goal is 50 to 100 mL/kilogram over the first 4 hours.
2. As patients tolerate, introduce a “BRAT” diet (bananas, rice, apples, toast). Patients should avoid raw fruit, caffeine, and lactose and sorbitol-containing products.
3. Antibiotics are recommended for adult patients with severe or prolonged diarrhea. (See section on acute infectious and traveler’s diarrhea). Antibiotics should be avoided in infectious diarrhea due to *E coli* O157:H7.
4. Antidiarrheal agents, especially in combination with antibiotics, have been shown to shorten the course of diarrhea. (See section on acute infectious and traveler’s diarrhea.)

Antibiotic-associated diarrhea often responds to withdrawal of the offending drug. Metronidazole or vancomycin may be indicated in selected situations (see section on *Clostridium difficile* infection). Almost all true diarrheal emergencies (eg, gastrointestinal (GI) bleed, adrenal insufficiency, thyroid storm, toxicologic exposures, acute radiation syndrome, and mesenteric ischemia) are of noninfectious origin. Patients with these conditions require intensive treatment and hospitalization. In addition, toxic

or severely dehydrated patients, particularly infants and the elderly warrant admission. Patients with an unclear diagnosis, but favorable examination findings after hydration, can be discharged home safely.

## **ACUTE INFECTIOUS AND TRAVELER'S DIARRHEA**

*Norovirus* causes 50% to 80% of all infectious diarrheas in the United States, followed much less frequently by non-Shiga toxin-producing *E coli*, *C difficile*, invasive bacteria (*Campylobacter*, *Shigella*, *Salmonella*), Shiga toxin-producing *E coli*, and protozoa. A history of foreign travel, with consumption of contaminated food or drink, is associated with an 80% probability of bacterial diarrhea, primarily toxin and nontoxin-producing strains of *E coli*.

### **Diagnosis and Differential**

Patients with severe abdominal pain, fever, and bloody stool should undergo stool studies for specific pathogens, including culture for *Salmonella*, *Shigella*, *Campylobacter*, and *E coli* O157:H7; assay for Shiga toxin; and microscopy or antigen assay for *Entamoeba histolytica*. Exposure of a traveler or hiker to untreated water, and illness that persists for more than 7 days, should prompt an evaluation for a protozoal pathogen. Stool should be tested by enzyme immunoassay for *E histolytica* antigen, *Giardia intestinalis* antigen, and *Cryptosporidium parvum* antigen.

### **Emergency Department Care and Disposition**

Treatment of moderately severe infectious diarrhea (including viral causes) includes antibiotics, antimotility agents, fluid resuscitation (oral or parenteral), and dietary modification.



1. **Ciprofloxacin** 500 milligrams as a single dose, or 500 milligrams twice daily for 3 days will shorten the duration of illness by approximately 24 hours. (Similar dosing for culture proven *Shigella* or enterotoxigenic, enteropathogenic, or enteroinvasive *E coli*. However, both antibiotics and antimotility agents should be avoided in cases of Shiga toxin-producing *E coli* O157:H7). **Trimethoprim/sulfamethoxazole**, TMP 5 milligrams/kilogram/dose:SMX 25 milligrams/kilogram/dose (maximum dose TMP 160 milligrams:SMX 800 milligrams) twice daily for 3 days is indicated for children or nursing mothers.
2. **Metronidazole** 750 milligrams PO three times daily for 5 to 10 days is indicated for *Giardia* or *Entamoeba* infection. Add **iodoquinol** 650 milligrams three times daily for 20 days, or **paromomycin** 500 milligrams three times daily for 5 to 10 days, for the latter.
3. Antimotility agents, such as **loperamide** 4 milligrams initially, then 2 milligrams following each unformed stool (16 milligrams/d maximum), will shorten the duration of symptoms when combined with an antibiotic. Alternative agents include **bismuth subsalicylate** 30 mL or 2 tablets every 30 min for 8 doses, or **diphenoxylate and atropine** 4 milligrams four times daily. Avoid antimotility agents in the subset of patients with bloody or suspected inflammatory diarrhea because of the potential for prolonged fever, toxic megacolon in *C difficile* patients, and hemolytic uremic syndrome in children infected with Shiga-toxin producing *E coli*.

Most patients can be discharged home. Educate patients regarding the need for frequent hand washing to minimize transmission. Provide work excuses to patients employed in the food, day care, and health care industries. Any toxic-appearing patient should be admitted. Consider admission for those at extremes of age as well.

Individuals should be counseled about the proper selection of food and beverages consumed when traveling abroad, as well as the use of water for drinking, brushing teeth, and the preparation of food and infant formula.

## ***CLOSTRIDIUM DIFFICILE*-ASSOCIATED INFECTION, DIARRHEA, AND COLITIS**

*Clostridium difficile* is an anaerobic bacillus which secretes two toxins that interact in a complex manner to cause illness ranging from diarrhea to pseudomembranous colitis. Pseudomembranous colitis is an inflammatory bowel disorder in which membrane-like yellowish plaques of exudate overlay and replace necrotic intestinal mucosa. Broad-spectrum antibiotics, most notably clindamycin, cephalosporins, ampicillin, amoxicillin, and fluoroquinolones, alter gut flora in such a way that *C difficile* can flourish within the colon, causing enteropathy. Transmission of the organism can occur from contact with humans and fomites. *C difficile* is the most common cause of infectious diarrhea in hospitalized patients and is now reported to affect healthy adults who were not exposed to a hospital setting.

## **Clinical Features**

Onset is typically 7 to 10 days after initiating antibiotic treatment, but may occur up to several weeks following treatment. Clinical manifestations can vary from frequent, watery, mucoid stools to a toxic picture, including profuse diarrhea, crampy abdominal pain, fever, leukocytosis, and dehydration.

## **Diagnosis and Differential**

The diagnosis is confirmed by the demonstration of *C difficile* toxin in stool. Colonoscopy is not routinely needed to confirm the diagnosis.

## **Emergency Department Care and Disposition**

1. Mild *C difficile* infection in an otherwise healthy patient can be treated with discontinuing the offending antibiotic, confirmation of infection, and clinical monitoring.
2. Oral **metronidazole** 500 milligrams orally every 6 hours for 10 to 14 days is the treatment of choice in patients with mild to moderate disease who do not respond to conservative measures.

3. Patients with severe diarrhea, those with a systemic response (eg, fever, leukocytosis, or severe abdominal pain), and those whose symptoms persist despite appropriate outpatient management, must be hospitalized and should receive **vancomycin** 125 to 250 milligrams orally 4 times daily for 10 to 14 days. The symptoms usually resolve within a few days.
4. Patients with pseudomembranous colitis complicated by toxic megacolon or intestinal perforation require immediate surgical consultation. Rarely, emergency colectomy may be required for fulminant colitis.

Use of antidiarrheal agents is controversial. Relapses occur in 10% to 25% of patients.

## **CROHN DISEASE**

Crohn disease is a chronic, idiopathic, granulomatous inflammatory disease, characterized by segmental ulceration of the GI tract anywhere from the mouth to the anus.

### **Clinical Features**

The clinical course is variable and unpredictable, with multiple remissions and exacerbations. Patients commonly report a history of recurring fever, abdominal pain, and diarrhea over several years before a definitive diagnosis is made. Abdominal pain, anorexia, diarrhea, and weight loss occur in most patients. Patients may also present with complications of the disease, such as intestinal obstruction, intraabdominal abscess, or a variety of extraintestinal manifestations. One-third of patients develop perianal fissures, fistulas, abscesses, or rectal prolapse. Fistulas occur between the ileum and sigmoid colon; the cecum, another ileal segment, or the skin; or between the colon and the vagina. Abscesses can be intraperitoneal, retroperitoneal, interloop, or intramesenteric. Obstruction, hemorrhage, and toxic megacolon also occur. Toxic megacolon can be associated with massive GI bleeding.

Up to 50% of patients develop extraintestinal manifestations, including arthritis, uveitis, nephrolithiasis, and skin disease (eg, erythema nodosum, pyoderma gangrenosum). Hepatobiliary disease, including gallstones, pericholangitis, and chronic active hepatitis is commonly seen, as is pancreatitis. Some patients develop thromboembolic disease as a result of a hypercoagulable state. Malabsorption, malnutrition, and chronic anemia develop in longstanding disease, and the incidence of GI tract carcinoma is triple that of the general population. The recurrence rate for those with Crohn disease is 25% to 50% when treated medically; higher for patients treated surgically.

## **Diagnosis and Differential**

The definitive diagnosis of Crohn disease is usually established months or years after the onset of symptoms. A careful and detailed history for previous bowel symptoms that preceded the acute presentation may provide clues to the correct diagnosis. Abdominal CT scanning is the most useful diagnostic test, potentially revealing bowel wall thickening, mesenteric edema, abscess formation, and fistulas, as well as extraintestinal complications (eg, gallstones, renal stones, sacroiliitis). Colonoscopy can detect early mucosal lesions, define the extent of colonic involvement, and identify colon cancer.

The differential diagnosis of Crohn disease includes lymphoma, ileocecal amebiasis, sarcoidosis, chronic mycotic infections, tuberculosis, Kaposi sarcoma, *Campylobacter* enteritis, and *Yersinia* ileocolitis. Most of these conditions are uncommon, and the latter two can be differentiated by stool cultures. When confined to the colon, ischemic colitis, infectious colitis, pseudomembranous enterocolitis, irritable bowel syndrome, and ulcerative colitis should be considered.

## **Emergency Department Care and Disposition**

Initial evaluation should determine the severity of the attack and identify significant complications. Laboratory evaluation includes complete blood

count, chemistries, and type and cross match when indicated. Plain abdominal x-rays may identify obstruction, perforation, and toxic megacolon, which may appear as a long, continuous segment of air filled colon greater than 6 cm in diameter. CT of the abdomen is the most useful test to confirm the diagnosis, and identify both intrainestinal and extraintestinal manifestations. Initial ED management includes intravenous fluid replacement, parenteral analgesia, bowel rest, correction of electrolyte abnormalities, and nasogastric suction if obstruction, ileus or toxic megacolon, is present. Additional treatment may include:

1. **Sulfasalazine** 3 to 5 grams/day is effective for mild to moderate Crohn disease, but has multiple toxic side effects, including GI and hypersensitivity reactions. **Mesalamine**, up to 4 grams/day, is equally effective, with fewer side effects.
2. Glucocorticoids (**prednisone**) 40 to 60 milligrams/day provide symptom relief, but does not alter the course of the disease.
3. Immunosuppressive drugs, **6-mercaptopurine** 1 to 1.5 milligrams/kilogram/day or **azathioprine** 2 to 2.5 milligrams/kilogram/day, are used as steroid-sparing agents, in healing fistulas, and in patients with serious surgical contraindications.
4. Antibiotics can help induce remission. **Ciprofloxacin** 500 milligrams every 8 to 12 hours, **metronidazole** 500 milligrams every 6 hours, and **rifaximin** 800 milligrams twice daily, are effective.
5. Patients with medically resistant, moderate to severe Crohn disease may benefit from the antitumor necrosis factor antibody **infliximab** 5 milligrams/kilogram intravenously.
6. Diarrhea can be controlled by **loperamide** 4 to 16 milligrams/day, **diphenoxylate** 5 to 20 milligrams/day, or **cholestyramine** 4 grams 1 to 6 times daily.

Hospital admission is recommended for those who demonstrate signs of fulminant colitis, peritonitis, obstruction, significant hemorrhage, severe dehydration, or electrolyte imbalance, or those with less severe disease who fail outpatient management. Surgical intervention is indicated in patients with intestinal obstruction or hemorrhage, perforation, abscess or fistula formation, toxic megacolon, or perianal disease, and in some patients who

fail medical therapy. Alterations in therapy should be discussed with a gastroenterologist and close follow-up must be ensured for patients discharged from the ED.

## **ULCERATIVE COLITIS**

Ulcerative colitis is an idiopathic chronic inflammatory and ulcerative disease of the colon and rectum, characterized clinically by intermittent episodes of crampy abdominal pain and bloody diarrhea, with complete remission between bouts.

### **Clinical Features**

Patients with mild disease (> 50%), typically limited to the rectum, have fewer than four bowel movements per day, no systemic symptoms, and few extraintestinal manifestations. Patients with moderate disease (25%) have colitis extending to the splenic flexure. Severe disease (pancolitis) is with frequent daily bowel movements, weight loss, fever, tachycardia, anemia, and more frequent extraintestinal manifestations, including peripheral arthritis, ankylosing spondylitis, episcleritis, uveitis, pyoderma gangrenosum, erythema nodosum, hepatobiliary disease, thromboembolic disease, renal stones, and malnutrition.

Complications include GI hemorrhage (most common), abscess and fistula formation, obstruction secondary to stricture formation, and acute perforation. There is a 10- to 30-fold increase in the risk of developing colon carcinoma. The most feared complication is toxic megacolon, which presents with fever, tachycardia, dehydration, and a tender, distended abdomen. X-ray reveals a long, continuous segment of air-filled colon > 6 cm in diameter. Perforation and peritonitis are life-threatening complications.

### **Diagnosis and Differential**

The diagnosis of ulcerative colitis may be considered with a history of abdominal cramps, diarrhea, and mucoid stools. Laboratory findings are nonspecific, and may include leukocytosis, anemia, thrombocytosis, decreased serum albumin levels, abnormal liver function test results, and negative stool studies for ova, parasites, and enteric pathogens. Abdominal CT scanning is important for the diagnosis of nonspecific abdominal pain or for suspected colitis. Colonoscopy can confirm the diagnosis and define the extent of colonic involvement. The differential diagnosis includes infectious, ischemic, radiation, antineoplastic agent induced, pseudomembranous, and Crohn colitis. When the disease is limited to the rectum, consider sexually acquired diseases, such as rectal syphilis, gonococcal proctitis, lymphogranuloma venereum, and inflammation caused by herpes simplex virus, *Entamoeba histolytica*, *Shigella*, and *Campylobacter*.

## **Emergency Department Care and Disposition**

Patients with severe disease should be admitted for intravenous fluid replacement, parenteral analgesia, bowel rest, correction of electrolyte abnormalities, and nasogastric suction if obstruction, ileus or toxic megacolon present. Consultation with both gastroenterology and surgery should be arranged for patients with significant GI hemorrhage, toxic megacolon, and bowel perforation. In addition, the following interventions should be considered:

1. Intravenous antibiotics, such as **ciprofloxacin** (400 milligrams every 12h to q8h) and **metronidazole** (500 milligrams every 6h).
2. Parenteral steroid treatment with either **hydrocortisone** 100 milligrams every 8h, **methylprednisolone** 16 milligrams every 8h, or **prednisolone** 30 milligrams every 12h.

The majority of patients with mild and moderate disease can be treated as outpatients. Therapy listed below should be discussed with a gastroenterologist, and close follow-up must be ensured.

1. For mild active proctitis and left sided colitis, **mesalamine** suppositories or enemas are effective. However, topical steroid preparations (**beclomethasone**, **hydrocortisone**) may be better tolerated.
2. For patients who do not respond to or tolerate topical therapy, oral **mesalamine** is an effective alternative.
3. If topical therapy or oral mesalamine is unsuccessful, **Prednisone** 40 to 60 milligrams/day PO can induce a remission. Once clinical remission is achieved, steroids should be slowly tapered and discontinued.
4. In refractory cases, a combination of glucocorticoids and immunomodulators, such as **6-mercaptopurine** 1 to 1.5 milligrams/kilogram/day or **azathioprine** 2 milligrams/kilogram/day should be considered.

Supportive measures include a nutritious diet, physical and psychological rest, replenishment of iron stores, dietary elimination of lactose, and addition of bulking agents, such as psyllium. Antidiarrheal agents can precipitate toxic megacolon and should be avoided.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 76, "Disorders Presenting Primarily with Diarrhea," by Nicholas E. Kman and Howard A. Werman.



## **CHAPTER 38**

# **Acute and Chronic Constipation**

Jonathan A. Maisel

Constipation is the most common digestive complaint in the United States. Gut motility is affected by diet, activity level, anatomic lesions, neurologic conditions, medications, toxins, hormones, rheumatologic conditions, infection, and psychiatric conditions.

## **CLINICAL FEATURES**

Constipation is demonstrated by the presence of hard stools that are difficult to pass. Several historical features may be helpful in eliciting the cause, including new medications or dietary supplements, a decrease in fluid or fiber intake, or a change in activity level. Acute onset implies obstruction until proven otherwise. Associated symptoms, such as vomiting, abdominal distention, and inability to pass flatus further suggest obstruction. A history of unexplained weight loss, rectal bleeding, or unexplained iron-deficiency anemia suggests colon cancer. A family history of colon cancer would escalate one's suspicion. Associated illnesses can help disclose the underlying diagnosis: cold intolerance (hypothyroidism), diverticulitis (inflammatory stricture), or nephrolithiasis (hyperparathyroidism). Diarrhea alone does not rule out constipation/obstruction, as liquid stool can pass around an obstructive source.

Physical examination should focus on detection of hernias or abdominal masses. Bowel sounds will be decreased in the setting of slow gut transit, but increased in the setting of obstruction. Rectal examination will detect masses, foreign bodies, hemorrhoids, abscesses, fecal impaction, anal fissures, or fecal blood. The latter, accompanied by weight loss or decreasing stool caliber, may confirm the presence of colon cancer. Fecal impaction itself can cause rectal bleeding from stercoral ulcers. The

presence of ascites in postmenopausal women raises suspicion of ovarian or uterine carcinoma.

## DIAGNOSIS AND DIFFERENTIAL

The differential diagnosis for constipation is extensive, as noted in [Table 38-1](#). Directed testing in acute constipation, based on level of suspicion, can include a complete blood count (to rule out anemia), thyroid panel (to rule out hypothyroidism), and electrolyte determinations (to rule out hypokalemia or hypercalcemia). Flat and erect abdominal films may be useful in confirming obstruction or assessing stool burden. CT scan of the abdomen and pelvis with IV and PO contrast may be necessary to identify bowel obstruction or other organic causes of constipation.

<b>Acute Causes:</b>
GI: quickly growing tumors, strictures, hernias, adhesions, inflammatory conditions, and volvulus
Medicinal: narcotic analgesic, antipsychotic, anticholinergic, antacid, antihistamine
Exercise and nutrition: decrease in level of exercise, fiber intake, fluid intake
Painful anal pathology: anal fissure, hemorrhoids, anorectal abscesses, proctitis
<b>Chronic Causes:</b>
GI: slowly growing tumor, colonic dysmotility, chronic anal pathology
Medicinal: chronic laxative abuse, narcotic analgesic, antipsychotic, anticholinergic, antacid, antihistamine
Neurologic: neuropathies, Parkinson disease, cerebral palsy, paraplegia
Endocrine: hypothyroidism, hyperparathyroidism, diabetes
Electrolyte abnormalities: hypomagnesia, hypercalcemia, hypokalemia
Rheumatologic: amyloidosis, scleroderma
Toxicologic: lead, iron

**TABLE 38-1** Differential Diagnosis of Constipation

Chronic constipation is usually a functional disorder that can be worked up on an outpatient basis. However, complications of chronic constipation, such as fecal impaction and intestinal pseudoobstruction, will require either manual, colonoscopic, or surgical intervention.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

Treatment of functional constipation is directed at symptomatic relief, as well as addressing lifestyle issues. Occasionally, specific treatment is required for complications of constipation, or for underlying disorders that can lead to organic constipation.

1. The most important prescription for functional constipation is a dietary and exercise regimen that includes fluids (1.5 L/d), fiber (10 grams/d), and exercise. Fiber in the form of bran (1 cup/d) or **psyllium** (**Metamucil** 1 tsp 3 times a day) increases stool volume and gut motility.
2. Medications can provide temporary relief. Stimulants can be either given PO, as with **anthraquinones** (eg, **Peri-Colace** 1 to 2 tablets PO daily or twice daily) or PR, as with **bisacodyl** (eg, **Dulcolax** 10 milligrams PR tid in adults or children). In the absence of renal failure, saline laxatives such as **milk of magnesia** 15 to 30 mL PO once or twice a day or **magnesium citrate** 240 mL PO once, are useful. Hyperosmolar agents, such as **lactulose** or **sorbitol** 15 to 30 mL PO once or twice a day may be helpful, as is **polyethylene glycol** (eg, **MiraLAX** 17 grams PO). In children, **glycerine rectal suppositories**, or **mineral oil** (age 5 to 11 years: 5 to 15 mL PO daily; age > 12 years: 15 to 45 mL PO daily) have been advocated.
3. **Enemas of soapsuds** (1500 mL PR) or **phosphate** (eg, **Fleets** 1 unit PR, 1 oz/10 kilograms in children) are generally reserved for severe cases or after fecal disimpaction. Use care to avoid rectal perforation.
4. Fecal impaction should be removed manually using local anesthetic lubricant and parenteral analgesia or sedation as required. In female patients, transvaginal pressure with the other hand may be helpful. An enema or suppositories to complete evacuation can follow. Following disimpaction, a regimen of medication should be prescribed to reestablish fecal flow.

All patients with apparent functional constipation can be managed as outpatients. Early follow-up is indicated in patients with recent severe constipation; chronic constipation associated with systemic symptoms, such as weight loss, anemia, or change in stool caliber; refractory constipation, and constipation requiring chronic laxative use. Patients with organic constipation secondary to obstruction require hospitalization and surgical evaluation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 77, “Acute and Chronic Constipation,” by Vito Rocco and Paul Krivickas.

# CHAPTER 39

## Gastrointestinal Bleeding

Mitchell C. Sokolosky

Gastrointestinal (GI) bleeding is a common problem in emergency medicine and should be considered life threatening until proven otherwise. Acute upper GI bleeding is more common than lower GI bleeding. *Upper GI bleeding* is defined as that originating proximal to the ligament of Treitz. Upper GI bleeds can result from peptic ulcer disease, erosive gastritis and esophagitis, esophageal and gastric varices, and Mallory-Weiss syndrome. Lower GI bleeds result from diverticular disease, followed by colitis, adenomatous polyps, and malignancies. Less common causes include vascular ectasia (AV malformation and angiodysplasia), Meckel diverticulum, inflammatory bowel disease, and trauma. What may initially appear to be lower GI bleeding may be upper GI bleeding in disguise.

### CLINICAL FEATURES

Most patients complain of hematemesis, hematochezia, or melena. Others will present with hypotension, tachycardia, angina, syncope, weakness, and confusion. Hematemesis or coffee-ground emesis suggests an upper GI source. Melena suggests a source proximal to the right colon. Hematochezia indicates a more distal colorectal lesion; however, approximately 10% of hematochezia may be associated with UGI bleeding. Weight loss and changes in bowel habits are classic symptoms of malignancy. Vomiting and retching, followed by hematemesis, is suggestive of a Mallory-Weiss tear. A history of medication or alcohol use should be sought. This history may suggest peptic ulcer disease, gastritis, or esophageal varices. Spider angiomas, palmar erythema, jaundice, and gynecomastia suggest underlying liver disease. Ingestion of iron or bismuth can simulate melena, and certain foods, such as beets, can simulate hematochezia; however, stool heme (guaiac) testing will be negative.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis may be obvious with the finding of hematemesis, hematochezia, or melena. A careful ear, nose, and throat (ENT) examination can exclude swallowed blood as a source. Nasogastric (NG) tube placement and aspiration may detect occult upper GI bleeding. A negative NG aspirate does not conclusively exclude an upper GI source. Guaiac testing of NG aspirate can yield both false-negative and false-positive results. Most reliable is gross inspection of the aspirate for a bloody, maroon, or coffee-ground appearance, reserving guaiac testing to confirm that what appears to be blood actually is. A rectal examination is mandatory to detect the presence of blood, its appearance (bright red, maroon, or melanotic), and the presence of masses. All patients with significant GI bleeding require type and crossmatch for blood. Other important tests include a complete blood count, electrolytes, blood urea nitrogen, creatinine, glucose, coagulation studies, and liver function tests. The initial hematocrit level may not reflect the actual amount of blood loss. Upper GI bleeding may elevate the blood urea nitrogen level. Routine abdominal radiographs are of limited value. Controversy remains as to whether angiography, scintigraphy, colonoscopy, or multidetector CT and in which order, is initial diagnostic procedure in the evaluation of lower GI bleeding.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Emergency stabilization (airway, breathing, and circulation) takes priority. Administer oxygen, insert large-bore intravenous catheters, and institute continuous monitoring.
2. Replace volume loss immediately with isotonic crystalloids (eg, normal saline or Ringer lactate). The decision to transfuse blood is based on clinical factors (continued active bleeding and no improvement in

perfusion after 2 L crystalloids) rather than initial hematocrit values. *The threshold for blood transfusion should be lower in the elderly.*

3. Replace coagulation factors, as needed.
4. A nasogastric tube is recommended in most patients with significant GI bleeding, regardless of the presumed source. If bright red blood or clots are found, perform *gentle* gastric lavage.
5. Consider early therapeutic endoscopy for significant upper GI bleeding. Flexible sigmoidoscopy or colonoscopy can aid in the diagnosis and treatment of some lower GI bleeding sources. *It is estimated that 80% of lower GI bleeding will resolve spontaneously.*
6. Proton pump inhibitors (eg, **Pantoprazole** 80 milligrams bolus followed by a infusion of 8 milligrams/h) reduce rebleeding and the need for surgery for the treatment of bleeding peptic ulcers and are recommended as an adjunct to endoscopic therapy. Histamine-2 antagonists are not beneficial in acute UGI hemorrhage.
7. Consider **octreotide** 25 to 50 micrograms bolus followed by 25 to 50 micrograms/h intravenously for patients with uncontrolled bleeding awaiting endoscopy or when endoscopy is unsuccessful.
8. Balloon tamponade with the Sengstaken-Blakemore tube or its variants should only be considered an adjunctive or temporizing measure and are rarely used due to the high complication rate.
9. A surgical and gastroenterology consult should be obtained in patients with uncontrolled bleeding. Patients who do not respond to medical and endoscopic intervention may require emergency surgical intervention.
10. Most patients with GI bleeding will require hospital admission and early referral to an endoscopist.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 78, “Upper Gastrointestinal Bleeding,” by David T. Overton; and Chapter 79, “Lower Gastrointestinal Bleeding,” by Bruce M. Lo.

# **CHAPTER 40**

## **Esophageal Emergencies**

Mitchell C. Sokolosky

Complaints of dysphagia, odynophagia, or ingested foreign body implicate the esophagus. Chest pain, upper gastrointestinal (GI) bleeding, malignancy, and mediastinitis may also be esophageal in nature. Many diseases of the esophagus can be evaluated over time in an outpatient setting, but several, such as esophageal foreign body and esophageal perforation, must be addressed emergently.

### **DYSPHAGIA**

Dysphagia is difficulty with swallowing. Most patients with dysphagia have an identifiable, organic cause. The two broad pathophysiologic groups of dysphagia are transfer dysphagia (oropharyngeal) and transport dysphagia (esophageal).

#### **Clinical Features**

A careful history is the key to the diagnosis of dysphagia. Determine whether solids, liquids, or both cause the symptoms and the time course and progression of symptoms. Dysphagia for solids that progresses to liquids suggests a mechanical or obstructive process. Dysphagia for both solids and liquids points to a motility disorder. A poorly chewed meat bolus may obstruct the esophagus and be the presenting sign for a variety of underlying esophageal pathologies. Esophageal filling proximal to the impacted bolus can cause inability to swallow secretions and can present an airway or aspiration risk. Physical examination of patients with dysphagia should focus on the head and neck and the neurologic examination although the examination is often normal.



## **Diagnosis and Differential**

The diagnosis of the underlying pathology of dysphagia is most often made outside the emergency department (ED). ED evaluation may include anteroposterior and lateral neck and chest x-rays. Direct laryngoscopy may identify structural lesions. Structural or obstructive causes of dysphagia include neoplasms (squamous cell is most common), esophageal strictures and webs, Schatzki ring, and diverticula. Motor lesions causing dysphagia include neuromuscular disorders (cerebrovascular accident is most common), achalasia, and diffuse esophageal spasm.

## **Emergency Department Care and Disposition**

1. Aspiration is a major concern with most causes of dysphagia.
2. Most causes of dysphagia can be further evaluated and managed in the outpatient setting. Barium swallow is usually the first test for patients with transport dysphagia. Oropharyngeal dysphagia is best worked up by video esophagography.
3. Many of the structural lesions ultimately will require dilatation as definitive therapy.

## **CHEST PAIN OF ESOPHAGEAL ORIGIN**

Differentiating esophageal pain from ischemic chest pain can be difficult or impossible in the ED. Patients with esophageal pain report symptoms that are also found in patients with coronary artery disease, and there is no historical feature that is sensitive or specific enough to differentiate the two. The best ED default assumption is that pain is cardiac in nature and not esophageal until proven otherwise.

## **Gastroesophageal Reflux Disease**

Reflux of gastric contents into the esophagus causes a wide array of symptoms and long-term effects. It affects up to 20% of the adult population in the United States.

### *Clinical Features*

Heartburn is the classic symptom of gastroesophageal reflux disease (GERD) although chest discomfort may be the only symptom. The association of pain with meals, postural changes, and relief of symptoms with antacids point to a diagnosis of GERD. Less obvious presentations of GERD include pulmonary symptoms, especially asthma exacerbations, and multiple ear, nose, and throat symptoms. GERD also has been implicated in the etiology of dental erosion, vocal cord ulcers and granulomas, laryngitis with hoarseness, chronic sinusitis, and chronic cough. Over time, GERD can cause complications such as strictures, inflammatory esophagitis, and Barrett esophagus (a premalignant condition).

### *Diagnosis and Differential*

Diagnosis is suggested by history and favorable response to antacid treatment. However, some patients with symptoms due to cardiac ischemia also report improvement with the same therapy. Unfortunately, like cardiac pain, GERD pain may be squeezing or pressure-like and include a history of onset with exertion or rest. Both types of pain may be accompanied by diaphoresis, pallor, radiation, and nausea and vomiting. An ECG and chest radiograph can be obtained in patients with ambiguous presentations. Given the serious outcome of unrecognized ischemic disease compared with the relatively benign nature of esophageal pain, a cautious approach is warranted.

### *Emergency Department Care and Disposition*

1. Comprehensive treatment of reflux disease is done on an outpatient basis and involves decreasing acid production, enhancing upper tract motility, and eliminating risk factors for the disease.

2. Mild disease often is treated empirically with an H<sub>2</sub> blocker (eg, **ranitidine** 150 milligrams PO twice daily) or proton pump inhibitor (eg, **omeprazole** 20 to 40 milligrams PO daily).
3. Prokinetic drugs (eg, **metoclopramide** 10 to 15 milligrams PO 30 min before meals and at bedtime) may reduce symptoms; decrease dose 50% in elderly patients.
4. Patients should avoid agents that exacerbate GERD (ethanol, caffeine, nicotine, chocolate, or fatty foods), sleep with the head of the bed elevated (30°), and avoid eating within 3 hours of going to bed at night.

## **Esophagitis**

Esophagitis can cause prolonged periods of chest pain and odynophagia. Esophagitis may be inflammatory (eg, GERD, NSAIDs, potassium chloride, doxycycline, tetracycline, clindamycin). Withdrawal of offending agent is generally curative with medication-induced esophagitis. Patients with reflux-induced esophagitis require acid-suppressive medications. Immunosuppressed patients can develop an infectious esophagitis, most commonly *Candida*. The diagnosis is established by outpatient endoscopy.

## **Esophageal Motility Disorder**

Esophageal dysmotility is the excessive, uncoordinated contraction of esophageal smooth muscle. Patients usually complain of dull, colicky chest pain at rest. Onset is typically in the fifth decade. Pain from spasm may respond to nitroglycerin, as well as calcium channel blockers and anticholinergic agents. *Nutcracker esophagus* is a specific type of motility disorder of unknown origin that causes noncardiac chest pain. Manometry helps confirm the diagnosis.

## **ESOPHAGEAL PERFORATION**

Intraluminal procedures are the most common cause of esophageal perforation. Other causes include transient increase in intraesophageal pressure (*Boerhaave syndrome*), trauma, foreign body, infection, tumor, and aortic pathology. Perforation of the esophagus is associated with a high mortality rate.

## **Clinical Features**

Pain is classically described as acute, severe, unrelenting, and diffuse and is reported in the chest, neck, and abdomen. Pain can radiate to the back and shoulders, or back pain may be the predominant symptom. Swallowing often exacerbates pain. Physical examination varies with the severity of the rupture and the elapsed time between the rupture and presentation. Abdominal rigidity with hypotension and fever often occur early. Tachycardia and tachypnea are common. Mediastinal emphysema takes time to develop. It is less commonly detected by examination or radiography in lower esophageal perforation, and its absence does not rule out perforation. A Hammon crunch can sometimes be auscultated. Pleural effusions develop in 50% of patients with intrathoracic perforations and are uncommon in cervical perforations.

## **Diagnosis and Differential**

Chest radiography can suggest the diagnosis. CT of the chest or emergency endoscopy is most often used to confirm the diagnosis. Selection of the procedure depends upon the clinical setting and the resources available. Pain resulting from esophageal perforation often is ascribed to acute myocardial infarction, pulmonary embolus, peptic ulcer disease, aortic catastrophe, or acute abdomen, which results in critical delays in diagnosis.

## **Emergency Department Care and Disposition**

1. Rapid, aggressive management is essential.

2. In the emergency department, initiate fluid resuscitation (see [Chapter 5](#)), and give broad-spectrum parental antibiotics to cover aerobic and anaerobic organisms. Examples include single drug coverage such as **piperacillin/tazobactam** 3.375 grams intravenously (IV) or double drug coverage with **cefotaxime** 2 grams IV or **ceftriaxone** 2 grams IV plus **clindamycin** 600 milligrams IV or **metronidazole** 15 milligrams/kilogram IV once, then 7.5 milligrams/kilogram q6h (max 1 gram/ dose).
3. Obtain emergent surgical consultation.
4. All of these patients require hospitalization.

## Swallowed Foreign Bodies

Children (18 to 48 months old) account for 80% of all cases of ingested foreign bodies. Coins, toys, and crayons typically lodge in the anatomically narrow proximal esophagus. Adult candidates are those with esophageal disease, prisoners, and psychiatric patients. In adults, most impactions are distal. Complications include airway obstruction, stricture, and perforation. Once an object transverses the pylorus, it usually continues through the GI tract. Objects that become lodged distal to the pylorus are usually irregular, have sharp edges, are wide (> 2.5 cm) or long (> 6 cm)

### *Clinical Features*

Objects lodged in the esophagus can produce retrosternal pain, dysphagia, coughing, choking, vomiting, aspiration, and the patient may be unable to swallow secretions. Adults with an esophageal foreign body generally provide unequivocal history. In the pediatric patient it may be necessary to rely on clues such as refusal to eat, vomiting, gagging, choking, stridor, neck or throat pain, dysphagia, and drooling.

### *Diagnosis and Differential*

Physical examination starts with an assessment of the airway. The nasopharynx, oropharynx, neck, and chest should also be examined.

Occasionally, a foreign body can be directly visualized in the oropharynx. Plain films are used to screen for radiopaque objects. Ingested, impacted bones can be seen on plain films only  $\leq 50\%$  of the time. CT scanning has replaced the barium swallow test to evaluate for nonradiopaque objects. Differential diagnosis includes dysphagia, esophageal carcinoma, and gastrointestinal (GI) reflux disease.

#### *Emergency Department Care and Disposition*

1. Patients in extremis or with pending airway compromise are resuscitated in standard fashion and may require active airway management.
2. Emergent endoscopy is indicated for complete distal obstruction of the esophagus with pooling of secretions (often distal esophageal food impaction).
3. Hospital admission is generally not needed if the foreign body is easily removed by endoscopy without complications.
4. In stable patients, indirect or fiberoptic laryngoscopy may allow removal of very proximal objects.
5. Consult surgery for worrisome foreign bodies that are in the more distal GI tract.

### **Food Impaction**

Meat is the most common cause of food impaction.

1. Complete esophageal obstruction requires emergency endoscopy.
2. Uncomplicated food impaction may be treated expectantly but should not be allowed to remain impacted for  $> 12$  to 24 hours.
3. The use of proteolytic enzymes (eg, Adolph Meat Tenderizer, which contains papain) to dissolve a meat bolus is contraindicated.
4. Glucagon (1 to 2 milligrams for adults) may be attempted but success rates are poor.

### **Coin Ingestion**

1. Obtain radiographs on all children suspected of swallowing coins to determine the presence and location of the object. Coins in the esophagus present their circular face on anteroposterior films, as opposed to coins in the trachea, which show that face on lateral films.
2. Coins should be removed by endoscopy if lodged in the esophagus.
3. Removal of a coin with a Foley balloon catheter should be done under fluoroscopy by experienced hands. Complications include aspiration, airway compromise, and mucosal laceration.
4. Once in the stomach, coins almost always pass spontaneously.

## **Button Battery Ingestion**

A button battery lodged in the esophagus is a true emergency requiring prompt removal because the battery may quickly induce mucosal injury and necrosis. Perforation may occur within 6 hours of ingestion.

1. Resuscitate the patient as needed.
2. Obtain radiographs to locate position of the battery.
3. Emergency endoscopy is indicated if battery is lodged in the esophagus. Foley balloon catheter technique may be considered if reliable history of ingestion  $\leq 2$  hours is obtained.
4. Batteries that have passed the esophagus can be managed expectantly with 24-hour follow-up examination. Repeat x-rays at 48 hour to ensure passage through pylorus. Most batteries pass through the body in 48 to 72 hours but may take longer.
5. Consult surgery if the patient develops symptoms or signs of GI tract injury.
6. The National Button Battery Ingestion Hotline at 202-625-3333 is a 24-hours, 7 days-a-week resource for help with management decisions.

## **Ingestion of Sharp Objects**

1. Sharp objects in the esophagus, stomach, or duodenum require immediate removal by endoscopy in order to prevent complications such as perforation.
2. If the object is distal to the duodenum at presentation and the patient is asymptomatic, obtain daily plain films to document passage.
3. Consider surgical removal if 3 days elapse without passage.
4. Consult surgery immediately if the patient develops symptoms or signs of intestinal injury (eg, pain, emesis, fever, GI bleeding).

## **Narcotic Ingestion**

1. The packets (condoms containing up to 5 grams of narcotic) ingested by a narcotic courier (body packer) are often visible on plain x-ray.
2. Endoscopy is contraindicated because of the risk of iatrogenic packet rupture, which may be fatal.
3. Observation until the packet reaches the rectum is the favored treatment if the packets appear to be passing intact through the GI tract.
4. Whole-bowel irrigation may aid in the process of packet removal.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 80, “Esophageal Emergencies, Gastroesophageal Reflux Disease, and Swallowed Foreign Bodies,” by Moss H. Mendelson.



## **CHAPTER 41**

# **Peptic Ulcer Disease and Gastritis**

Matthew C. Gratton

Peptic ulcer disease (PUD) is a chronic illness manifested by recurrent ulcerations in the stomach and duodenum. Acid and pepsin are crucial for ulcer development, but the great majority of ulcers are directly related to infection with *Helicobacter pylori* or nonsteroidal anti-inflammatory drug (NSAID) use. Gastritis is acute or chronic gastric mucosal inflammation and has various causes. Dyspepsia is upper abdominal discomfort with or without other symptoms that can have various causes or be functional.

## **CLINICAL FEATURES**

PUD typically presents with burning epigastric pain, though it may be described as sharp, dull, an ache, or an “empty” or “hungry” feeling. It may be relieved by the ingestion of food, milk or antacids, presumably due to an acid buffering or dilution effect. The pain recurs as the gastric contents empty and the recurrent pain classically awakens the patient at night. Atypical presentations are common in the elderly and may include no pain, pain that is not relieved by food, nausea, vomiting, anorexia, weight loss, and/or bleeding.

A change in the character of the pain may herald the onset of a complication. Abrupt onset of severe pain is typical of perforation with spillage of gastric or duodenal contents into the peritoneal cavity. Back pain may represent pancreatitis from a posterior perforation. Nausea, vomiting, early satiety and weight loss may occur with gastric outlet obstruction or cancer. Vomiting blood or passing melanotic stools with or without hemodynamic instability represent a bleeding complication.

## **DIAGNOSIS AND DIFFERENTIAL**

PUD cannot be definitively diagnosed on clinical grounds, but it can be strongly suspected in the presence of a “classic” history (as above) accompanied by “benign” physical examination findings and normal vital signs with or without mild epigastric tenderness. Examination findings that may be indicative of PUD complications include: a rigid abdomen consistent with peritonitis in perforation; abdominal distension and succussion splash consistent with gastric outlet obstruction; occult or gross rectal blood or blood in nasogastric aspirate consistent with bleeding.

The differential diagnosis of epigastric pain is extensive. Pain radiating into the chest, water brash, and belching may point to gastroesophageal reflux disease; more severe pain in the right upper quadrant (RUQ) radiating around the right side with tenderness suggests cholelithiasis or biliary colic; pain radiating into the back is common with pancreatitis and a concomitant mass may represent a pseudocyst or if the mass is pulsatile it could represent an abdominal aortic aneurysm. Chronic pain, anorexia, and weight loss with or without a mass may represent cancer. Myocardial ischemia may present as epigastric pain and should be strongly considered in the appropriate clinical setting.

Some ancillary tests may be helpful to exclude PUD complications and to narrow the differential. A normal CBC rules out chronic (but not acute) bleeding. Elevated liver enzymes may indicate hepatitis and elevated lipase may indicate pancreatitis. An upright CXR may show the free air of a perforation and an abdominal US examination may show cholecystitis, cholelithiasis, or an abdominal aortic aneurysm. An ECG and troponin are indicated if myocardial ischemia is suspected.

The gold standard for diagnosis of PUD is visualization of an ulcer by upper GI endoscopy. Endoscopy is indicated in most patients with upper GI bleeding and in any patient with certain “alarm” features consistent with cancer: age > 55 year, unexplained weight loss, early satiety or anorexia, persistent vomiting, dysphagia, anemia, abdominal mass, or jaundice. Because of the strong association of *H pylori* infection with PUD, testing for the presence of *H pylori* is usually indicated, but this is generally more appropriate at the time of follow-up.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

After PUD is diagnosed, the goal of treatment is to heal the ulcer while relieving pain and preventing complications and avoiding recurrence. If the patient is infected with *H pylori* then it must be eradicated in order to prevent ulcer recurrence. NSAIDs should be stopped whenever possible.

1. Proton pump inhibitors (PPIs) heal ulcers faster than other therapies and also have an inhibitory effect on *H pylori*. PPIs should be taken about 30 to 60 min prior to a meal. Include: **omeprazole** 20 to 40 milligrams daily; **esomeprazole** 20 to 40 milligrams daily; **lansoprazole** 15 to 30 milligrams daily; **pantoprazole** 20 to 40 milligrams daily; and **rabeprazole** 20 milligrams daily.
2. Histamine-2 receptor antagonists (H<sub>2</sub>RAs) inhibit acid secretion and are available over the counter. H<sub>2</sub>RAs include: **cimetidine** 200 to 400 milligrams twice a day; **famotidine** 10 to 20 milligrams twice a day; **nizatidine** 75 to 150 milligrams twice a day; and **ranitidine** 75 to 150 milligrams twice a day.
3. Liquid antacids relieve pain and heal ulcers by buffering gastric acid. Due to the minimal side effects of PPIs and H<sub>2</sub>RAs, liquid antacids are generally used on an as needed basis for pain relief. Typical dosing is 15 mL 1 hour after meals and at bedtime.
4. If present, *H pylori* infection should be treated, though this would only rarely be initiated in the ED. There are multiple therapies, but the most common is “triple therapy” for 14 days with a PPI, clarithromycin, and either amoxicillin or metronidazole.
5. Patients with complications always require consultation and most require admission for continued treatment. For the treatment of bleeding, see [Chapter 39](#). For perforation provide resuscitation as needed, place a nasogastric tube, start broad-spectrum antibiotics, and obtain immediate surgical consultation. For gastric outlet obstruction provide resuscitation as needed, place a nasogastric tube, and admit for continued treatment.

6. When uncomplicated PUD is suspected in a stable patient, the great majority can be discharged home on a PPI or an H<sub>2</sub> RA with a liquid antacid for breakthrough pain and with recommendations to follow-up with their primary care provider for further evaluation as indicated. Patients with “alarm” features who are stable enough for discharge, should be referred for endoscopy.
7. Patients should be told that PUD is a presumptive diagnosis and that they should return for further evaluation or treatment if any of the following occur: worsening pain, increased vomiting, hematemesis or melena, weakness or syncope, fever, or chest pain.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 81, “Peptic Ulcer Disease and Gastritis,” by Matthew C. Gratton.

## **CHAPTER 42**

# **Pancreatitis and Cholecystitis**

Casey Glass

Acute pancreatitis (AP) is a common cause of abdominal pain, and the diagnosis is based primarily on the patient's history and clinical examination findings. The severity of the disease may range from mild local inflammation to multisystem organ failure secondary to a systemic inflammatory response. Cholelithiasis and alcohol abuse are the most common causes, but there are many potential etiologies. Patients without risk factors often develop pancreatitis secondary to medications or severe hyperlipidemia. Commonly used medications associated with pancreatitis include acetaminophen, carbamazepine, enalapril, estrogens, erythromycin, furosemide, hydrochlorothiazide, opiates, steroids, tetracycline, and trimethoprim-sulfamethoxazole.

## **PANCREATITIS**

### **Clinical Features**

The most common symptom is a midepigastic, constant, boring pain radiating to the back, which is often associated with nausea, vomiting, abdominal distention, and exacerbation in the supine position. Low-grade fevers, tachycardia, and hypotension may be present. Epigastric tenderness is common, whereas peritonitis is a late finding.

Physical findings are dependent on the severity of disease. Physical examination findings include epigastric tenderness but tenderness may localize more to the right or left upper quadrant of the abdomen. Bowel sounds may be diminished and abdominal distention may be present secondary to ileus. Refractory hypotensive shock, renal failure, fever, altered mental status, and respiratory failure may accompany the most severe disease.

## **Diagnosis and Differential**

The diagnosis should be suspected by the history and physical examination. The presence of two of the three following features makes the diagnosis more likely: (1) history and examination findings consistent with acute pancreatitis, (2) lipase or amylase levels at least 2 to 3 times the upper limit of normal, or (3) imaging findings consistent with pancreatic inflammation. Serum lipase and amylase are the most common tests used to assist in the diagnosis but lipase is the preferred diagnostic test as it is more accurate. There are many sources of extrapancreatic amylase, making it relatively nonspecific. Normal serum amylase does not exclude the diagnosis of acute pancreatitis. There is no benefit to ordering both tests. The absolute levels do not correlate with the severity of disease.

A CBC will identify leukocytosis or anemia. Liver studies can demonstrate associated biliary involvement. An elevated alkaline phosphatase level suggests biliary disease and gallstone pancreatitis. Persistent hypocalcemia (<7 milligrams/100 mL), hypoxia, increasing serum urea nitrogen, and metabolic acidosis are associated with a potentially complicated course.

Imaging can help confirm the diagnosis of pancreatitis, evaluate biliary involvement, and exclude causes of abdominal pain. Abdominal CT scan is preferred over ultrasound as the latter is often limited by bowel gas overlying the pancreas. In the face of a typical clinical picture and laboratory results, emergency imaging may not be needed.

The differential diagnosis includes referred chest pain secondary to ischemic heart disease, pulmonary pathology such as pneumonia or empyema, hepatitis, cholecystitis or biliary colic, ascending cholangitis, renal colic, small bowel obstruction, peptic ulcer disease or gastritis, and acute aortic pathology such as aneurysm or dissection.

## **Emergency Department Care and Disposition**

Care for the patient with pancreatitis includes fluid resuscitation; management of nausea, vomiting, and pain; and diligent monitoring of vital

signs and pulse oximetry.

1. Initiate aggressive fluid resuscitation with crystalloid intravenous fluid. Pressors are indicated for hypotension not responsive to adequate fluid resuscitation.
2. Patients should be made npo to allow pancreatic rest.
3. Administer antiemetics, such as **ondansetron** 4 milligrams or **prochlorperazine** 5 to 10 milligrams to reduce vomiting (see [Chapter 36](#)). A nasogastric tube is indicated only for intractable vomiting.
4. Administer parenteral analgesia for patient comfort. Intravenous opioids such as **morphine** 0.1 milligram/kilogram are often required.
5. Administer oxygen to maintain a pulse oximetry reading of 95% oxygen saturation. Treat respiratory failure aggressively.
6. Treat patients with infected pseudocyst, abscess, or infected peripancreatic fluid with **imipenem-cilastatin** 500 milligrams IV, **meropenem** 1 gram IV, or **ciprofloxacin** 400 milligrams IV and **metronidazole** 500 milligrams IV.
7. Patients with severe systemic disease will require intubation, intensive monitoring, bladder catheterization, and transfusion of blood and blood products as needed. Symptomatic hypocalcemia should be corrected. Laparotomy may be indicated for hemorrhage or abscess drainage.
8. Consult gastroenterology for patients with gallstone pancreatitis for endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy.
9. Most patients will require hospitalization. Patients who demonstrate poor prognostic signs (dropping hemoglobin, poor urine output, persistent hypotension, hypoxia, acidosis, or hypocalcemia) despite aggressive early treatment should be admitted to the intensive care unit with surgical consultation.
10. Patients with mild disease, no biliary tract disease, and no evidence of systemic complications may be managed as outpatients with close follow-up if they tolerate clear liquids and oral analgesics in the ED. Instruct patients to increase their diet as tolerated once nausea is controlled.

## CHOLECYSTITIS

Biliary tract emergencies most often result from obstruction of the gallbladder or biliary duct by gallstones. The 4 most common biliary tract emergencies caused by gallstones are biliary colic, cholecystitis, gallstone pancreatitis, and ascending cholangitis. Biliary disease affects all age groups, especially diabetics and the elderly. Gallstones, although common in the general population, remain asymptomatic in most patients. Common risk factors for gallstones and cholecystitis include advanced age, female sex and parity, obesity, rapid weight loss or prolonged fasting, familial tendency, use of some medications, Asian ancestry, chronic liver disease, and hemolytic disorders (eg, sickle cell disease).

## **Clinical Features**

Patients with biliary disease present with a wide range of symptoms. Biliary colic may present with epigastric or right upper quadrant pain, may range from mild to severe, and, although classically described as intermittent or colicky, is often constant. Nausea and vomiting are usually present. Pain may be referred to the right shoulder or left upper back. It may begin after eating but often bears no association to meals. Acute episodes of biliary colic typically last for 1 to 5 hours, followed by a gradual or sudden resolution of symptoms. Recurrent episodes are usually infrequent, generally at intervals longer than 1 week. Biliary colic seems to follow a circadian pattern, with highest incidence of symptoms between 9 PM and 4 AM.

Physical examination commonly demonstrates right upper quadrant or epigastric tenderness without findings of peritonitis.

Acute cholecystitis presents with pain similar to that of biliary colic that persists for longer than the typical 5 hours. Fever, chills, nausea, emesis, and anorexia are common. Past history of similar attacks or known gallstones may be reported. As the gallbladder becomes progressively inflamed, the initial poorly localized upper abdominal pain often becomes sharp and localized to the right upper quadrant. The patient may have moderate to severe distress and may appear toxic. Choledocholithiasis often presents with midline pain that radiates to the middle of the back.



Examination findings include tenderness in the right upper quadrant or epigastrium, and Murphy's sign, that is increased pain or inspiratory arrest during deep subcostal palpation of the right upper quadrant during deep inspiration. Murphy's sign is the most sensitive physical examination finding for the diagnosis of cholecystitis. Generalized abdominal rigidity suggests perforation and diffuse peritonitis. Volume depletion is common, but jaundice is unusual. Acalculous cholecystitis occurs in 5% to 10% of patients with cholecystitis, has a more rapid, aggressive clinical course, and occurs more frequently in patients with diabetes, the elderly, trauma or burn victims, after prolonged labor or major surgery, or with systemic vasculitides.

Ascending cholangitis, a life-threatening condition with high mortality, results from complete biliary obstruction (often a common bile duct stone; less commonly a tumor) with bacterial superinfection. Patients often present in extremis with jaundice, fever, confusion, and shock. Examination findings can be subtle. Patients commonly have focal right upper quadrant pain and nausea. Jaundice may or may not be present. The Charcot triad of fever, jaundice, and right upper quadrant pain is suggestive but all three components are usually not present at once.

## **Diagnosis and Differential**

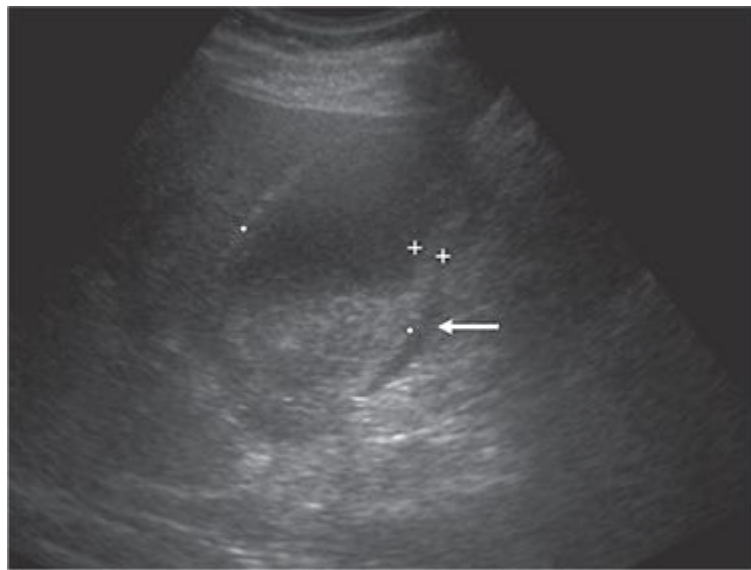
Suspicion of gallbladder or biliary tract disease must be maintained in any patient who presents with upper abdominal pain. The differential diagnosis is similar to that of acute pancreatitis (see Pancreatitis: Diagnosis and Differential, earlier).

Patients with uncomplicated biliary colic usually have normal laboratory findings. The diagnosis is usually made based on the patient presentation, response to therapy, and examining the test results in aggregate.

Laboratory studies that may aid in diagnosis include a white blood cell count; leukocytosis with left shift suggests acute cholecystitis, pancreatitis, or cholangitis, but a normal white blood cell count does not exclude them. Serum bilirubin and alkaline phosphatase levels may be normal or mildly elevated in patients suffering from biliary colic or cholecystitis. Serum bilirubin and alkaline phosphatase levels are usually elevated in cases of

choledocholithiasis and ascending cholangitis. Serum lipase or amylase levels should be checked to help exclude associated pancreatitis.

Ultrasound of the hepatobiliary tract is the initial diagnostic study of choice for patients with suspected biliary colic or cholecystitis and has a sensitivity and specificity for cholecystitis of 94% and 78% respectively. It can detect stones as small as 2 mm and signs of cholecystitis which include a thickened gallbladder wall ( $> 3$  to 5 mm), gallbladder distention ( $> 4$  cm in short-axis view), and pericholecystic fluid. A positive sonographic Murphy's sign is very sensitive for diagnosis of cholecystitis when it is elicited during the scan. Ultrasound has a strong positive predictive value (92%) when both a sonographic Murphy's sign and gallstones are present. Choledocholithiasis is suggested when the common bile duct diameter is greater than 5 to 7 mm. ([Fig. 42-1](#)).



**FIGURE 42-1.** Abdominal US image showing acute cholecystitis with pericholecystic fluid (white arrow), gallbladder wall thickening (white pluses), and enlarged short-axis dimension (white dots). (Courtesy of Mustafa Secil, MD.)

Computed tomography of the abdomen is most useful when other intraabdominal processes are suspected. Radionuclide cholescintigraphy (technetium-iminodiacetic acid [HIDA]) or diisopropyl iminodiacetic acid

([DISIDA] scans) offers a sensitivity of 97% and a specificity of 90% for cholecystitis. A reasonable emergency department approach to suspected cholecystitis would be to obtain an ultrasound scan and then a radionuclide scan if ultrasound fails to establish the diagnosis.

## **Emergency Department Care and Disposition**

Care for the patient with biliary disease includes fluid resuscitation and management of nausea, vomiting, and pain. Only biliary colic can be managed without the aid of consultants. ED treatment includes the following measures:

1. Initiate aggressive fluid resuscitation with crystalloid intravenous fluid. Pressors are indicated for hypotension not responsive to adequate fluid resuscitation.
2. Patients should be made *npo* to allow pancreatic rest.
3. Administer antiemetics, such as **ondansetron** 4 milligrams or **prochlorperazine** 5 to 10 milligrams to reduce vomiting (see [Chapter 36](#)). A nasogastric tube is indicated only for intractable vomiting.
4. Administer parenteral analgesia for patient comfort. Intravenous opioids such as **morphine** 0.1 milligram/kilogram are often required. The intravenous nonsteroidal anti-inflammatory drug (NSAID) **ketorolac** 30 milligrams IV may also be helpful.
5. A nasogastric tube to low suction should be considered if the patient is distended or actively vomiting, or if vomiting is intractable to antiemetics.
6. Patients with acute biliary obstruction may require urgent decompression via endoscopic sphincterotomy of the ampulla of Vater.
7. Early antibiotic therapy should be initiated in any patient with suspected cholecystitis or cholangitis. Adequate therapy for uncomplicated cases of cholecystitis includes a parenteral third-generation cephalosporin (**cefotaxime** or **ceftriaxone** 1 gram IV q12 to q24h) plus **metronidazole** 500 milligrams IV. Those with ascending cholangitis, sepsis, or obvious peritonitis are best managed with triple coverage by using **ampicillin** (0.5 to 1.0 gram IV q6h), **gentamicin** (1 to 2 milligrams/kilogram IV q8h),

and **clindamycin** (600 milligrams IV q6h, or the equivalent substitutes (eg, metronidazole for clindamycin, third-generation cephalosporins or piperacillin/tazobactam, or a fluoroquinolone for ampicillin).

8. Patients diagnosed with acute cholecystitis, gallstone pancreatitis, or ascending cholangitis require immediate surgical consultation with hospital admission. Patients with choledocholithiasis, gallstone pancreatitis, or ascending cholangitis may also require urgent gastroenterology consultation to facilitate ERCP and sphincterotomy. Signs of systemic toxicity or sepsis warrant admission to the intensive care unit pending surgical treatment.
9. Patients with uncomplicated biliary colic whose symptoms abate with supportive therapy within 4 to 6 hours of onset can be discharged home if they are able to maintain oral hydration. Oral opioid analgesics may be prescribed for the next 24 to 48 hours for the common residual abdominal aching. Timely outpatient follow-up should be arranged with a surgical consultant or the patient's primary care physician. The patient should be carefully instructed to return to the emergency department if fever develops, abdominal pain worsens, for intractable vomiting, or if another significant attack occurs before follow-up.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 82, "Pancreatitis and Cholecystitis" by Ridvan Atilla and Cem Oktay.

# **CHAPTER 43**

## **Acute Appendicitis**

Charles E. Stewart

Appendicitis is one of the most common surgical emergencies. Despite advances in laboratory testing and imaging, accurate diagnosis of appendicitis remains a challenge. Complications from misdiagnosis of appendicitis include intraabdominal abscess, wound infection, adhesion formation, bowel obstruction, and infertility.

### **CLINICAL FEATURES**

The most reliable symptom in appendicitis is abdominal pain. The early signs of appendicitis are quite nonspecific and progress with time. The location of the pain depends on the location of the appendix. Pain commonly begins in the periumbilical or epigastric region. As peritoneal irritation occurs, the pain will often localize to the right lower quadrant. Other symptoms associated with appendicitis include anorexia, nausea, and vomiting but these symptoms are neither sensitive nor specific. As the pain increases, irritation of the bladder and/or colon may cause dysuria, tenesmus, or other symptoms. Many patients have the “bump” sign, where the patient notes an increase in the abdominal pain associated with bumps in the ride to the hospital. If the pain suddenly decreases the examiner should consider appendiceal perforation.

The classic point of maximal tenderness is in the right lower quadrant just below the middle of a line connecting the umbilicus and the right anterior superior iliac spine. (McBurney’s point). Patients may also have pain referred to the right lower quadrant when palpating the left lower quadrant (Rovsing sign), pain elicited by extending the right leg to the hip while lying in the left lateral decubitus position (psoas sign), or pain elicited by passively flexing the right hip and knee and internally rotating the hip (obturator sign). Patients with a pelvic appendix may be quite tender on

rectal examination, and patients with a retrocecal appendix may have more prominent flank pain than abdominal pain. No individual physical finding is sensitive or specific enough to rule in or rule out the diagnosis.

Fever is a relatively late finding in appendicitis and rarely exceeds 39°C (102.2°F), unless rupture or other complications occur.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of acute appendicitis is primarily clinical. Symptoms with high sensitivity for appendicitis include right lower quadrant pain, pain that occurs before vomiting, and absence of prior similar pain. Migration of the pain is thought to be highly specific for appendicitis. Physical signs with high specificity include right lower abdominal rigidity and positive psoas sign. Additional studies, such as complete blood count, C-reactive protein, urinalysis, and imaging studies, may be performed if the diagnosis is unclear. A pregnancy test must be performed in all females of reproductive age. A normal WBC does not rule out appendicitis. Urinalysis is useful to help rule out other diagnoses but pyuria and hematuria can occur when an inflamed appendix irritates the ureter.

The differential diagnosis of right lower quadrant pain is wide and includes other gastrointestinal processes (eg, inflammatory bowel disease, hernia, abscess, volvulus, diverticulitis), gynecologic or urological processes (eg, ectopic pregnancy, ovarian torsion, renal colic, genitourinary (GU) infection or abscess), or musculoskeletal processes (eg, muscular hematoma or abscess).

**Plain radiographs** of the abdomen are not helpful.

**Computed tomography (CT)** is the imaging study of choice with an overall sensitivity of 96% and PPV of 96%. CT findings suggesting acute appendicitis include pericecal inflammation, abscess, and periappendiceal phlegmon or fluid collections ([Fig. 43-1](#)). CT has been shown to change management in women, decreasing unnecessary tests. CT findings of appendicitis may be lacking in the thin patient. CT may be conducted with or without contrast administered orally, intravenously, or rectally, depending on institutional experience and preference. Unenhanced CT

scanning has 92% sensitivity and 96% specificity. Increased concerns about accumulated radiation exposure for children, potentially childbearing females, and pregnant patients have led to interest in alternatives to the CT scan.



**FIGURE 43-1.** Acute appendicitis on contrast CT scan as evidenced by dilated and inflamed appendix.

**Ultrasonography** has a high sensitivity but is limited both by operator skill and in evaluating a ruptured appendix or an abnormally located (eg, retrocecal) appendix. Graded compression ultrasonography is the initial modality of choice in both children and the pregnant patient to decrease radiation exposure. Overall sensitivity of ultrasound is 86% with PPV of 95%.

**Magnetic resonance imaging** for the diagnosis of appendicitis is evolving as a reliable technology that avoids completely the ionizing radiation risks. IV contrast with MRI should be avoided in the pregnant patient as it crosses the placenta.

Very young children, the elderly, pregnant women, and patients with AIDS have higher rates of misdiagnosis of appendicitis, with increased morbidity and mortality rates. Patients younger than 6 years have a high misdiagnosis rate due to poor communication skills and the association of many nonspecific symptoms. Elderly patients may have decreased perceptions of symptoms. Pregnant patients are at risk for misdiagnosis even though appendicitis is the most common extrauterine surgical emergency in pregnancy. Patients with acquired immunodeficiency syndrome are susceptible to complications from appendicitis because of delays in diagnosis due to their frequently preexisting gastrointestinal symptoms and their immunocompromised state.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Surgical consultation should be obtained before imaging when the diagnosis is thought to be clear. Patient should have nothing by mouth and should have intravenous (IV) access and fluids. The treatment for acute appendicitis is appendectomy.
2. Control pain with opioid analgesics, such as **fentanyl** 1 to 2 micrograms/kilogram IV q1 to q4h or **morphine**, 0.1 milligram/kilogram.
3. Antibiotics given before surgery decrease the incidence of postoperative wound infection or, in cases of perforation, postoperative abscess formation. Several antibiotic regimens to cover anaerobes, enterococci, and gram-negative intestinal flora have been recommended, including **piperacillin/tazobactam** 3.375 grams IV or **ampicillin/sulbactam** 3 grams IV. Consult with the surgeon regarding the antibiotic regimen and timing.
4. In patients for whom the diagnosis is not clear, admit for observation, serial examinations, and surgical consultation. This is a safe option for high-risk patients (pediatric, geriatric, pregnant, or immunocompromised).



5. Stable, nontoxic-appearing patients with adequate pain control who can tolerate oral hydration, have no significant comorbidities, and are able to return for reevaluation in 12 hours may be considered for discharge and 12-hour follow-up. These patients should be instructed to avoid strong analgesics, and should return if they develop increased pain, localization of the pain, fever, nausea, or other signs or symptoms of illness that are worsening or not resolving.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 84, “Acute Appendicitis,” by E. Paul DeKoning.

# **CHAPTER 44**

## **Diverticulitis**

James O'Neill

### **DIVERTICULITIS**

Diverticular disease is a common GI disorder that occurs when small herniations through the wall of the colon, or diverticula, become inflamed or infected.

#### **Clinical Features**

The most common symptom is a steady, deep discomfort in the left lower quadrant of the abdomen. Other symptoms include tenesmus and changes in bowel habits, such as diarrhea or increasing constipation and nausea/vomiting. Urinary tract symptoms are less common. Patients with a redundant sigmoid colon, of Asian descent, or with right-sided disease may complain of pain in other abdominal regions, including the right lower quadrant. The presentation can mimic other diseases, such as appendicitis. Half of patients will describe a similar prior episode.

Patients have a low-grade fever, but the temperature may be higher in patients with generalized peritonitis and in those with an abscess. Physical findings range from mild abdominal tenderness to severe pain, obstruction, and peritonitis. Occult blood may be present in the stool. A pelvic examination should be performed in female patients to exclude a gynecologic source of symptoms.

#### **Diagnosis and Differential**

The differential diagnosis includes acute appendicitis, colitis (ischemic or infectious), inflammatory bowel disease (Crohn disease or ulcerative colitis), colon cancer, irritable bowel syndrome, pseudomembranous colitis, epiploic appendagitis, gallbladder disease, incarcerated hernia, mesenteric infarction, complicated ulcer disease, peritonitis, obstruction, ovarian torsion, ectopic pregnancy, ovarian cyst or mass, pelvic inflammatory disease, sarcoidosis, collagen vascular disease, cystitis, kidney stone, renal pathology, and pancreatic disease.

Diverticulitis can be diagnosed by clinical history and examination alone. In stable patients with past similar acute presentations, no further diagnostic evaluation is necessary unless the patient fails to improve with conservative medical treatment. If a patient does not have a prior diagnosis or the current episode is different from past episodes, diagnostic imaging should be performed to rule out other intraabdominal pathology and evaluate for complications. CT scan is the preferred imaging modality for its ability to evaluate the severity of disease and the presence of complications. CT with IV and oral contrast has documented sensitivities of 97% and specificities approaching 100%. Laboratory tests, such as a CBC, liver function tests, and urinalysis, are rarely diagnostic but may help exclude other diagnoses.

## **Emergency Department Care and Disposition**

ED care begins with fluid and electrolyte replacement, pain and nausea control. Ill appearing patients, those with uncontrolled pain, vomiting, peritoneal signs, signs of systemic infection, comorbidities or immunosuppression, and those with complicated diverticulitis (eg, phlegmon, abscess, obstruction, fistula, or perforation) require admission and surgical consultation.

1. Place the patient on complete bowel rest. Opiates, such as **morphine** 0.1 milligram/kilogram IV, may be required for pain. Nasogastric suction may be indicated in patients with bowel obstruction or adynamic ileus.
2. Administer IV antibiotics to patients requiring admission. Options include **metronidazole** 500 milligrams IV with either **ciprofloxacin** 400 milligrams IV or **levofloxacin** 750 milligrams IV. Alternate single agent

treatment options include **ampicillin-sulbactam**, 3 grams IV, **piperacillin-tazobactam**, 3.35 grams IV, **ertapenem**, 1 gram IV, **ticarcillin-clavulanate**, 3.1 grams IV or **moxifloxacin**, 400 milligrams IV. Patients with very severe disease may require extended broad-spectrum antibiotics such as **imipenem** 500 milligrams IV, **meropenem** 1 gram IV, or **doripenem** 500 milligrams IV.

3. Immunocompetent patients with uncomplicated diverticulitis who look well, have mild findings on physical examination and in whom pain is controlled with oral analgesia may be managed as outpatients with oral antibiotics for 7 to 14 days, on a clear liquid diet that is advanced as tolerated, and close follow-up (2 to 3 days). Patients should contact their physicians or return to the ED if they develop increasing abdominal pain or fever or are unable to tolerate oral intake.

Oral antibiotic regimens include **metronidazole** 500 milligrams every 8 hours plus either **ciprofloxacin** 500 milligrams every 12 hours or **clindamycin** 300 milligrams every 6 hours or trimethoprim-sulfamethoxazole DS, 1 tablet every 12 hours. Monotherapy includes **amoxicillin-clavulanate** 875 milligrams every 12 hours, and **moxifloxacin**, 400 milligrams PO once a day.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 85, "Diverticulitis," by Autumn Graham.

## **CHAPTER 45**

# **Intestinal Obstruction and Volvulus**

Mark Hess

Intestinal obstruction results from mechanical blockage or the loss of normal peristalsis. Adynamic or paralytic ileus is more common and usually self-limiting. Common causes of mechanical small bowel obstruction (SBO) are adhesions due to previous surgery, incarcerated hernias, or inflammatory diseases. Other causes to consider are inflammatory bowel diseases, congenital anomalies, and foreign bodies. The most frequent causes of large bowel obstructions are cancer, diverticulitis with stricture, sigmoid volvulus, and fecal impaction. Consider intussusception in children. Sigmoid volvulus is more common in the elderly taking anticholinergic medications while cecal volvulus is more common in gravid patients. Intestinal pseudoobstruction (Ogilvie syndrome) may mimic large bowel obstruction. The elderly and patients taking anticholinergic medications are at increased risk for pseudoobstruction.

## **CLINICAL FEATURES**

Crampy, intermittent, progressive abdominal pain and inability to have a bowel movement or to pass flatus are common presenting complaints. Vomiting, bilious in proximal obstructions and feculent in distal obstruction, is usually present. Patients with partial SBO can still pass flatus. Physical signs vary from abdominal distention, localized or general tenderness, to obvious signs of peritonitis. Localization of pain and the presence of abdominal surgical scars, hernia, or masses may provide clues to the site of obstruction. Active, high-pitched bowel sounds can be heard in mechanical SBO. Rectal examination may demonstrate fecal impaction, rectal carcinoma, or occult blood. The presence of stool in the rectum does not exclude obstruction. Consider a pelvic examination in women. Systemic

symptoms and signs depend on the extent of dehydration and the presence of bowel necrosis or infection.

## DIAGNOSIS AND DIFFERENTIAL

Suspect intestinal obstruction in any patient with abdominal pain, distention, and vomiting, especially in patients with previous abdominal surgery or groin hernias.

Flat and upright abdominal radiographs and an upright chest x-ray can screen for obstruction (see [Fig. 45-1](#)), confirm severe constipation, or diagnose hollow viscus perforation with free air. The diagnostic procedure of choice in the ED is CT scanning using IV and oral contrast when possible. CT scanning can delineate partial versus complete bowel obstruction, partial SBO versus ileus, and strangulated versus simple SBO.



**FIGURE 45-1.** Sigmoid volvulus. Note that the open portion of the “C” formed by the twisted large bowel points toward the left side in the case of sigmoid volvulus.

Laboratory tests may include a complete blood count, electrolytes, blood urea nitrogen, creatinine, lactate levels, coagulation profile, and type and cross-match. Suspect abscess, gangrene, or peritonitis if leukocytosis > 20,000 or left shift is noted. An elevated hematocrit is consistent with dehydration.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

ED care is directed at vigorous fluid resuscitation with crystalloids, careful monitoring of response, and prompt surgical consultation. Surgical intervention is usually necessary to treat a mechanical obstruction.

1. Decompress the bowel with a nasogastric tube especially if vomiting or distension is present.
2. Administer preoperative broad-spectrum intravenous antibiotics coverage such as **piperacillin/tazobactam** 3.375 grams or **ampicillin/ sulbactam** 3.0 grams or double drug coverage with **cefotaxime** 2 grams or **ceftriaxone** 2 grams plus **clindamycin** 600 milligrams or **metronidazole** 1 gram.
3. When the diagnosis is uncertain or if adynamic ileus is suspected, conservative measures, such as intravenous fluids and observation without surgical intervention, may be appropriate.
4. In patients with pseudoobstruction, colonoscopy is both diagnostic and therapeutic. Surgery is not indicated.

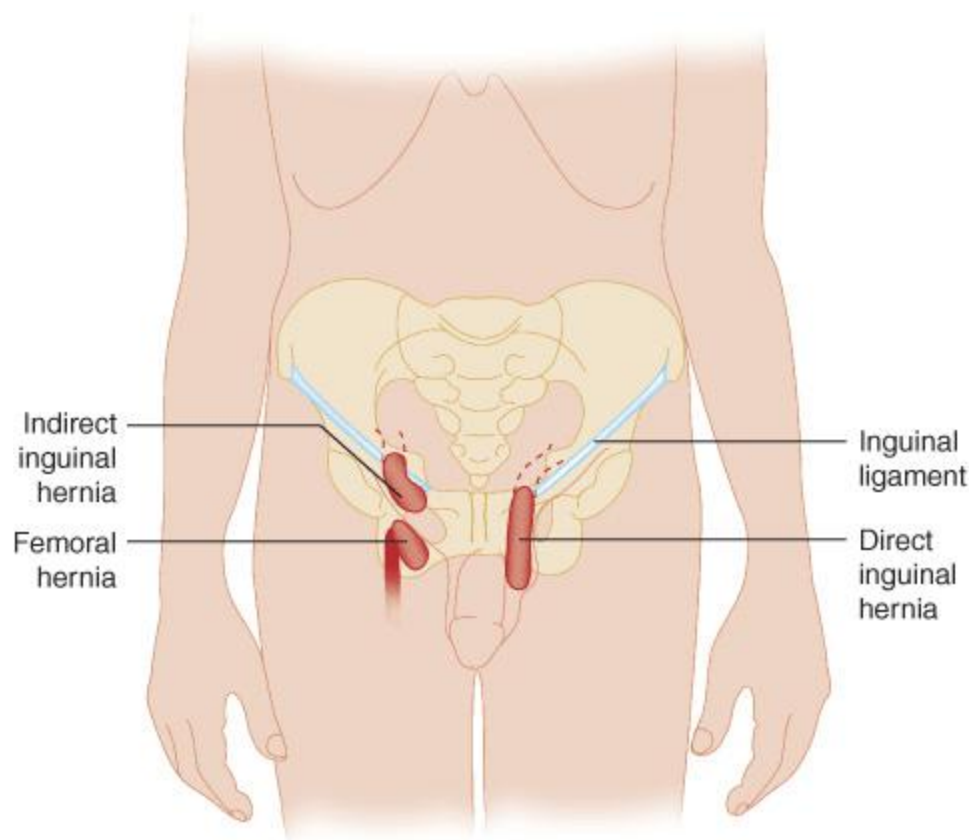
For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 86, "Bowel Obstruction and Volvulus," by Salvator J. Vicario and Timothy G. Price.

# CHAPTER 46

## Hernia in Adults and Children

Dave W. Lu

Hernia is a protrusion of any viscus from its normal cavity. Hernias are classified by anatomic location, hernia contents, and the status of those contents (eg, reducible, incarcerated, or strangulated). Hernia is typically used to describe a protrusion of bowel through the abdominal wall. The most common abdominal hernias are inguinal, ventral, and femoral hernias ([Fig. 46-1](#)).



**FIGURE 46-1.** Groin hernias.



Predisposing factors include family history, lack of developmental maturity, undescended testes, genitourinary abnormalities, conditions that increase intraabdominal pressure (eg, ascites or pregnancy), chronic obstructive pulmonary disease, and surgical incision sites.

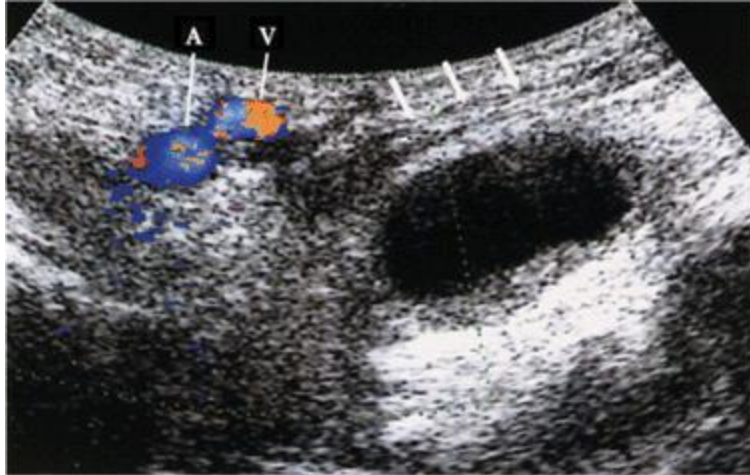
## **CLINICAL FEATURES**

Most hernias are detected on routine physical examination or inadvertently by the patient. When the contents of a hernia can be easily returned to their original cavity by manipulation, the hernia is defined as *reducible*. A hernia becomes *incarcerated* when its contents are not reducible. Incarcerated hernias may lead to bowel obstruction and strangulation. *Strangulation* refers to vascular compromise of the incarcerated contents and is an acute surgical emergency. When not relieved, strangulation may lead to gangrene, perforation, peritonitis, and septic shock.

Symptoms other than an obvious protruding mass from the abdominal wall include localized pain, nausea, and vomiting. Signs of strangulation include severe pain and tenderness, induration and erythema over the site. Children may exhibit irritability and poor feeding. Careful evaluation for obstruction is essential.

## **DIAGNOSIS AND DIFFERENTIAL**

Physical examination is the predominant means of diagnosis. Laboratory testing is of minimal value. Ultrasonographic detection of hernias is operator and body habitus dependent, but can be helpful in pediatric and pregnant patients where radiation exposure is a concern ([Fig. 46-2](#)). Computed tomography remains the best radiographic test for the evaluation of hernias.



**FIGURE 46-2.** Ultrasonographic detection of incarcerated hernia. An incarcerated obturator hernia is demonstrated deep in the femoral region. It locates posterior to the pectineus muscle (arrows) and medial to the femoral artery (A) and vein (V). (Reproduced with permission from Ma OJ, Mateer JR, Blaivas M (eds): *Emergency Ultrasound*, 2nd ed. Copyright © 2008 The McGraw-Hill Companies, All rights reserved.)

The differential diagnosis of a groin mass includes direct or indirect hernia, testicular torsion, tumor, groin abscess, hydrocele, varicocele, and hidradenitis. In children, retracted or undescended testes may be mistaken for inguinal hernias.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Do not attempt reduction if signs of strangulation exist so as not to introduce dead bowel into the abdomen.

1. To reduce a hernia, (*a*) place the patient in Trendelenburg, (*b*) externally rotate and flex the ipsilateral leg into the frog-leg position, (*c*) administer adequate analgesia or procedural sedation; children will require procedural sedation, (*d*) place a padded ice pack to reduce swelling and blood flow to the area, and (*e*) grasp and elongate the hernia neck with

one hand, and with the other hand apply firm, steady pressure to guide the hernia through the fascial defect.

2. Adults with easily reducible hernias can be referred for outpatient surgical evaluation and repair. Patients should avoid heavy lifting and return to the ED if herniation recurs and cannot be reduced promptly. Discuss signs of obstruction.
3. Incarcerated hernias that can't be reduced with 1 or 2 attempts and strangulated hernias require emergent surgical consultation and intervention. Give nothing by mouth. Initiate intravenous fluid resuscitation and administer intravenous opioid analgesia. Broad-spectrum antibiotics, such as **cefoxitin** 2 grams IV or **piperacillin/tazobactam** 3.375 grams IV, are advised if there is evidence of perforation or strangulation.
4. Infants with successfully reduced inguinal hernias should have surgical repair within 24 to 72 hours because one-third will redevelop incarceration.
5. Children with uncomplicated umbilical hernias may be discharged and followed longitudinally by their primary care providers. Refer children older than 4 years or those with hernias greater than 2 cm in diameter for surgical evaluation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 87, "Hernias in Adults," by Donald Byars.

# **CHAPTER 47**

## **Anorectal Disorders**

Chad E. Branecki

Anorectal disorders may be due to local disease processes or underlying serious systemic disorders. Most anorectal diseases originate in the anal crypts, glands, internal hemorrhoidal plexus, and external hemorrhoid veins. More serious life-threatening infections tend to lie in the deeper tissues such as the ischiorectal and pelvirectal spaces.

After a detailed history, a digital examination of the rectum should be performed, followed by anoscopy in the left lateral decubitus position. The supine or lithotomy position should be used for debilitated patients.

### **ANAL TAGS**

Skin tags are usually asymptomatic minor projections of the skin at the anal verge, which may be from residual prior hemorrhoids. Most are asymptomatic but inflammation may cause itching or pain.

### **HEMORRHOIDS**

Engorgement, prolapse, or thrombosis of the internal or external hemorrhoidal vein(s) is termed hemorrhoids.

#### **Diagnosis and Differential**

Internal hemorrhoids are not readily palpable and are best visualized through an anoscope. They are found at 2, 5, and 9 o'clock positions when patients are prone. Constipation, pregnancy, ascites, ovarian tumors, radiation fibrosis, and increased portal venous pressure are some of the

common causes of hemorrhoids. Rectal and sigmoid colon tumors should be considered in patients older than 40 years.

## **Clinical Features**

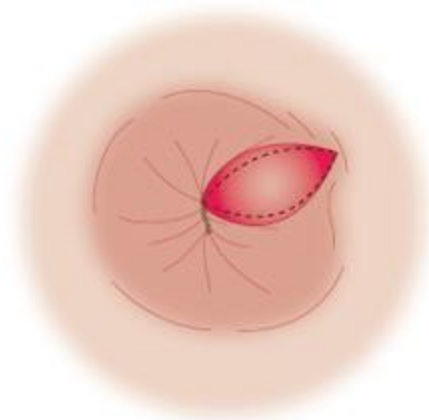
Patients report painless, bright red rectal blood on the surface of the stool, toilet tissue or dripping into the toilet bowl after defecation. Thrombosed hemorrhoids are usually painful and may appear as a bluish-purple mass protruding from the rectum. Large hemorrhoids may result in prolapse that may spontaneously reduce or require periodic manual reduction by patients or clinicians. They may become incarcerated and gangrenous, and require surgical intervention. Prolapse may cause mucous discharge and pruritus. If not reduced, severe bleeding, thrombosis, infarction, incarceration, urinary retention, or sepsis may occur.

## **Emergency Department Care and Disposition**

Unless a complication is present, management is usually nonsurgical.

1. Hot sitz baths for at least 15 min, 3 times per day, and after each bowel movement will reduce pain and swelling. After the sitz baths, the anus should be gently but thoroughly dried.
2. Topical steroids and analgesics may provide temporary relief. Bulk laxatives, such as psyllium seed compounds or stool softeners, should be used after the acute phase has subsided. Laxatives causing liquid stool are contraindicated as they may result in cryptitis and sepsis.
3. Surgical treatment is indicated for severe, intractable pain, continued bleeding, incarceration, or strangulation.
4. Acute and recently thrombosed painful hemorrhoids (<48 hours) can be treated with clot excision. After analgesia, with a long acting local anesthetic such as 0.5% bupivacaine with epinephrine, an elliptical skin incision is made over the hemorrhoids and the thrombosed clot is evacuated. ([Fig. 47-1](#)). Hemostasis is achieved by packing and pressure dressing. The pressure dressing may be removed after about 6 hours,

when the patient takes the first sitz bath. Refer for definitive hemorrhoidectomy.



**FIGURE 47-1.** Elliptical incision of thrombosed external hemorrhoid.

## **CRYPTITIS**

Sphincter spasm and superficial trauma from diarrhea, or repeated passage of large hard stools cause breakdown of the mucosa over the crypts. Infecting organisms enter the crypts and cause inflammation of anal glands, abscess formation, fissures, and fistulae. Common symptoms include anal pain during bowel movements and itching, with or without rectal bleeding. Diagnosis is made by palpation of tender, swollen crypts with associated hypertrophied papillae. Anoscopy allows visualization of the inflamed crypts in posterior midline of the anal ring. Bulk laxatives, additional roughage, hot sitz baths, and warm rectal irrigations enhance healing. Surgical treatment may be needed in refractory cases.

## **ANAL FISSURES**

Anal fissures are superficial linear tears of the anal canal usually caused by local trauma (eg, passage of hard stool), and are the most common cause of

painful rectal bleeding.

## Clinical Features

Patients complain of sharp cutting pain with defecation that subsides between bowel movements. Bleeding is bright and in small quantities. Rectal examination is very painful and often not possible without application of topical anesthetic agents. Most fissures are located in the posterior midline. A sentinel pile may be noted in patients with chronic fissures. A nonposterior midline fissure should alert the physician to consider serious causes, such as Crohn disease, ulcerative colitis, carcinomas, AIDS, tuberculosis, and sexually transmitted diseases.

Treatment is aimed at relieving sphincter spasm and pain, and preventing stricture formation. Hot sitz baths and the addition of bran (fiber) to the diet are helpful. Use of topical analgesics or steroids may be temporarily helpful. Surgical excision of the fissure may be required if the area does not heal after adequate treatment.

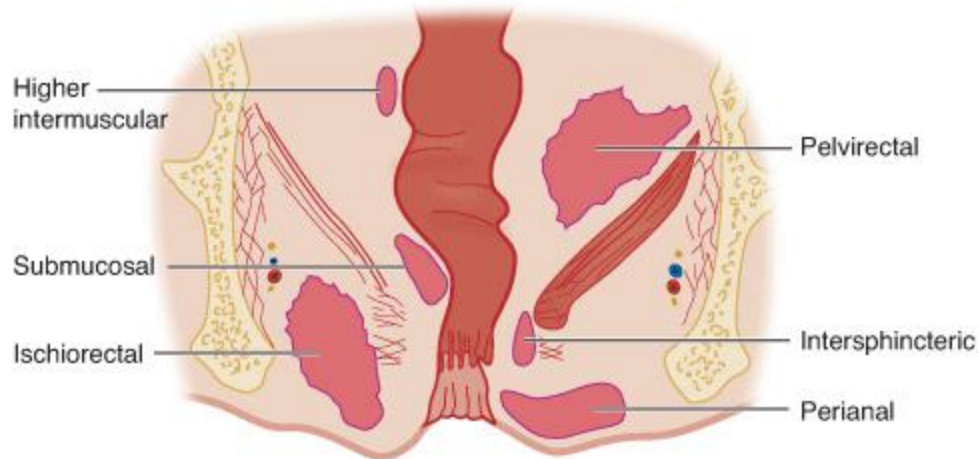
## FISTULA IN ANO

An anal fistula is an abnormal inflammatory tract, originating from an infected anal gland. Fistulae commonly result from perianal or ischiorectal abscess. Crohn disease, ulcerative colitis, tuberculosis, gonococcal proctitis, and carcinomas should also be considered in the etiology. Persistent bloody, malodorous discharge occurs as long as the fistula remains open. Blockage of the tract causes recurrent bouts of inflammation and abscess formation.

Ultrasonography with a 7 MHz endoprobe and enhanced with 3% hydrogen peroxide may aid in the diagnosis. Non-ill appearing patients can be treated with analgesics, antipyretics, and oral antibiotics such as **ciprofloxacin** 750 milligrams twice daily and **metronidazole** 500 milligrams four times daily × 7 days. Surgical excision is the definitive treatment and should not be delayed in ill appearing patients. Sitz baths and local cleaning will temporize the condition before surgery.

# ANORECTAL ABSCESSSES

Abscesses start from the anal crypts and spread to involve the perianal, intersphincteric, ischiorectal, or deep perianal space. Perianal abscess is the most common and found at the anal verge ([Fig. 47-2](#)).



**FIGURE 47-2.** Anatomic classification of common anorectal abscesses.

## Clinical Features

Persistent dull, aching, throbbing pain that increases prior to defecation is typical. As the abscess progresses, pain and tenderness interfere with walking or sitting. Fever, leukocytosis, and a painful tender mass may be present upon digital rectal examination.

## Emergency Department Care and Disposition

Simple perianal abscess without systemic illness is may be safely incised in the ED. Other perirectal abscesses, such as supralevator or ischiorectal abscesses, should be drained in the operating room. After adequate local



and systemic analgesia, a cruciate incision is made over the abscess, and the “dog ears” are excised. Packing usually is not required. Sitz baths should be started the next day. Antibiotics usually are not necessary unless systemic infection or toxicity is present.

## **PROCTITIS**

Proctitis is inflammation of the rectal mucosa. Common causes include prior radiation treatments, autoimmune disorders, vasculitis, ischemia, sexually transmitted infections (eg, syphilis, gonorrhea, chlamydia, lymphogranuloma venereum, herpes simplex, chancroid, human papillomavirus), and other infectious diseases.

### **Clinical Features**

Symptoms include anorectal pain, itching, discharge, diarrhea, bleeding, and lower abdominal cramping. Mucosal inflammation, erythema, bleeding, ulcers, or discharge may be noted using anoscopy.

### **Emergency Department Care and Disposition**

Obtain cultures if an infectious cause is suspected. Stool softeners, sitz baths, good anal hygiene, and analgesics will provide some relief. Patient with enteric pathogens and sexually transmitted infections will require antibiotics directed at the suspected underlying pathogen. Arrange outpatient follow-up.

## **RECTAL PROLAPSE**

Prolapse (procidentia) may involve the mucosa alone or all layers of the rectum. In addition, intussusception of the rectum may present as a prolapse.

## **Clinical Features**

Most patients complain of protruding mass, mucous discharge, associated bleeding, and pruritus. Partial prolapse involves only the rectal mucosa and tends to protrude only a few centimeters from the dentate line. Complete prolapse involves all layers of the rectum, and appears like a red, ball-like mass and may extend up to 15 cm.

## **Emergency Department Care and Disposition**

In children, the prolapse can be gently reduced under proper analgesia and sedation. The child should then be referred to a specialist to ensure the prolapse is not due to an underlying condition. Every effort should be made to prevent the child from being constipated. In adults, reduction can be more difficult if the rectal walls have become edematous. Generous amounts of granulated sugar applied 15 min prior to the reduction may aid in the process. Lubricated gauze should be taped in place over the anal verge for a few hours after reduction. If the prolapse cannot be easily reduced, or there is evidence of ischemia, emergency surgical consultation and hospitalization are warranted.

## **ANORECTAL TUMORS**

Factors such as smoking, anal intercourse, HIV, and genital warts are associated with anorectal cancer. Neoplasms that occur in this group include adenocarcinoma, malignant melanoma, and Kaposi sarcoma. Patients present with nonspecific symptoms including sensation of a mass, pruritus, pain, and blood on the stool. Constipation, anorexia, weight loss, narrowing of the stool caliber, and tenesmus eventually develop. Anal margin neoplasms frequently present as an ulcer that fails to heal in a timely manner. Virtually all anorectal tumors can be detected by careful visual examination of the perianal area, digital palpation of the distal rectum and

anal canal, and procto- or sigmoidoscopic examination. Complications of anorectal tumors include rectal prolapse, prolonged blood loss, perirectal abscesses, or fistulae. Refer all patients for proctoscopic or sigmoidoscopic examination and biopsy if the history or physical examination is suspicious for neoplasms.

## **RECTAL FOREIGN BODIES**

Not all patients are forthcoming with accurate history of rectal foreign body insertion. Patients may instead complain of abdominal pain, anorectal bleeding, or discharge.

### **Clinical Features**

Most foreign bodies are in the rectal ampulla and are palpable through digital and proctoscopic examination. Obtain abdominal and pelvis x-rays to demonstrate the position, shape, number of foreign bodies. An upright film or CT scan may be useful to detect free air, indicative of perforation.

### **Emergency Department Care and Disposition**

Although some low lying rectal foreign bodies can be removed in the ED, many require surgical consultation and intervention, especially if they are made of glass or contain sharp edges.

1. In the ED, procedural sedation accompanied by perianal and submucosal analgesia is used. Adequate sphincter relaxation is essential. Local infiltration anesthesia injected through a 30-gauge needle into the internal sphincter muscle circumferentially will provide good relaxation. Anal lubrication, the aid of obstetric forceps, a speculum or snares, and having the patient bear down may all be helpful in the extraction of the foreign body.

2. Large bulbar objects may create a vacuum-like effect proximally, making removal by simple traction impossible. In these cases, the vacuum can be overcome by passing a catheter around the foreign body into the ampulla and injecting air.
3. Occasionally, passing a Foley catheter proximal to the foreign body, inflating the balloon, and applying gentle traction may help maneuver the foreign body into a more desirable position for ease of removal.
4. Reevaluate the anus and rectum after foreign body removal for lacerations and perforation.

## **PRURITUS ANI**

Pruritus ani can occur from a variety of anal and systemic problems. Common causes include diet, infectious agents, irritants, and tight-fitting undergarments. Pinworms (*Enterobius vermicularis*) are the most common cause of anal pruritus in children. The skin appears normal in early mild cases. In severe exacerbations the perianal area will appear reddened, excoriated, and moist. Increased fiber, sitz baths, antihistamines, **zinc oxide ointment** or **1% hydrocortisone cream** can be used to treat acute symptoms and enhance healing. Any underlying cause should also be treated. Consider referral to proctologist or dermatologist for resistant cases.

## **PILONIDAL SINUS**

Pilonidal sinuses or cysts occur in the midline in the upper part of the natal cleft, which overlies the lower sacrum and coccyx. A pilonidal sinus is usually caused by foreign body granuloma reaction to ingrowing hair. Because of their proximity to the anus, infected pilonidal cysts (abscesses) are sometimes mistakenly diagnosed as perirectal abscesses.

### **Clinical Features**

Pilonidal disease may present as a painless cyst, an infected abscess, or a chronic recurring cyst with drainage. Ultrasound can be used to determine the extent of the abscess before incision and drainage.

## **Emergency Department Care and Disposition**

Acute infections may be drained in the ED and packed. The patient is placed prone and the buttocks are retracted. After appropriate sedation and local anesthesia, the abscess is drained and loculations are gently broken. The wound is packed loosely with gauze, and a bulky dressing is applied. The patient is advised to start sitz baths the following day. Antibiotics or cultures usually are not necessary, unless the patient is immunocompromised or there is evidence of surrounding cellulitis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 88, "Anorectal Disorders," by Brian E. Burgess

## **CHAPTER 48**

# **Jaundice, Hepatic Disorders, and Hepatic Failure**

Joshua Gentges

## **JAUNDICE**

Jaundice, a yellowish discoloration of the skin, sclerae, and mucous membranes, results from hyperbilirubinemia (breakdown of hemoglobin) and the deposition of bile pigments. Etiologies include disorders of bilirubin metabolism (eg, hemolysis) and hepatocellular causes due to infections, drugs and toxins, metabolic disease, granulomatous disease, and bile duct obstruction. Hyperbilirubinemia can be divided into 2 types. The unconjugated form results from increased bilirubin production or a liver defect in its uptake or conjugation. The conjugated form occurs in the setting of intra- or extrahepatic cholestasis, resulting in decreased excretion of conjugated bilirubin.

### **Clinical Features**

Sudden onset of jaundice in a previously healthy young person and a prodrome of fever, malaise, myalgias, and a tender enlarged liver point to hepatitis (probably viral) as a likely cause. Heavy ethanol use suggests alcoholic hepatitis. In the setting of alcoholic liver disease and cirrhosis, jaundice usually develops gradually. A family history of jaundice or a history of recurrent mild jaundice that spontaneously resolves usually accompanies inherited causes of jaundice such as Gilbert syndrome. Cholecystitis may not cause jaundice unless there is acute biliary obstruction present, such as with a retained common bile duct gallstone. Painless jaundice in an older patient classically suggests pancreatic or hepatobiliary malignancy. Patients with a known prior malignancy and a

hard, nodular liver accompanied by jaundice are likely to be found to have liver metastases. Biliary tract scarring or strictures always must be suspected as a cause of jaundice in patients with a prior history of biliary tract surgery, pancreatitis, cholangitis, or inflammatory bowel disease. Hepatomegaly with jaundice, accompanied by pedal edema, jugular venous distention, and a gallop rhythm suggest chronic heart failure.

## **Diagnosis and Differential**

Initial laboratory tests that should be obtained in the workup of a jaundiced patient include serum bilirubin level (total and direct fractions; indirect fraction can be deduced by simple subtraction), serum aminotransferases and alkaline phosphatase levels, urinalysis to check for bilirubin and urobilinogen, and a complete blood count (CBC). Additional laboratory tests may be indicated based on the clinical setting (serum amylase and lipase levels, prothrombin time [PT], INR, electrolytes and glucose levels, blood urea nitrogen [BUN] and creatinine levels, viral hepatitis panels, drug levels, and pregnancy test). With normal liver enzyme levels, the jaundice is more likely to be caused by sepsis or systemic infection, inborn errors of metabolism, or pregnancy, rather than by primary hepatic disease. With abnormally elevated liver enzymes, the pattern of abnormalities may suggest the etiology. Aminotransferase elevation, if predominant, suggests hepatocellular diseases such as viral or toxic hepatitis or cirrhosis, whereas markedly elevated alkaline phosphatase levels (2 to 3 times that of normal levels) and gamma-glutamyl transferase (GGT) points to intra- or extrahepatic obstruction (gallstones, stricture, or malignancy). A Coombs test and hemoglobin electrophoresis may be useful if anemia is present in addition to normal liver aminotransferase levels (hemolysis and hemoglobinopathy). If clinical features and initial laboratory results indicate conjugated hyperbilirubinemia, ultrasound studies of the biliary tract, liver, and pancreas should be performed to rule out gallstones, dilated extrahepatic biliary ducts, or mass or tumor in the liver, pancreas, and portal region. Computed tomography is more costly and not as sensitive as ultrasound for detection of gallstones.

## **Emergency Department Care and Disposition**

1. In some situations, discharge from the emergency department pending further outpatient workup may be appropriate: if a patient is hemodynamically stable with new onset jaundice and has no evidence of liver failure or acute biliary obstruction, and if appropriate laboratory studies have been ordered, timely follow-up is available, and the patient is reliable and has adequate social support.
2. If extrahepatic biliary obstruction is suspected, surgical consultation should be obtained in the emergency department.

## **HEPATITIS**

Hepatitis is an inflammation of the liver stemming from toxic, metabolic, or infectious insult. Patients can present to the emergency department anywhere along the spectrum of disease from asymptomatic infection to fulminant liver failure to chronic cirrhosis.

## **ACUTE HEPATITIS**

### **Clinical Features**

Acute hepatitis should be considered in patients with right upper quadrant or epigastric abdominal pain, nausea, vomiting, diarrhea, jaundice, or pruritis. The presence of altered mental status, abnormal bruising, or bleeding suggest fulminant hepatic failure or a chronic process. Patients with cholestasis may notice pale stools or dark urine. Historical clues and risk factors which may help determine the etiology of hepatitis include ingestion of wild mushrooms, raw seafood, herbal remedies, acute and chronic use of medications, ethanol abuse, IV drug use, unprotected sexual activity, positive HIV status, and travel to countries with endemic parasitic or spirochetal liver disease. Hepatitis A (HAV) is transmitted predominantly



by the fecal-oral route, most commonly from improper food handling or from asymptomatic children to adults. Hepatitis B (HBV) is acquired sexually, by transfusion, or by IV drug use. Hepatitis C (HCV) transmission occurs via exposure to contaminated blood or blood products. Both hepatitis B and C may lead to chronic infection and cirrhosis over 10 to 20 years. Other viral diseases, including acute HIV infection, can cause acute hepatitis. Toxic insults from medications may cause hepatocellular necrosis (acetaminophen, phenytoin, statins, INH), cholestasis (oral contraceptives, anabolic steroids), steatohepatitis (valproic acid, amiodarone), as well as chronic disease (nitrofurantoin, minocycline). Acetaminophen and toxic mushroom ingestions are important causes of acute hepatitis and liver failure. (See [Chapter 106](#) “Analgesics,” [Chapter 128](#) “Poisonous Plants and Mushrooms”).

A prodrome of nausea, vomiting, malaise and fever followed by liver enlargement, abdominal pain and jaundice suggests acute viral hepatitis. Viral hepatitis may range in severity from asymptomatic infection to fulminant hepatic failure to chronic cirrhosis. A few days of generalized pruritus and dark urine may precede the onset of gastrointestinal (GI) symptoms and jaundice.

Patients with previously undiagnosed mild alcohol-induced hepatitis may complain of gradual onset of anorexia, nausea, fever, dark urine, jaundice, weight loss, abdominal pain, and generalized weakness. Physical examination demonstrates a tender enlarged liver, low-grade fever, and icteric mucous membranes, sclera, or skin. Alcoholic liver disease can range from asymptomatic hepatic steatosis (fatty liver) to alcoholic hepatitis to alcoholic cirrhosis.

Fulminant hepatic failure, defined as acute hepatocellular necrosis with rapid development of encephalopathy and liver failure in <8 weeks, is rare. Patients present with encephalopathy, coagulopathy, and rapidly worsening jaundice.

[Table 48-1](#) provides a summary of key clinical features associated with acute and chronic hepatitis.

Diagnosis	Acute Hepatitis (hepatitis A virus, HBV, HCV, toxic)	Chronic Hepatitis/ Cirrhosis (HBV, HCV, alcohol liver disease)	Liver Failure (end-stage HBV/ HCV, toxic)
Nausea/vomiting	+	+	+
Fever	+	-	-
Pain	+	+	±
Altered mental status	-	+	+
Bruising/bleeding	-	+	+
Physical examination			
Jaundice	+	+	+
Hepatomegaly	+	-	±
Ascites	-	+	+
Edema	-	+	+
Spider nevus	-	+	±
Lab abnormalities			
Elevated ALT/AST	+	+	±
AST/ALT >2	+	±	±
Elevated prothrombin time/ international normalized ratio	-	±	+
Elevated ammonia	-	±	+
Low albumin	-	+	+
Direct bilirubinemia	-	+	±
Indirect bilirubinemia	+	+	±
Urobilinogen	+	+	+
Elevated blood urea nitro- gen/creatinine	-	-	±
Radiologic findings			
Ascites	-	±	+
Fatty liver	+	-	-
Cirrhosis	-	+	+

Key: ALT = alanine aminotransferase, AST = aspartate aminotransferase, HBV = hepatitis B virus, HCV = hepatitis C virus, + = typically present, - = typically absent, ± = variable.

**TABLE 48-1** Clinical Features of Hepatitis

## **Diagnosis and Differential**

Serum transaminase levels (GGT, aspartate aminotransferase [AST], and alanine aminotransferase [ALT]) should be checked because elevations are suggestive of hepatitis. Values in the hundreds of units per liter are consistent with viral inflammation, but elevations into the thousands suggest hepatocellular necrosis, extensive liver injury, and more fulminant disease. In acute and chronic viral hepatitis, the ratio of AST to ALT is usually less than 1, whereas a ratio greater than 2 is more suggestive of alcoholic hepatitis. Serum alkaline phosphatase level also should be determined; if elevated more than 3-fold above normal, cholestasis should be suspected (a concurrently elevated GGT supports this suspicion). Total serum bilirubin level and its direct fraction also may be useful because a conjugated (direct) fraction of 30% or higher is consistent with viral hepatitis. The magnitude of transaminase elevation is not a reliable marker of disease severity, but a persistent total bilirubin level above 20 milligrams/dL or a PT prolonged by more than a few seconds or elevated INR indicates significant liver dysfunction and a poor prognosis. Serum electrolytes, BUN, and creatinine levels should be checked if there is clinical suspicion of volume depletion or electrolyte abnormalities. Abnormal mental status should prompt an immediate determination of serum glucose level, which may be low due to poor oral intake or hepatic failure. Other causes of abnormal mental status such as hypoxia, sepsis, intoxication, structural intracranial process, or encephalopathy must be considered. A CBC may be useful because an early transient neutropenia followed by a relative lymphocytosis with atypical forms is often seen with viral hepatitis. Anemia, if present, may be more suggestive of alcoholic hepatitis, decompensated cirrhosis, GI bleeding, or a hemolytic process. Serologic studies to determine the specific viral agent responsible may be ordered in the emergency department to facilitate the final diagnosis, but these results are rarely immediately available. Acetaminophen levels should be checked if concern of toxic ingestion exists. The differential diagnosis includes viral hepatitis, alcohol- or toxin-induced hepatitis, medication

effects, infectious mononucleosis, cholecystitis, ascending cholangitis, sarcoidosis, lymphoma, liver metastases, and pancreatic or biliary tumors.

## **Emergency Department Care and Disposition**

1. With the exception of acetaminophen toxicity, treatment for acute hepatitis is supportive.
2. Most patients with acute viral hepatitis can be managed successfully as outpatients with emphasis on rest, adequate oral intake, strict personal hygiene, and avoidance of hepatotoxins (ethanol and drugs). Patients should be instructed to return for worsening symptoms, in particular vomiting, fever, jaundice, or abdominal pain. Follow-up arrangements should be made.
3. Patients with mild alcohol-induced hepatitis may be managed as outpatients with emphasis on nutritional supplementation, including thiamine, folate, magnesium, and potassium supplements, adequate oral intake, and strict avoidance of alcohol and other hepatotoxins. Patients should be instructed to return for worsening symptoms, in particular vomiting, fever, jaundice, or abdominal pain. Follow-up arrangements should be made. Patients who require admission should also be given prophylactic treatment for alcohol withdrawal.
4. Patients with any of the following should be admitted to the hospital: encephalopathy, PT prolonged by more than a few seconds, elevated INR, intractable vomiting, hypoglycemia, bilirubin level above 20 milligrams/dL, age older than 45 years, pregnancy, immunosuppression, or suspected toxin-induced hepatitis.
5. Correct volume depletion and electrolyte imbalances with IV crystalloid. Hypoglycemia should be treated initially with 1 ampule of 50% dextrose in water IV followed by the addition of dextrose to IV fluids and careful monitoring.
6. Admit patients with fulminant hepatic failure to the intensive care unit, with aggressive support of circulation and respiration, monitoring and treatment of increased intracranial pressure if present, correction of hypoglycemia and coagulopathy, administration of oral lactulose or neomycin, and a protein-restricted diet (see the following section on

treatment of cirrhosis). Consult with a hepatologist and liver transplant service.

## **CIRRHOSIS/CHRONIC LIVER FAILURE**

Cirrhosis is often caused by ethanol or chronic viral hepatitis; less common causes include drugs or toxins, hemochromatosis, Wilson disease, and primary (idiopathic) biliary cirrhosis.

### **Clinical Features**

Patients with cirrhosis generally report a gradual deterioration in their health, with anorexia, muscle loss (often masked by edema or ascites), fatigue, nausea, emesis, diarrhea, and increasing abdominal girth (ascites). Low-grade intermittent or continuous fever also may be present. Physical examination findings include jaundice, ascites, a small firm liver, splenomegaly, pedal edema, and spider angiomas. Hepatic encephalopathy, characterized by a fluctuating level of consciousness and confusion and, possibly, hyperreflexia, spasticity, generalized seizures, and coma also may be present. Asterixis (“liver flap”) is characteristic but not specific for encephalopathy due to liver failure. Patients with cirrhosis often come to the emergency department because of worsening ascites or edema, complications such as spontaneous bacterial peritonitis (abdominal pain), encephalopathy, GI or variceal bleeding (see [Chapter 39](#) “Gastrointestinal Bleeding”), and various concurrent infections (urinary tract infection, pneumonia, etc).

Spontaneous bacterial peritonitis (SBP), the most common complication of cirrhotic ascites, should be suspected in any cirrhotic patient with fever, abdominal pain or tenderness, worsening ascites, sub-acute functional decline, or encephalopathy. Other subtle clues to SBP include deteriorating renal function, hypothermia, and diarrhea.

Hepatic encephalopathy may be worsened or precipitated by a large protein load, occult GI bleed, infection, electrolyte imbalance, renal failure, and medications. Hepatic encephalopathy is a diagnosis of exclusion. In the

cirrhotic patient presenting with altered mental status or lethargy, multiple other causes must first be ruled out.

Hepatorenal syndrome, a refractory form of acute renal failure that occurs in cirrhotic patients, may develop in the setting of sepsis, acute dehydration, overzealous diuresis, or high-volume paracentesis.

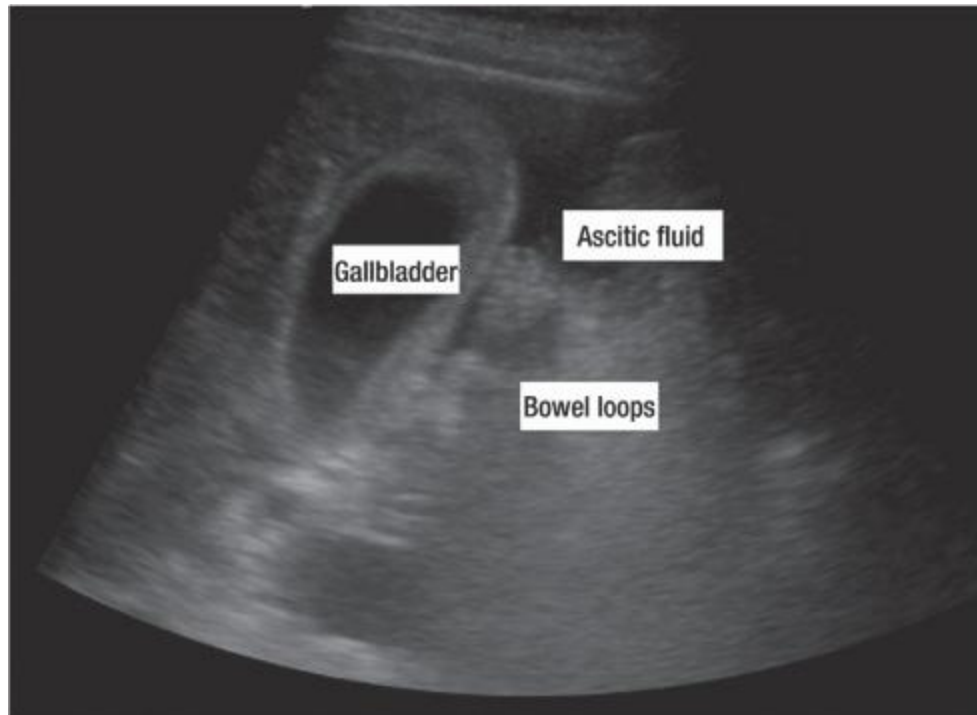
## Diagnosis and Differential

Laboratory studies include serum transaminases (ALT and AST), serum alkaline phosphatase, total and direct bilirubin, serum albumin, serum glucose and electrolytes, ammonia, BUN and creatinine, CBC, and PT/INR. In advanced cirrhosis, transaminase and bilirubin levels may be mildly elevated or normal. Serum albumin is usually low and PT/INR are elevated indicating significant hepatic dysfunction. Elevated serum ammonia suggests hepatic encephalopathy, although (as in acute liver disease) levels do not correlate with cause and hyperammonemia does not obviate a thorough search for other causes of altered mental status. If concurrent pancreatitis is suspected, serum lipase and amylase levels should be checked. Patients with fever with or without leukocytosis should be evaluated for infection.

Patients diagnosed with ascites for the first time or those with ascites who develop fever, abdominal pain, GI bleeding or encephalopathy should undergo ultrasound guided paracentesis to check for bacterial peritonitis. Ascitic fluid should be tested for total protein and glucose level, lactate dehydrogenase, Gram stain, and white blood cell count (WBC) with differential. A total WBC greater than  $1000/\text{mm}^3$  or neutrophil count greater than  $250/\text{mm}^3$  is diagnostic for SBP. Culture results from ascitic fluid are often negative, but placing 10 mL ascitic fluid in a blood culture bottle may improve the yield. Gram-negative Enterobacteriaceae (*Escherichia coli*, *Klebsiella*, etc) account for 63% of SBP cases, followed by pneumococcus (15%) and the enterococci (6% to 10%).

Ultrasound can also identify infectious or mass lesions, and hepatic and portal thrombosis ([Fig. 48-1](#)). Abdominal CT may also help elucidate

structural problems. Consider head CT in patients with mental status changes.



**FIGURE 48-1.** Sonographic image of ascitic fluid showing bowel loops and an edematous gallbladder wall. (Courtesy of and used with permission of Michael S. Antonis, DO, RDMS, MedStar Health.)

## **Emergency Department Care and Disposition**

1. Patients with abdominal pain, fever, acidosis, leukocytosis, significant hypo or hypervolemia, new onset or worsening encephalopathy, coagulopathy with bleeding, or significant electrolyte abnormalities should be admitted to the hospital. Hepatorenal syndrome warrants nephrology consultation.
2. Patients with mild to moderate volume ascites and no sign of infection or other complication can sometimes be managed on an outpatient basis. Recommended diuretics for management of ascites include

**spironolactone**, 50 to 200 milligrams/d, and **amiloride**, 5 to 10 milligrams/d. Abstinence from alcohol and other hepatotoxins is essential for outpatient management. A protein-restricted diet helps prevent the complication of hepatic encephalopathy. Outpatient management and medication changes must be coordinated with the patient's follow-up physician.

3. Paracentesis is necessary for symptomatic relief of ascites or to diagnose SBP. Administer **albumin**, 1.5 grams/kilogram IV before paracentesis, to guard against complications related to fluid shifts. Removal of more than 1 L of ascitic fluid can lead to hypotension, so careful monitoring is required.
4. Initiate antibiotics in patients with SBP. **Cefotaxime** 2 grams IV every 8 hours (double the dose for critical cases), or **piperacillin-tazobactam** 3.375 grams IV every 6 hours, or **ampicillin-sulbactam** 3 grams IV every 6 hours, or **ticarcillin-clavulanate** 3.1 grams IV every 6 hours, or **ceftriaxone** 2 grams IV every 24 hours are acceptable choices.
5. The mainstay of therapy for hepatic encephalopathy is **lactulose**, 20 grams PO or 300 mL of the syrup diluted with 700 mL of water or normal saline as a 30 min retention enema. Patients should be placed on a protein-restricted diet.
6. Suspect gastroesophageal variceal bleeding in patients with hematemesis, melena, or hematochezia. Variceal bleeding is discussed in [Chapter 39](#) "Gastrointestinal Bleeding".
7. Correct coagulopathy in patients who are bleeding or are scheduled for a procedure: give **vitamin K**, 10 milligrams PO or IV. Fresh frozen plasma can be given. Replete platelets with pooled donor platelets.
8. Aggressive treatment of comorbidities, including alcohol related syndromes (withdrawal, ketoacidosis, Wernicke-Korsakoff syndrome), sepsis, ventilatory and circulatory dysfunction, electrolyte abnormalities, and hypoglycemia is essential.
9. Admit all patients with acute hepatic failure (prolonged PT, hypoglycemia, coagulopathy, encephalopathy, marked jaundice) to the intensive care unit. Aggressive support is required. Consult with a hepatologist and transplant team.



For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 83, “Hepatic Disorders, Jaundice, and Hepatic Failure,” by Susan R. O’Mara and Kulleni Gebreyes.

The author gratefully acknowledges the contribution of Gregory S. Hall, author of “Jaundice, Hepatic Disorders and Hepatic Failure” in *Emergency Medicine Manual*, 6 th ed.

## **CHAPTER 49**

# **Complications of General Surgical Procedures**

Daniel J. Egan

As surgical procedures take place more commonly in outpatient settings and inpatient lengths of stay decrease, the emergency physician will encounter an increasing number of postoperative patients and their complications. Common clinical situations presenting to the emergency department include: fever, respiratory complications, genitourinary complaints, wound infections, vascular problems, and complications of drug therapy. Specific problems not covered in other chapters of this book are discussed here.

## **CLINICAL FEATURES**

### **Fever**

The causes of postoperative fever are the 5 Ws: wind (respiratory), water (urinary tract infection [UTI]), wound, walking (deep venous thrombosis [DVT]), and wonder drugs (drug fever or pseudomembranous colitis [PMC]). Fever in the first 24 hours is usually due to atelectasis, but wound infections with necrotizing fasciitis, or clostridial infections must also be considered. In the first 72 hours, pneumonia, atelectasis, intravenous catheter-related thrombophlebitis, and infections are the major causes. UTIs are seen 3 to 5 days postoperatively. DVT does not typically occur until 5 days after the procedure, and wound infections generally manifest 7 to 10 days after surgery. Antibiotic-induced PMC is seen 6 weeks after surgery.

### **Respiratory Complications**

Postoperative pain, splinting, and inadequate clearance of secretions contribute to the development of atelectasis. Fever, tachypnea, tachycardia,

and mild hypoxia may be seen. Pneumonia may develop 24 to 96 hours later (see [Chapter 30](#)). Pulmonary embolism can occur any time postoperatively (see [Chapter 25](#)).

## **Genitourinary Complications**

UTIs may occur after any procedure, but are more common after instrumentation of the GU tract or bladder catheterization. Elderly men, patients undergoing anorectal surgery and those receiving spinal or epidural anesthesia are at increased risk for urinary retention presenting with lower abdominal pain and the inability to urinate (see [Chapter 54](#)). Decreased urine output should raise concerns for renal failure resulting from multiple causes (see [Chapter 50](#)). Volume depletion is the most common cause.

## **Wound Complications**

Hematomas result from inadequate hemostasis leading to pain and swelling at the surgical site. Careful evaluation, including possibly opening a small portion of the wound, to rule out infection must be undertaken. Seromas are collections of clear fluid under the wound. Wound infections may present with pain swelling, erythema drainage and tenderness. Risk factors include extremes of age, diabetes, poor nutrition, necrotic tissue, poor perfusion, foreign bodies, and hematomas. Necrotizing fasciitis should be considered in a systemically ill patient with rapidly expanding infection (see [Chapter 90](#)). Superficial or deep fascial wound dehiscence can occur due to diabetes, poor nutrition, chronic steroid use, and inadequate or improper closure of the wound. Operative exploration may be required to determine the extent of dehiscence.

## **Vascular Complications**

Superficial thrombophlebitis manifests with erythema, warmth, and fullness of the affected vein. It usually occurs in the upper extremities after

intravenous catheter insertion or in the lower extremities due to stasis in varicose veins. DVT commonly occurs in the lower extremities postoperatively (see [Chapter 25](#)).

## **Drug Therapy Complications**

Numerous medications may lead to fever without any associated concomitant infection. Additionally, many antibiotics prescribed perioperatively can cause antibiotic-induced diarrhea. PMC, the most serious diarrheal complication, is caused by *Clostridium difficile* toxin. Watery or even bloody diarrhea, fever, and crampy abdominal pain are the usual complaints.

## **DIAGNOSIS AND DIFFERENTIAL**

Postoperative patients with fever should have an evaluation focusing on the elements detailed above. Patients with suspected respiratory complications should have chest x-rays. Radiographs may demonstrate atelectasis, pneumonia, or pneumothorax. Additional imaging like CT or ultrasound may be indicated based on the operative procedure performed.

Patients with oliguria or anuria should be evaluated for signs of hypovolemia or urinary retention. Diagnosis of PMC is established by demonstrating *C difficile* cytotoxin in the stool. Nevertheless, in 27% of cases, the assay may be negative.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Contact the surgeon who performed the procedure to discuss patients who present with postoperative complications. Patients who are toxic appearing, have underlying debilitating conditions or elderly require hospitalization.

1. Patients with mild atelectasis and no evidence of hypoxemia may be managed as outpatients with pain control and increased deep breathing.
2. Postoperative pneumonia may be polymicrobial. Admission and antibiotic therapy to cover nosocomial infections such as *Pseudomonas* and methicillin-resistant *Staph aureus* is usually recommended (see [Chapter 30](#)).
3. Nontoxic patients with UTI can be managed as outpatients with oral antibiotic therapy geared toward appropriate organisms. Consider gram-positive flora when instrumentation has occurred. Ill appearing patients require admission.
4. Wound hematomas may require removal of some sutures and evacuation. Consultation with the surgeon before treatment is appropriate. Seromas can be confirmed and treated with needle aspiration. Admission may not be necessary for either of these processes.
5. Wound infections can often be treated with oral antibiotics unless the patient shows signs of systemic toxicity or carries significant comorbidities. Perineal infections are often polymicrobial requiring parenteral antibiotics and admission. Immediate surgical debridement and broadspectrum parenteral antibiotics are indicated for necrotizing fasciitis (see [Chapter 90](#)).
6. Superficial thrombophlebitis is treated as an outpatient with NSAIDs, local heat application and elevation. Antibiotics may be indicated if surrounding cellulitis or lymphangitis are noted. Suppurative thrombophlebitis requires hospitalization and surgical excision.
7. Patients with suspected antibiotic-induced PMC will require fluid resuscitation and likely empiric therapy. Oral or intravenous metronidazole and oral vancomycin are treatments for this condition.

## **SPECIFIC CONSIDERATIONS**

### **Complications of Breast Surgery**

Although overall rates of complications are low following breast surgery, wound infections, hematomas, seromas, pneumothorax, and necrosis of the

skin flaps may be seen. Lymphedema of the ipsilateral arm may occur after mastectomy.

## **Complications of Gastrointestinal Surgery**

Stimulation of the splanchnic nerves during intraabdominal surgery may lead to dysmotility and a paralytic ileus. After gastrointestinal surgery, small bowel tone returns to normal within 24 hours and colonic function within 3 to 5 days.

Patients develop nausea, vomiting, constipation, abdominal distention and pain. An adynamic ileus typically resolves after bowel rest, nasogastric suction and intravenous hydration. Prolonged ileus should prompt an investigation for nonneuronal causes like peritonitis, intra-abdominal abscesses, hemoperitoneum, pneumonia, sepsis, electrolyte imbalance, or medications. Abdominal imaging, complete blood cell count, electrolytes, blood urea nitrogen, creatinine, and urinalysis should be obtained. Occasionally, surgical intervention may be necessary for obstruction due to adhesions.

*Intraabdominal abscesses* are caused by preoperative contamination, intraoperative spillage of bowel contents or postoperative anastomotic leaks. Diagnosis can be confirmed by computed tomography or ultrasonography. Antibiotic therapy as well as either percutaneous or surgical drainage will be required.

*Pancreatitis* occurs especially after direct manipulation of the pancreatic duct. The clinical spectrum extends from mild nausea and vomiting to severe abdominal pain and hemodynamic instability. Complications like pleural effusion and severe hemorrhage may occur. Serum amylase measurements are not specific and measurement of a lipase is more reliable.

*Cholecystitis and biliary colic* have been reported as postoperative complications. Elderly patients are more prone to develop acalculous cholecystitis. Characteristic lab findings of a calculous or obstructive process may be absent.

*Fistulas*, internal or external, may result from either technical complications or direct bowel injury. Fistulas can lead to electrolyte abnormalities and require surgical consultation and possible hospitalization.

*Anastomotic leaks* occur primarily after esophageal, gastric and colonic procedures and can cause devastating consequences as a result of infection. Esophageal leaks occur within 10 days of the operation and carry very high morbidity and mortality.

*Complications of bariatric surgery* remain common although mortality after the procedures is low. In the weeks after surgery, patients are at risk for leaks and bleeding. Dumping syndrome is seen in gastric bypass procedures due to the rapid influx of hyperosmolar chyme into the small intestine resulting in fluid sequestration and hypovolemia. Patients experience nausea, vomiting, epigastric discomfort, palpitations, dizziness, and sometimes syncope. Other complications include gastroesophageal reflux, vitamin and electrolyte deficiencies, ulcers, obstruction, gastric slippage, and band erosion.

*Complications of laparoscopic procedures* include problems related to pneumoperitoneum, traumatic injury from insertion of the needle and trocar, and retained stones after cholecystectomy.

*Complications of transabdominal feeding tubes and percutaneous endoscopic gastrostomy tubes* include infections, hemorrhage, peritonitis, aspiration, wound dehiscence, sepsis, and obstruction of the tube. Dislodged tubes should be replaced with the appropriately sized tube (same type if possible, or a temporary foley catheter).

*Acute complications arising from stomas* (ileostomy or colostomy) are usually due to technical errors of stoma placement. Later complications can be from the underlying disease such as Crohn's disease or cancer. Ischemia, necrosis, skin maceration, bleeding, parastomal hernia, and prolapse may be seen.

The most common *complications of colonoscopy* are hemorrhage and perforation. Hemorrhage occurs typically due to polypectomy, biopsies, or mucosal lacerations or tearing. Perforation may be immediately apparent or symptoms may be delayed for several hours to days. Upright chest or abdominal radiographs may reveal free air but CT should be obtained if the films are unrevealing and suspicion is high.

*Rectal surgery complications* include urinary retention (frequently after hemorrhoidectomy), constipation, prolapse, bleeding, and infections.

*Tetanus* has been known to occur in surgical wounds although, by far, this rare disease is more common after minor trauma.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 90, “Complications of General Surgical Procedures,” by Edmond A. Hooker.



## **SECTION 7 Renal and Genitourinary Disorders**

# **CHAPTER 50**

## **Acute Renal Failure**

Marc D. Squillante

Renal dysfunction and acute renal failure present with a wide variety of manifestations, depending on the underlying etiology. Although the initial symptoms may be those of the primary cause, ultimately patients will develop deterioration of renal function. Renal failure can be caused by hypovolemia from any cause, cardiac disease, vascular or thrombotic disorders, glomerular diseases, diseases affecting the renal tubules, nephrotoxic drugs, and a variety of anatomic problems of the genitourinary tract.

### **CLINICAL FEATURES**

Deterioration in renal function leads to excessive accumulation of nitrogenous waste products in the serum. Patients usually have signs and symptoms of their underlying causative disorder but eventually develop stigmata of renal failure. Volume overload, hypertension, pulmonary edema, mental status changes or neurologic symptoms, nausea and vomiting, bone and joint problems, anemia, and increased susceptibility to infection (a leading cause of death) can occur as patients develop more chronic uremia.

### **DIAGNOSIS AND DIFFERENTIAL**

History and physical examination usually provide clues to etiology. Signs and symptoms of the underlying causative disorder should be vigorously sought. Physical examination should assess vital signs, volume status, establish urinary tract patency and output, and search for signs of chemical intoxication, drug usage, muscle damage, infections, or associated systemic diseases. Diagnostic studies include urinalysis, blood urea nitrogen and

creatinine levels, serum electrolytes, urinary sodium and creatinine, and urinary osmolality. Analysis of these tests allows most patients to be categorized as prerenal, renal, or postrenal. Fractional excretion of sodium can be calculated to help in this categorization ([Table 50-1](#)). Normal urinary sediment may be seen in prerenal and postrenal failure, hemolytic-uremic syndrome, and thrombotic thrombocytopenic purpura. The presence of albumin may indicate glomerulonephritis or malignant hypertension. Granular casts are seen in acute tubular necrosis. Albumin and red blood cell casts are found in glomerulonephritis, malignant hypertension, and autoimmune disease. White blood cell casts are seen in interstitial nephritis and pyelonephritis. Crystals can be present with renal calculi and certain drugs (sulfas, ethylene glycol, and radiocontrast agents). Renal ultrasound is the radiologic procedure of choice in most patients with renal failure when upper tract obstruction and hydronephrosis is suspected. Color flow Doppler can assess renal perfusion and diagnosis large vessel causes of renal failure. Bedside sonography can quickly diagnose some treatable causes and give guidance for fluid resuscitation; inspiratory collapse of the intrahepatic IVC can give a good measure of volume status and fluid responsiveness (see [Fig. 50-1](#)).

Test Employed	Prerenal	Renal*	Postrenal <sup>†</sup>
Urine sodium (mEq/L)	<20	>40	>40
FE <sub>Na</sub> (%) <sup>‡</sup>	<1	>1	>1
RFI <sup>#</sup>	<1	>1	>1
Urine osmolality (mOsm/L)	>500	<350	<350
Urine: serum creatinine	>40:1	<20:1	<20:1
Blood urea nitrogen: creatinine	>20:1	10:1	>10:1

\*FE<sub>Na</sub> may be less than 1 in patients with intrinsic renal failure plus glomerulonephritis, hepatorenal syndrome, radiocontrast acute tubular necrosis, myoglobinuric and hemoglobinuric acute renal failure, renal allograft rejection, and certain drugs (angiotensin-converting enzyme inhibitors and nonsteroidal anti-inflammatory agents).

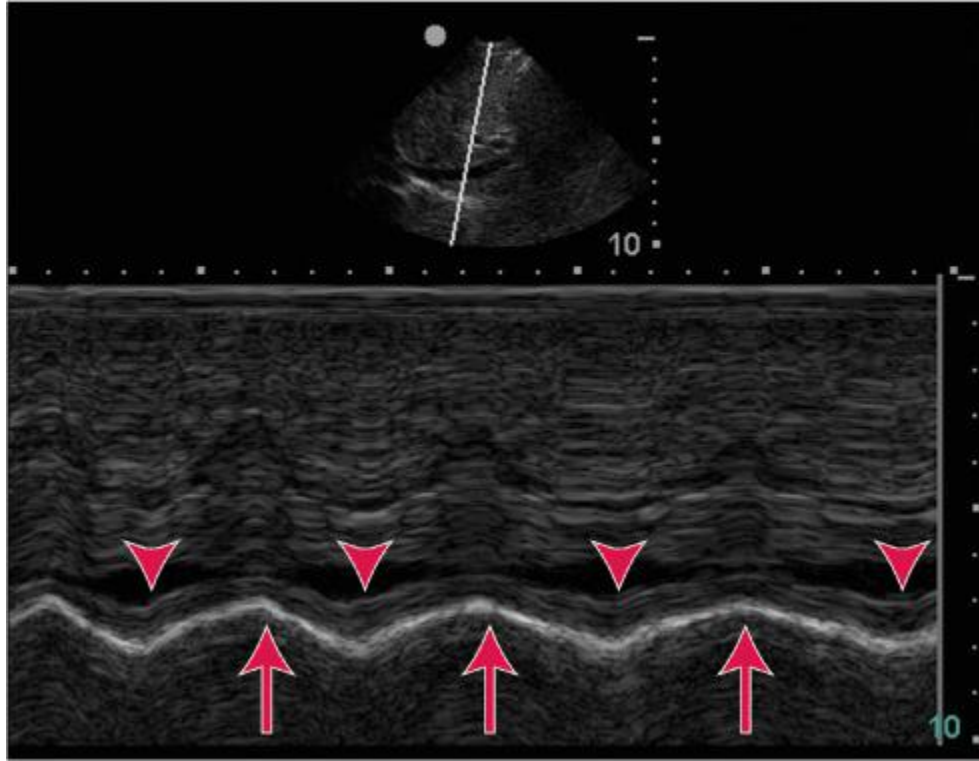
<sup>†</sup>One can see indices similar to prerenal early in the course of obstruction. With continued obstruction, tubular function is impaired and indices mimic those of renal causes.

<sup>‡</sup>FE<sub>Na</sub> = ([urine sodium/serum sodium] + [urine creatinine/serum creatinine]) × 100.

<sup>#</sup>RFI = (serum sodium + [urine creatinine/serum creatinine]) × 100.

Key: FE<sub>Na</sub> = fractional excretion of sodium, RFI = renal failure index.

**TABLE 50-1** Laboratory Studies Aiding in the Differential Diagnosis of Acute



**FIGURE 50-1.** An almost fully collapsed inferior vena cava at inspiration (*arrows*) and expiration (*arrowheads*) as might be expected in prerenal acute renal failure. (Courtesy of Michael B. Stone, MD, RDMS.)

Prerenal failure is produced by conditions that decrease renal perfusion and is the most common cause of community-acquired acute renal failure (70% of cases). It also is a common precursor to ischemic and nephrotoxic causes of intrinsic renal failure. Common causes of prerenal failure include hypovolemic states (vomiting/diarrhea, diuretics and other antihypertensives, reduced intake), fluid sequestration (cirrhosis, pancreatitis, burns, septic shock, others) blood loss, or decreased cardiac output from cardiac dysfunction. ***Intrinsic renal failure*** has vascular and ischemic etiologies; glomerular and tubulointerstitial diseases are also causative. Ischemic renal failure, traditionally known as acute tubular necrosis (ATN), is now called acute kidney injury. ATN, due to severe and prolonged prerenal etiologies, causes most cases of intrinsic renal failure; ATN is also the most common cause of hospital-acquired renal failure.

Nephrotoxins (both physician prescribed and environmental) are the second most common cause of ATN. *Postrenal azotemia* occurs primarily in elderly men with high-grade prostatic obstruction. Lesions of the external genitalia (ie, strictures) are also common causes. Significant permanent loss of renal function occurs over 10 to 14 days with complete obstruction, and worsens with associated UTI. See the parent chapter in Tintinalli's 7th edition for a more complete list.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

ED goals in the initial care of patients with acute renal failure focus on treating the underlying cause and correcting fluid and electrolyte derangements. Efforts should be made to prevent further renal damage and provide supportive care until renal function has recovered (see [Chapter 4](#) for treatment of electrolyte and acid-base disorders).

### **Prerenal Failure**

1. Effective intravascular volume should be restored with isotonic fluids (normal saline or lactated Ringer solution) at a rapid rate in appropriate patients; volume resuscitation is the first priority.
2. If cardiac failure is causing prerenal azotemia, cardiac output should be optimized to improve renal perfusion, and reduction in intravascular volume (ie, with diuretics) may be appropriate.

### **Renal Failure (Intrinsic)**

Adequate circulating volume must be restored first; hypovolemia potentiates and exacerbates all forms of renal failure. Ischemia or nephrotoxic agents are the most common causes of intrinsic renal failure. History, physical examination, and baseline laboratory tests should provide

clues to the diagnosis. Nephrotoxic agents (drugs and intravenous contrast) should be avoided.

1. Low-dose **dopamine** (1 to 5 micrograms/kilogram/min) may improve renal blood flow and urine output, but it does not lower mortality rates or improve recovery.
2. Renally excreted drugs (digoxin, magnesium, sedatives, and narcotics) should be used with caution because therapeutic doses may accumulate to excess and cause serious side effects. Fluid restriction may be required. Interventions useful in the prevention of radiocontrast nephropathy include acetylcysteine, fenoldopam, and crystalloid infusions.

## **Postrenal Failure**

Appropriate urinary drainage should be established; the exact procedure depends on the level of obstruction.

1. A Foley catheter should be placed to relieve obstruction caused by prostatic hypertrophy. There is no support for the practice of intermittent catheter clamping to prevent hypotension and hematuria; urine should be completely and rapidly drained.
2. Percutaneous nephrostomy may be required for ureteral occlusion until definitive surgery to correct the obstruction can take place once the patient is stabilized.
3. For the acutely anuric patient, obstruction is the major consideration. If no urine is obtained on initial bladder catheterization, emergency urologic consultation should be considered.
4. With chronic urinary retention, postobstructive diuresis may occur due to osmotic diuresis or tubular dysfunction. Patients may become suddenly hypovolemic and hypotensive. Urine output must be closely monitored, with appropriate fluid replacement.

## **Dialysis**

If treatment of the underlying cause fails to improve renal function, hemodialysis or peritoneal dialysis should be considered.

1. The nephrology consultant usually makes decisions about dialysis. Dialysis often is initiated when the blood urea nitrogen is greater than 100 milligrams/dL or serum creatinine is greater than 10 milligrams/dL.
2. Patients with complications of acute renal failure such as cardiac instability (due to metabolic acidosis and hyperkalemia), intractable volume overload, hyperkalemia, and uremia (ie, encephalopathy, pericarditis, and bleeding diathesis) not easily corrected by other measures should be considered for emergency dialysis. However, morality in renal failure has changed little since the advent of dialysis.

## **Disposition**

Patients with new onset renal failure usually require hospital admission, often to an intensive care unit. Transferring patients to another institution should be considered if nephrology consultation and dialysis facilities are not available.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 91, "Acute Renal Failure," by Richard Sinert and Peter R. Peacock, Jr.



# **CHAPTER 51**

## **Rhabdomyolysis**

Michael D. Levine

Rhabdomyolysis is a syndrome that involves skeletal muscle injury, necrosis, and release of intracellular contents, including myoglobin and creatinine kinase. If left untreated, complications can occur such as renal failure, compartment syndrome, and peripheral neuropathy.

### **CLINICAL FEATURES**

Obtaining a history which includes risk factors for the development of rhabdomyolysis should increase the suspicion for this syndrome. Importantly, classic signs of rhabdomyolysis may not always be present.

Historical clues to suggest a patient may be at risk for rhabdomyolysis include the following: injuries that can cause compartment syndrome or prolonged muscular compression, including traumatic crush injuries, acutely casted long-bone fractures, heat stroke, electrical injuries, and lightning strikes; prolonged immobilization; drug intoxication with numerous agents including amphetamines, phencyclidine (PCP), cocaine, or antihistamines; excessive muscular activity, seizures, dystonia, or delirium tremens; and diseases such as dermatomyositis, polymyositis, or neuroleptic malignant syndrome. Commonly prescribed medications associated with the development of rhabdomyolysis include antipsychotics, lipid-lowering agents (ie, statins and clofibrate), narcotics, zidovudine, and colchicine.

Classically, patients complain of myalgias, muscle stiffness, malaise, and a low-grade fever. Dark-colored urine often occurs with myoglobinuria. However, these classic findings may be absent in up to 50% of patients with rhabdomyolysis syndromes. Other nonspecific symptoms may include nausea, vomiting, abdominal pain, or palpitations. Signs and symptoms of renal failure can occur as complications from rhabdomyolysis.

The postural muscles of the calves, thighs, and lower back are the most often involved muscle groups, and the involved muscles can be localized or diffuse. The involved muscles are often tender to palpation, but objective swelling may be subtle or absent, especially prior to rehydration.

There are many complications of rhabdomyolysis including acute kidney injury, which can be either oliguric (most commonly), or nonoliguric. Myoglobin breakdown, which occurs in the setting of dehydration and aciduria (pH <5.6), results in exposure of ferriheme, which is nephrotoxic. The risk of acute kidney injury correlates poorly with the total rise in creatine kinase (CK) or amount of myoglobinuria. Additional complications include metabolic derangements including hyperkalemia, hyperuricemia and hypocalcemia, mechanical complications, and occasionally disseminated intravascular coagulation (DIC). Hypercalcemia and hypophosphatemia occur later. Mechanical complications of rhabdomyolysis include both an acute compartment syndrome, as well as peripheral neuropathy due to muscular edema with subsequent nerve compression.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of rhabdomyolysis typically requires a serum CK to be elevated at least 5-fold greater than the upper limit of normal, excluding cardiac or neurologic causes of the elevation. In general, the serum CK will begin to rise 2 to 12 hours after the initial muscle injury, and will peak after 1 to 3 days, in the absence of ongoing injury. The value should decline approximately 39% daily. Myoglobinuria can be detected once plasma myoglobin concentrations exceed 1.5 milligrams/dL. A dark discoloration of the urine or unexplained elevations of lactate dehydrogenase or aminotransferases may be additional clues to the diagnosis.

Myoglobin contains heme. Qualitative tests, such as the urine dipstick, which uses an orthotoluidine reaction, cannot differentiate between hemoglobin, myoglobin, and red blood cells. Thus, the presence of blood on a urine dipstick with only a few or no corresponding red blood cells on microscopy also suggests the diagnosis of rhabdomyolysis.

All patients suspected of having rhabdomyolysis should have a CK, electrolytes, blood urea nitrogen, calcium, and urinalysis obtained. Additional laboratory tests should be obtained based on the clinical scenario.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. The primary focus of therapy should be aggressive intravenous (IV) hydration with crystalloids. The exact recommendations vary, but a rapid correction of fluid deficits, followed by supraphysiologic maintenance fluids should be performed. Some advocate 2.5 cc/kilogram/h of maintenance fluids, while others target a urine output of 200 to 300 cc/h. Urinary alkalinization or forced diuresis have not been clearly proven to improve outcome. Patients with significant comorbidities require close observation and titration of fluids to prevent fluid overload.
2. Electrolytes should be monitored. In general, asymptomatic early hypocalcemia does not require specific therapies, and phosphorus correction should only occur with levels  $> 7$  milligrams/dL or  $< 1$  milligram/dL. Hyperkalemia, in contrast, may require aggressive therapy (see [Chapter 4](#)).
3. Placement of a urinary catheter may be needed in critically ill individuals in order to accurately monitor urine output.
4. The use of nephrotoxic drugs, including nonsteroidal anti-inflammatory (NSAID) medications should be avoided, if possible.
5. Patients without significant comorbidities with mild, exertional rhabdomyolysis can be hydrated in the emergency department, and often released. Those with significant elevations in the CK, those with acute kidney injury, and those with underlying comorbidities should be admitted for continued hydration and evaluation of renal function.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 92, “Rhabdomyolysis,” by Francis L. Counselman

and Bruce M. Lo.

## **CHAPTER 52**

# **Emergencies in Renal Failure and Dialysis Patients**

Jonathan A. Maisel

Patients with end-stage renal disease (ESRD) may sustain multiple complications of their disease process and treatment. Emergent dialysis is most commonly required for hyperkalemia, severe metabolic acidosis, and pulmonary edema resistant to alternative therapy. (See the appropriate chapters for discussion of the management of hypertension, heart failure, bleeding disorders, and electrolyte disorders.)

## **CARDIOVASCULAR COMPLICATIONS**

Creatine protein kinase (and the MB fraction), troponin I, and troponin T are not significantly elevated in ESRD patients undergoing regular dialysis, and have been shown to be specific markers of myocardial ischemia in these patients. Hypertension occurs in 80% to 90% of patients starting dialysis. Management includes control of blood volume, followed by use of adrenergic-blocking drugs, angiotensin-converting enzyme inhibitors, or vasodilating agents. Congestive heart failure (CHF) may be caused by hypertension, coronary ischemia, and valvular disease, as well as uremic cardiomyopathy, fluid overload, and arteriovenous (AV) fistulas (high output failure). Treatment is similar to that in non-ESRD patients. Nitrates are helpful, and possibly furosemide (60 to 100 milligrams) may help, even in oliguric patients, as furosemide causes pulmonary vessel vasodilatation. Preload can be further reduced by inducing diarrhea with sorbitol and with phlebotomy (minimum 150 mL). Blood should be collected in a transfusion bag so that it may be transfused back to the patient during subsequent dialysis. Hemodialysis (HD) is the definitive treatment. Pericarditis in ESRD patients is usually due to worsening uremia. Electrocardiographic (ECG) changes typical of acute pericarditis are not seen. Pericardial friction

rubs are louder than in most other forms of pericarditis, often palpable, and frequently persist after the metabolic abnormalities have been corrected. Uremic pericarditis is treated with intensive dialysis. Cardiac tamponade is the most serious complication of uremic pericarditis. It presents with changes in mental status, hypotension, or dyspnea. An enlarged heart on chest x-ray may suggest the diagnosis, which can be confirmed with echocardiography. Hemodynamically significant pericardial effusions require pericardiocentesis under fluoroscopic or ultrasonographic guidance.

## **NEUROLOGIC COMPLICATIONS**

Uremic encephalopathy presents with cognitive defects, memory loss, slurred speech, and asterixis. The progressive neurologic symptoms of uremia are the most common indications for initiating HD. It should remain a diagnosis of exclusion until structural, vascular, infectious, toxic, and metabolic causes of neurologic dysfunction have been ruled out. Peripheral neuropathy, manifested by paresthesias, diminished deep tendon reflexes, impaired vibration sense, muscle wasting, and weakness, occurs in 60% to 100% of patients with ESRD. Autonomic dysfunction, characterized by postural dizziness, gastric fullness, bowel dysfunction, reduced sweating, reduced heart rate variability, and baroreceptor control impairment, is common in ESRD patients, but is not responsible for intradialytic hypotension. Stroke is seen in 6% of HD patients, with 52% of cases caused by intracranial hemorrhage (subdural hematoma in particular). Stroke may be caused by cerebrovascular disease, head trauma, bleeding dyscrasias, anticoagulation, excessive ultrafiltration, or hypertension. It should be considered in any ESRD patient presenting with a change in mental status.

## **HEMATOLOGIC COMPLICATIONS**

Anemia is caused by decreased erythropoietin, blood loss from dialysis, frequent phlebotomy, and decreased red cell survival. Factitious anemia reflects changes in plasma volume related to dialysis. Abnormal hemostasis

in ESRD is multifactorial in origin, resulting in an increased risk of gastrointestinal (GI) tract bleeding, subcapsular liver hematomas, subdural hematomas, and intraocular bleeding. Immunologic compromise, caused by impaired leukocyte chemotaxis and phagocytosis, leads to high mortality rates from infection. Dialysis does not appear to improve immune system function.

## **GASTROINTESTINAL COMPLICATIONS**

Anorexia, nausea, and vomiting are common symptoms of uremia, and are used as an indication to initiate dialysis, and assess its efficacy. Chronic constipation is common, due to decreased fluid intake, and the use of phosphate-binding gels.

## **COMPLICATIONS OF HEMODIALYSIS**

Hypotension is the most frequent complication of HD. Excessive ultrafiltration from underestimation of the patient's ideal blood volume (dry weight) is the most common cause of intradialytic hypotension. Cardiac compensation for fluid loss may be compromised by diastolic dysfunction common in ESRD patients. Other causes of intradialytic hypotension include myocardial dysfunction from ischemia, hypoxia, arrhythmias, and pericardial tamponade; abnormalities of vascular tone secondary to sepsis, overproduction of nitric oxide, and antihypertensive medications; and volume loss from inadequate oral intake, vomiting, diarrhea, GI bleeding, or blood tubing or filter leaks. Treatment consists of Trendelenberg positioning, oral salt solution, or infusion of parenteral normal saline solution. If these interventions fail, excessive ultrafiltration is unlikely, and further evaluation will be required.

Dialysis disequilibrium, caused by cerebral edema following large solute clearances, is characterized by nausea, vomiting, and hypertension, which can progress to seizures, coma, and death. Treatment consists of terminating dialysis, and administering mannitol intravenously to increase serum

osmolarity. This syndrome should be distinguished from other neurologic disorders, such as subdural hematoma, stroke, hypertensive crisis, hypoxia, and seizures.

## COMPLICATIONS OF VASCULAR ACCESS

Complications of vascular access account for more inpatient hospital days than any other complication of HD. Thrombosis or stenosis present with loss of the bruit and a thrill over the access. These need to be treated within 24 hours with angiographic clot removal, angioplasty, or direct injection of thrombolytic (eg, **alteplase** 2.2 milligrams) into the access. Vascular access infections often present with signs of systemic sepsis, including fever, hypotension, and an elevated white blood cell (WBC) count. Classic signs of pain, erythema, swelling, and discharge are often missing. *Staphylococcus aureus* is the most common infecting organism, followed by gram-negative bacteria. Patients usually require hospitalization, and treatment with **vancomycin** (15 milligrams/kilogram), and an aminoglycoside (eg, **gentamycin** 100 milligrams intravenously). Potential life-threatening hemorrhage from a vascular access may result from a ruptured aneurysm or anastomosis, or over-anticoagulation. Bleeding can often be controlled with 5 to 10 min of pressure at the puncture site. If this fails, the addition of an adsorbable gelatin sponge soaked in reconstituted thrombin, or a prothrombotic gauze (eg, **HemCon** or **QuikClot**), followed by 10 min of direct pressure may be effective. Life-threatening hemorrhage may require placement of a tourniquet proximal to the access, and vascular surgery consultation. If the etiology is excessive anticoagulation, the effects of heparin can be reversed with protamine 0.01 milligram/unit heparin dispensed during dialysis (10 to 20 milligrams **protamine** if the heparin dose is unknown). If a newly inserted vascular access continues to bleed, **desmopressin** acetate (0.3 microgram/ kilogram intravenously) can be given as an adjunct to direct pressure.

## COMPLICATIONS OF PERITONEAL DIALYSIS



Peritonitis is the most common complication of peritoneal dialysis (PD). Signs and symptoms are similar to those seen in other patients with peritonitis, and include fever, abdominal pain, and rebound tenderness. A cloudy effluent supports the diagnosis. Peritoneal fluid should be sent to the laboratory for cell count, Gramstain, culture, and sensitivity. With peritonitis, cell counts usually reveal  $> 100$  leukocytes/mm<sup>3</sup>, with  $> 50\%$  neutrophils. Gram stain is positive in only 10% to 40% of culture-proven peritonitis. Organisms isolated include *Staphylococcus epidermidis*, *S aureus*, *Streptococcus* species, and gram-negative bacteria. Empiric therapy begins with a few rapid exchanges of dialysate to decrease the number of inflammatory cells within the peritoneum. The addition of **heparin** (500 to 1000 units/L dialysate) decreases fibrin clot formation. Empiric antibiotics, covering gram-positive organisms (eg, **cephalothin** or **vancomycin** 500 milligrams/L dialysate) and gram-negative organisms (eg, **gentamycin** 100 milligrams/L dialysate), are added to the dialysate. Inpatient versus outpatient treatment of PD-related peritonitis should be based on clinical presentation.

Infections around the PD catheter are characterized by pain, erythema, swelling, and discharge. Causative organisms are *S aureus* and *Pseudomonas aeruginosa*. Outpatient treatment consists of a first-generation cephalosporin or ciprofloxacin.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 93, “Emergencies in Renal Failure and Dialysis Patients,” by Mark Spektor and Richard Sinert.

## CHAPTER 53

# Urinary Tract Infections and Hematuria

Ann T. McIntosh

Urinary tract infections (UTIs) account for up to 3% of emergency department visits. Urethritis and cystitis are infections of the lower urinary tract. Pyelonephritis is an infection of the upper urinary tract. Up to 80% of UTIs are caused by *Escherichia coli*. The rest are caused by *Staphylococcus saprophyticus*, *Klebsiella*, *Proteus*, *Enterobacter*, *Pseudomonas*, and *Chlamydia trachomatis*.

Adults at risk for UTI include women between 18 and 30 years of age, and the elderly of both sexes. Males younger than 50 years of age with symptoms of dysuria or urinary frequency usually have urethritis caused by sexually transmitted infections. UTIs in children are discussed in [Chapter 75](#).

## CLINICAL FEATURES

Typical symptoms of lower urinary tract infections are dysuria, frequency, and urgency. The addition of flank pain; costovertebral angle (CVA) tenderness; fever; and systemic symptoms, often nausea and vomiting; constitute pyelonephritis. Subclinical pyelonephritis is present in 25% to 30% of patients with cystitis. Atypical symptoms are found in patients at risk for complicated UTI. Suspect UTI in elderly or debilitated patients presenting with weakness, general malaise, generalized abdominal pain, or mental status changes. Urethral or vaginal discharge is more consistent with urethritis and vaginitis, and the possibility of a sexually transmitted disease. Asymptomatic bacteriuria is defined as two positive cultures without symptoms. Since cultures are not available acutely, asymptomatic bacteriuria is diagnosed in the emergency department when bacteria are found on microscopy in patients with no symptoms. Asymptomatic bacteriuria is commonly found in patients with indwelling catheters, up to

30% of pregnant women, and 40% of female nursing home patients. Empiric treatment is recommended for asymptomatic bacteriuria during pregnancy.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of UTI is based on patient symptoms and signs, with individualized assessment of urine dipstick, urinalysis, and culture in selected patients. Typically, urine dipstick and urine microscopy is performed at minimum; woman of childbearing potential should have a pregnancy test. Clean catch specimens are adequate for most patients; catheterization should be used in a patient that cannot void spontaneously, is immobilized, or is too ill or obese to be able to provide a clean voided specimen. Although the gold standard for the diagnosis is urine culture, it is not required in all cases diagnosed in the ED. Uncomplicated lower urinary tract infections (woman with symptoms, pyuria, dipstick positive for nitrite and/or leukocyte esterase) can usually be managed as an outpatient without a culture. Obtain a culture in all other cases.

*Criteria for complicated UTI* includes positive laboratory testing in the setting of: prior history of UTI (reoccurrence in less than 1 month or more than 3 infections per year, which defines recurrent); UTI with an atypical organism (non-*E coli*) or known antibiotic resistance; a functionally or anatomically abnormal urinary tract; comorbidities (metabolic diseases, carcinoma, immune suppression, sickle cell anemia); advanced neurologic disease; advanced age; nursing home residency; indwelling catheter or recent urinary tract instrumentation; pregnancy; or male sex.

The urine nitrite reaction is greater than 90% specific but only about 50% sensitive in the diagnosis of UTI. A positive result with symptoms and bacteriuria is confirmatory. UTI with *Enterococcus*, *Pseudomonas* or *Acineobacter* results in a negative nitrite test. The leukocyte esterase reaction is more sensitive (77%) but less specific (54%) than the nitrite reaction. If it is positive, it is supportive of UTI. In summary, a positive urine dipstick nitrate or leukocyte test result supports the diagnosis of UTI; a negative test result does not exclude it.

A urine white blood count per high power field (WBC/HPF) of greater than 2 to 5 cells in women and 1 to 2 cells in men, in a patient with appropriate symptoms, is suggestive of a UTI. In a symptomatic patient with less than 5 WBC/HPF, one must consider causes of false-negative pyuria. These include dilute urine, systemic leukopenia, partially treated UTI, and obstruction of an infected kidney. Any bacteria on an uncentrifuged specimen is abnormal, and more than 1 to 2 bacteria per HPF in a centrifuged specimen is 95% sensitive and more than 60% specific for UTI. False-negative results may occur in a low colony count infection or in the case of *Chlamydia*. False-positive results may occur due to contamination with fecal or vaginal flora.

In patients with urinary catheters, the diagnosis of UTI is difficult as both pyuria and asymptomatic bacteruria are near-universal by the fourth 4 week indwelling. Treatment is only recommended in symptomatic patients; see [Chapter 57](#) for detailed criteria of symptomatic catheter-associated infection. Renal imaging should be considered acutely in the severely ill, if there is suspicion for a stone associated with infection, and with a poor initial response to therapy.

Differential diagnostic considerations include: upper and lower urinary tract infections, urethritis due to sexually transmitted infections (which are more common than cystitis/pyelonephritis in males younger than 50 years of age), vaginitis, (both sexually and nonsexually transmitted), vulvodynia, prostatitis, epididymitis, and intraabdominal pathology.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Treatment is determined by whether the urinary tract infection is complicated or uncomplicated.

Acute pyelonephritis can be treated as an outpatient if the patient has normal anatomy and is otherwise healthy. Urine culture, larger doses, and longer duration of antibiotics are recommended.

1. Uncomplicated UTI. Empiric treatment is best based on local resistance patterns. For uncomplicated lower urinary tract infections in women, **TMP-SMX DS** (160/800 milligrams twice a day for 3- to 5-days) is recommended as first choice in areas, where *E coli* resistance is less than 20%. However, 20% to 30% of patients given 3- to 5-day therapy will experience treatment failure or rapid relapse. **Nitrofurantoin** (100 milligrams 4 times a day or 100 milligrams extended release twice a day for 5 days) is a first-choice antibiotic with lower resistance. Nitrofurantoin is recommended for asymptomatic bacteriuria during pregnancy.
2. Complicated UTI. Use fluoroquinolones (**ciprofloxacin** 500 milligrams twice a day or **levofloxacin** 500 milligrams once a day), **cefpodoxime** (200 milligrams twice a day), or **fosfomicin** (3 grams once), in males, cases where symptoms suggest upper urinary tract involvement or have been present for more than a week, infection is recurrent, follow-up is unsure, there are complicating factors, or local resistance to TMP-SMX is greater than 20%. Duration of therapy should be 10 to 14 days. Ciprofloxacin resistance may preclude its effective use in some communities.
3. Use caution with nitrofurantoin and the fluoroquinolones in the elderly and in patients with renal insufficiency.
4. If there is suspicion for concomitant infection with gonorrhea and/or *Chlamydia*, antibiotic choice is more complex. (See [Chapter 87](#) “Sexually Transmitted Diseases”).
5. Consider 1 to 2 days of an oral bladder analgesic such as **phenazopyridine** 200 milligrams, 3 times a day.
6. Discharge instructions must include instructions to return for increased pain, fever, vomiting or intolerance of medications, to take the entire course of antibiotics, and to follow up with primary care provider. Encourage fluids, (cranberry juice may be helpful), and frequent voiding.
7. Admission is indicated for pyelonephritis associated with intractable vomiting and should be considered for complicated UTIs. Empiric antibiotic therapy should be initiated in the ED: **ciprofloxacin** 400 milligrams IV every 12 hours, **ceftriaxone**, 1 gram IV once daily, **gentamicin**, or **tobramycin**, 3.0 milligrams/kilogram/d divided every 8

hours ± **ampicillin** 1 to 2 grams every 4 hours. For patients with unstable vital signs, see [Chapter 89](#) “Septic Shock”.

## **HEMATURIA**

Hematuria is blood in the urine. It is either visible to the eye, gross hematuria, requiring 1 mL of whole blood per liter; or microscopic, only seen under the microscope, defined as greater than 3 to 5 RBCs per HPF.

### **Clinical Features**

Gross hematuria suggests a lower urinary tract source; microscopic hematuria suggests a renal source. Asymptomatic hematuria is more often due to neoplasm or vascular causes than infection. Asymptomatic hematuria is defined as greater than 3 to 5 RBCs per HPF on 2 of 3 properly collected urine specimens in a patient with no symptoms.

### **Diagnosis and Differential**

A urine dipstick is positive with approximately 5 to 20 red blood cells per mL of urine. All positive dipsticks should be followed by microscopy. False-positive results can occur with the presence of myoglobin, porphyrins, free hemoglobin (as opposed to intact RBCs) due to hemolysis, and povidone-iodine. Catheterization usually does not cause an abnormal result. False-negative results can be seen with very high specific gravity. Differential diagnostic considerations are numerous. Consider the patients' age, sex, demographic characteristics, habits, potential risk factors for urologic malignancy, comorbidities, or any history of recent urinary tract instrumentation. The most common causes of hematuria are UTI, nephrolithiasis, neoplasms, benign prostatic hypertrophy, glomerulonephritis, and schistosomiasis (most common cause worldwide). In the ED, consider strenuous exercise, poststreptococcal infection (in younger patients) and life threats including malignant hypertension, eroding

abdominal aortic aneurysm, coagulopathy, foreign body, immune-mediated disease (Henoch-Schönlein purpura, pulmonary-renal syndromes), sickle cell disease complications, and renal vein thrombosis.

## **Emergency Department Care and Disposition**

1. Treatment of hematuria is directed at the cause. ED management consists of the minimization of complications and appropriate referral or admission for further evaluation.
2. All hematuria should be followed up by either primary care or urology within 2 weeks.
3. Admit patients with infection associated with an obstructive stone, intractable pain, intolerance of medications or oral fluids, newly diagnosed glomerular nephritis, significant anemia, renal insufficiency, significant comorbidity, bladder outlet obstruction, pregnancy with preeclampsia, pyelonephritis, obstructive stone; or any potentially lifethreatening causes of hematuria.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 94, “Urinary Tract Infections,” by David S. Howes and Mark P. Bogner and Chapter 97, “Hematuria and Hematospermia,” by David S. Howes and Mark P. Bogner.

# **CHAPTER 54**

## **Acute Urinary Retention**

Casey Glass

### **CLINICAL FEATURES**

Urinary retention can be either acute or chronic. The most common cause of retention is outlet obstruction secondary to benign prostatic hypertrophy in men although medication use, acute neurologic dysfunction, urinary tract bleeding or calculi, and other anatomic obstruction are also common causes in both sexes. Acute syndromes typically present with rapid onset of lower abdominal pain occasionally radiating to the lower back. Patients typically complain of difficulty voiding but some may not volunteer this information. Chronic obstruction usually presents with lower abdominal pain and the patient may note incomplete voiding or the need to void frequently.

The history should address previous episodes of obstruction, recent medication changes and over-the-counter medicine use. Assess for any history of trauma or neurologic disability or symptoms of infection. It is critical to know if any recent urologic procedures or urinary catheterizations have been performed. The duration of symptoms is also important as it is associated with the development of postobstructive diuresis and renal dysfunction.

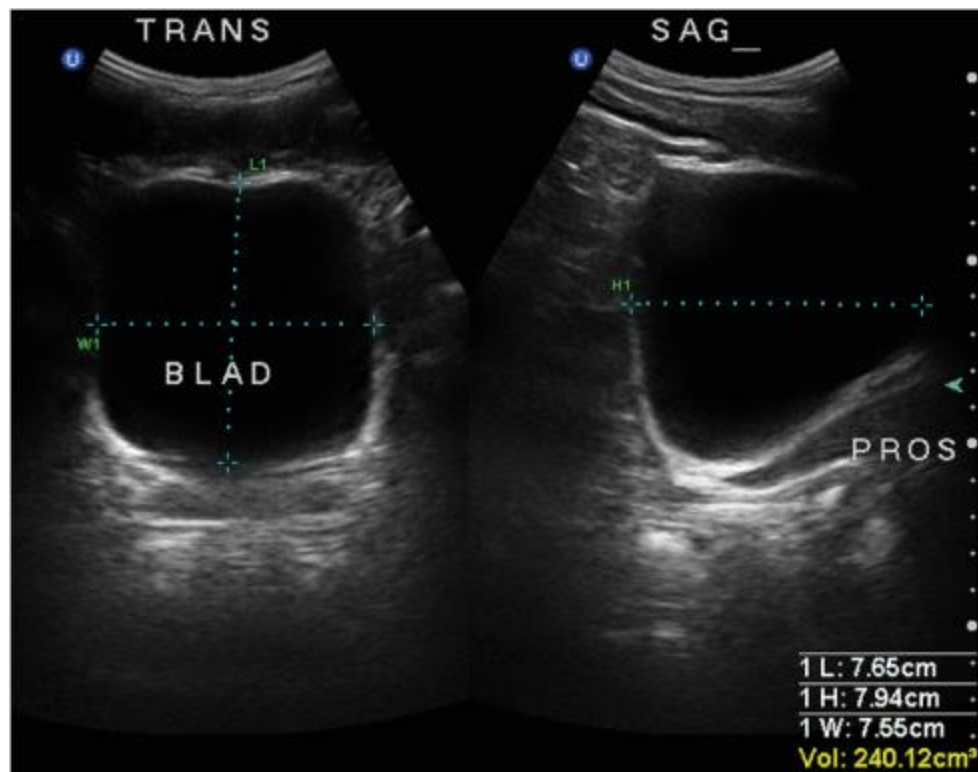
### **DIAGNOSIS AND DIFFERENTIAL**

Physical examination should address the functional and anatomic assessment of the lower urinary tract. Palpate the abdomen for a suprapubic mass corresponding to the distended urinary bladder. The penis should be examined for stricture at the meatus or palpable abnormalities of the penile urethra. The female lower urinary tract should be evaluated for bladder



prolapse or stricture of the urethral meatus. In men the prostate should be assessed for size, texture, and tenderness. Perineal sensation and anal sphincter tone should be documented. A comprehensive neurologic examination should be performed.

Bedside ultrasound can be very helpful in distinguishing both the degree of obstruction and in discriminating obstruction from the sensation of fullness associated with bladder spasm in conditions like inflammatory or infectious cystitis. The patient should first be encouraged to attempt to void. After a voiding attempt the bladder is imaged with a low-frequency sector format probe in both the transverse and sagittal views ([Fig. 54-1](#)). Many manufacturers have a calculation package available to estimate the retained urine volume. Residual volumes  $> 50$  to  $150$  cc are consistent with urinary retention, however, volumes are typically greater than  $300$  cc.



**FIGURE 54-1.** Transverse and sagittal views of the urinary bladder. The prostate is visualized as a medium echogenicity structure posterior and caudal to the bladder. Anterior-posterior, cradio-caudal, and transverse

measurements of the bladder are obtained for calculation of the bladder volume. (Reproduced with permission from Casey Glass, MD.)

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The goals of emergency department care are relieving the discomfort of retention, assessing for any secondary injury to the renal system, and treatment of the primary cause of retention.

1. Most patients with bladder outlet obstruction are in distress, and passage of a urethral catheter alleviates their pain and their urinary retention. Copious intraurethral lubrication including a topical anesthetic (2% lidocaine jelly) should be used, and a 16-French Coudé catheter is recommended if straight catheters fail. The catheter should be passed to its fullest extent to obtain free urine flow before inflating the balloon. A catheter should not be placed if there is suspicion of trauma to the urethra, either secondary to a traumatic event or recent instrumentation. The catheter should be left indwelling and connected to a leg drainage bag.
2. The patient with obstruction from hematuria represents a special case. A 3-way Foley catheter should be placed and the bladder irrigated until returning fluid is free of blood. These patients are likely to need admission for continued irrigation as the catheter often becomes blocked with clot following placement.
3. Failure to pass a urethral Foley or recent urologic procedure or instrumentation requires the involvement of a urologist for catheter placement, consultation should not be delayed. Urgent urologic consultation is also indicated for obstruction secondary to stricture, prostatitis, or urethral trauma.
4. Urine should be sent for routine analysis as well as culture. Electrolytes, BUN, and creatinine should be assessed for postobstructive renal failure. Urine output should be monitored quantitatively.

5. Oxybutynin can be prescribed for control of bladder spasms. This anticholinergic medicine can itself cause a functional obstruction. Patients may also require pain medication for control of discomfort from bladder spasms. Prescription of an  $\alpha$ -blocker is indicated for male patients in whom BPH is the suspected etiology of their obstruction.
6. Antibiotics are not indicated unless there is also evidence of cystitis.
7. If urinary retention has been chronic, postobstructive diuresis may occur even in the presence of normal blood urea nitrogen and creatinine levels. In such patients, close monitoring of urinary output is indicated, and they should be observed for 4 to 6 hours after catheterization.
8. Precipitating causes of retention must be addressed. Offending medications should be discontinued. Infectious or neurologic causes must be completely evaluated (urgency for work-up depends on patient acuity and comorbidities).
9. In all cases of urinary retention, urologic follow-up in 3 to 7 days is indicated for a complete genitourinary evaluation. Patients should usually expect to have the catheter removed at that visit.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 95, "Acute Urinary Retention," by David Hung-Tsang Yen and Chen-Hsen Lee.

# CHAPTER 55

## Male Genital Problems

Eric J. Brown

### TESTICULAR TORSION

#### Clinical Features

Testicular torsion must be the primary consideration in any male (in any age group) complaining of testicular pain. Pain usually occurs suddenly, is severe, and is felt in the lower abdominal quadrant, the inguinal canal, or the testis. The pain may be constant or intermittent but is not positional because torsion is primarily an ischemic event. Although symptom onset tends to occur after exertion, the testicle also may twist from unilateral cremasteric muscle contraction during sleep. Early in presentation, the affected testicle is firm, tender, elevated, and in a transverse lie compared to the contralateral testicle. The most sensitive finding (99% sensitive) in torsion is the unilateral absence of the cremasteric reflex.

#### Diagnosis and Differential

In indeterminate cases, color-flow duplex ultrasound and, less commonly, radionuclide imaging may be helpful. In addition, urinalysis is typically ordered, but pyuria does not rule out testicular torsion.

*Torsion of the appendages* is more common than testicular torsion but is not dangerous because the appendix testis and appendix epididymis have no known function. If the patient is seen early, diagnosis can be supported by the following: pain is most intense near the head of the epididymis or testis; there is an isolated tender nodule; or the pathognomonic blue dot appearance of a cyanotic appendage is illuminated through thin prepubertal

scrotal skin. If normal intratesticular blood flow can be demonstrated with color Doppler, immediate surgery is not necessary because most appendages calcify or degenerate over 10 to 14 days and cause no harm. The differential for testicular torsion also includes epididymitis, inguinal hernia, hydrocele, and scrotal hematoma.

## **Emergency Department Care and Disposition**

1. When the diagnosis is obvious, urologic consultation is indicated for exploration because imaging tests can be too time consuming. Testicular salvage is related to duration of symptoms with excellent salvage rates with <6 hours of symptoms.
2. The emergency physician can attempt manual detorsion. Most testes twist in a lateral to medial direction, so detorsion is performed in a medial to lateral direction, similar to the opening of a book. The endpoint for successful detorsion is pain relief; urologic referral is still indicated.
3. Urology is consulted early in the patient's course even when confirmatory testing is planned. When the diagnosis of testicular torsion cannot be ruled out by diagnostic studies or examination, urologic consultation is indicated.

## **EPIDIDYMITIS AND ORCHITIS**

### **Clinical Features**

Epididymitis is characterized by gradual onset of pain due to inflammatory causes. Bacterial infection is the most common, with infecting agents dependent on the patient's age. In patients younger than 40 years, epididymitis is due primarily to sexually transmitted diseases; culture or DNA probe for gonococcus and *Chlamydia* is indicated in males younger than 40 years even in the absence of urethral discharge. Common urinary pathogens predominate in older men. Epididymitis causes lower abdominal, inguinal canal, scrotal, or testicular pain alone or in combination. Due to the

inflammatory nature of the pain, patients with epididymitis may note transient pain relief when elevating the scrotal contents while recumbent (positive Prehn sign).

## Diagnosis and Differential

Initially, when tenderness is well localized to the epididymis, the clinical diagnosis is clear. However, progression of inflammation results in the physical examination finding of a single, large testicular mass (epididymo-orchitis), which is difficult to differentiate from testicular torsion or carcinoma. Testicular malignancy should be suspected in patients presenting with asymptomatic testicular mass, firmness, or induration. Ten percent of tumors present with pain due to hemorrhage within the tumor. Orchitis in isolation is rare; it usually occurs with viral or syphilitic disease.

## Emergency Department Care and Disposition

1. If the patient appears toxic, admission is indicated for intravenous antibiotics (eg, **ceftriaxone** 1 to 2 grams every 12 hours IV or **trimethoprim/ sulfamethoxazole** 5 milligrams/kilogram IV of trimethoprim component every 6 hours).
2. Outpatient treatment is the norm in patients who do not appear toxic; urologic follow-up within 1 week is indicated. Age <40: Treat gonorrhea and *Chlamydia* with **ceftriaxone** 250 milligrams IM single dose plus **doxycycline** 100 milligrams PO twice daily for 10 days. Age > 40: Treat gram-negative bacilli with **levofloxacin** 500 milligrams PO daily for 10 days or **ofloxacin** 300 milligrams PO twice daily for 10 days.
3. In addition, scrotal elevation, ice application, nonsteroidal anti-inflammatory drugs, opioids for analgesia, and stool softeners are indicated.
4. Orchitis is treated with disease-specific therapy, symptomatic support, and urologic follow-up. (Patients at risk for syphilitic disease should be treated as directed in [Chapter 86](#).)

## ACUTE PROSTATITIS

Patients present with varying complaints of suprapubic or genital pain, back pain, perineal pain, voiding difficulties, frequency, dysuria, pain with ejaculation, and fever and chills. Patients at risk include those with anatomic or neurophysiologic lower urinary tract obstruction, acute epididymitis or urethritis, unprotected rectal intercourse, phimosis, and indwelling urethral catheter. The causative organism is *Escherichia coli* in most cases, with *Pseudomonas*, *Klebsiella*, *Enterobacter*, *Serratia*, or *Staphylococcus* causing the remainder. Physical examination usually reveals perineal, rectal, and prostate tenderness. The diagnosis is clinical as urinalysis and culture are often negative even after prostate massage. Treatment is **ciprofloxacin** 500 milligrams twice daily, or **levofloxacin** 500 milligrams PO daily, or **ofloxacin** 300 milligrams PO twice daily. All treatments should be for a total of 30 days. Pain medicine may be required. Admission is not necessary unless the patient is septic, immunocompromised, has significant comorbidities, or has worsened on outpatient therapy.

## SCROTUM

*Scrotal abscesses* may be localized to the scrotal wall or may arise from extensions of infections of intrascrotal contents (ie, testis, epididymis, and bulbous urethra). A simple hair follicle scrotal wall abscess can be managed by incision and drainage; no antibiotics are required in immunocompetent patients. When a scrotal wall abscess is suspected of coming from an intrascrotal infection, ultrasound and retrograde urethrography may demonstrate pathology in the testis, and/or epididymis, and in the urethra, respectively. Definitive care of any complex abscess calls for a urology consultation.

*Fournier gangrene* is a polymicrobial infection of the perineal subcutaneous tissues. Diabetic males are at highest risk, but any immunocompromise can be associated with the disease. Prompt diagnosis is

essential to prevent extensive tissue loss. Early surgical consultation is recommended for at-risk patients who present with scrotal, rectal, or genital pain. Treatment mainstays include aggressive fluid resuscitation with normal saline solution and broad-spectrum antibiotics to cover gram-positive, gram-negative, and anaerobic organisms: **imipenem** 1 gram IV every 8 hours or **meropenem** 500 milligrams to 1 gram IV every 8 hours plus **vancomycin** 1 gram IV every 12 hours if methicillin-resistant *Staphylococcus aureus* is suspected. Care usually includes hyperbaric oxygen therapy (if readily available) and surgical debridement.

## PENIS

*Balanoposthitis* is inflammation of the glans (balanitis) and foreskin (posthitis). Upon foreskin retraction, the glans and prepuce appear purulent, excoriated, malodorous, and tender. Treatment consists of cleaning with mild soap, ensuring adequate dryness, applying antifungal creams (**nystatin** 100,000 units/g, 4 times daily or **clotrimazole** 1% cream bid) and using an oral azole (**fluconazole** 150 milligrams single dose, reevaluate at 7 days for repeat dosing). Urologic referral is needed for reassessment and possible circumcision. An oral cephalosporin (eg, **cephalexin** 500 milligrams 4 times daily) should be prescribed in cases of secondary bacterial infection.

*Phimosis* is the inability to retract the foreskin proximally ([Fig. 55-1](#)). Hemostatic dilation of the preputial ostium relieves the urinary retention until definitive dorsal slit or circumcision can be performed. Topical steroid therapy, such as **hydrocortisone** 1% cream for 4 to 6 weeks, reduces the rate of required circumcision.

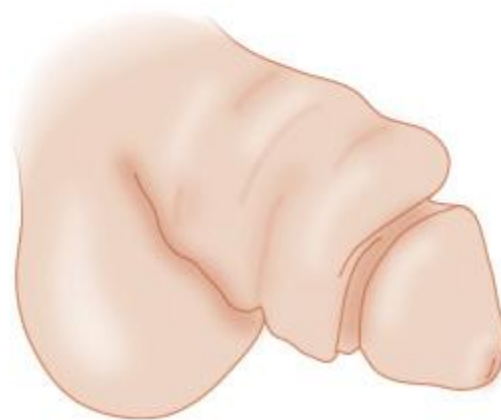
*Paraphimosis* is the inability to reduce the proximal edematous foreskin distally over the glans (see [Fig. 55-1](#)). Paraphimosis is a true urologic emergency because resulting glans edema and venous engorgement can progress to arterial compromise and gangrene. If surrounding tissue edema can be successfully compressed, as by wrapping the glans with 2 × 2-in elastic bandages for 5 min, the foreskin may be reduced. Making several puncture wounds with a small (22- to 25-gauge) needle may help with expression of glans edema fluid. Local anesthetic block of the penis is



helpful if patients cannot tolerate the discomfort associated with edema compression and removal. If arterial compromise is suspected or has occurred, local infiltration of the constricting band with 1% plain **lidocaine** followed by superficial vertical incision of the band will decompress the glans and allow foreskin reduction.



Phimosis



Paraphimosis

**FIGURE 55-1.** Phimosis and paraphimosis.

*Penile entrapment* injuries occur when various objects are wrapped around the penis. Such objects should be removed, and urethral integrity (retrograde urethrogram) and distal penile arterial blood supply (Doppler studies) should be confirmed when indicated.

*Penile fracture* occurs when there is an acute tear of the penile tunica albuginea. The penis is acutely swollen, discolored, and tender in a patient with history of intercourse-associated trauma accompanied by a snapping sound. Retrograde urethrography may be indicated for assurance of urethral integrity. Urologic consultation is indicated.

*Peyronie disease* presents with patients noting sudden or gradual onset of dorsal penile curvature with erections. Examination shows a thickened plaque on the dorsal penile shaft. Assurance and urologic follow-up are indicated.

*Priapism* is a painful pathologic erection that may be associated with urinary retention. Infection and impotence are other complications. In most cases, the initial therapy for priapism is **terbutaline** 0.25 to 0.5 milligrams (repeated in 20 min, if needed) subcutaneously in the deltoid area. If patients present early (within 4 hours), oral **pseudoephedrine** (60 to 120 milligrams) may be effective. Patients with priapism from sickle cell disease are usually treated with simple or exchange transfusion. Corporal aspiration and irrigation with normal saline solution or an  $\alpha$ -adrenergic antagonist is the next step and may need to be performed by the emergency physician when urologic consultation is not available. Even when emergency physicians provide stabilizing care, urologic consultation is indicated in all cases.

## URETHRA

*Urethral stricture* is becoming more common due to the high incidence of sexually transmitted diseases. If a patient's bladder cannot be cannulated with a 14- or 16-French Foley or Coudé catheter, the differential diagnosis includes urethral stricture, voluntary external sphincter spasm, bladder-neck contracture, or benign prostatic hypertrophy. Retrograde urethrography can be performed to delineate the location and extent of urethral stricture. Endoscopy is necessary to confirm bladder neck contracture or define the extent of an obstructing prostate gland. Suspected voluntary external sphincter spasm can be overcome by holding the patient's penis upright and encouraging him to relax his perineum and breathe slowly during the

procedure. After no more than 3 gentle attempts to pass a 12-French Coudé catheter into a urethra prepared with anesthetic lubricant, urologic consultation should be obtained. In an emergency situation, suprapubic cystotomy can be performed. The infraumbilical and suprapubic areas are prepped with povidone-iodine solution. A 25- to 27-gauge spinal needle is used to locate the bladder (emergency department ultrasound can facilitate this), followed by placement of the cystotomy with the Seldinger technique. Urologic follow-up should occur within 48 hours.

*Urethral foreign bodies* are associated with bloody urine and slow, painful urination. Radiography of the bladder and urethral areas may disclose a foreign body. Removal of the foreign body may be achieved with a gentle milking action; retrograde urethrography or endoscopy is required in such cases to confirm an intact urethra. Often, urologic consultation for endoscopy or open cystotomy is required for foreign body removal.

For further reading in *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 96, "Male Genital Problems," by Bret A. Nicks and David E. Manthey.

# **CHAPTER 56**

## **Urologic Stone Disease**

Geetika Gupta

The acute phenomenon of renal stones migrating down the ureter is referred to as *renal colic*. Adults and children can develop kidney stones. In adults, the condition is 3 times more common in males than in females; kidney stones usually occur in the third to fifth decade of life. Children under the age of 16 years old constitute 7% of cases seen, with the distribution being equal between the sexes.

### **CLINICAL FEATURES**

Patients usually present with an acute onset of severe pain, which may be associated with nausea, vomiting, and diaphoresis. Patients are frequently anxious, pacing, or writhing and are unable to hold still or converse. The pain is sharp and episodic in nature due to the intermittent obstruction of the ureter and is relieved after the stone passes. The pain typically originates in either flank with subsequent radiation around the abdomen toward the groin. However, as the stone passes into the distal ureter, where 75% of stones are diagnosed, the pain may be located in the anterior abdominal or suprapubic area. Vesicular stones may present with intermittent dysuria and terminal hematuria. Children may present in a similar fashion, but up to 30% have only painless hematuria. Vital signs may demonstrate tachycardia and an elevated blood pressure, which are secondary to pain. Pyrexia may be present if there is a concomitant urinary tract infection. Examination may show costovertebral tenderness or abdominal tenderness, guarding, or rigidity. Hematuria may be present in 85% of patients with renal colic.

### **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of urologic stones and renal colic is based on clinical judgement. A urinalysis will help rule out infection and assess for microscopic hematuria.

All females of childbearing age should have a pregnancy test. Imaging is controversial. It allows confirmation of the ureteral stone, rules out other diagnoses, identifies complications, establishes the location of the stone, and assists with the management if the stone fails to pass spontaneously. The use of noncontrast helical computed tomography is the mainstay of diagnosis in the emergency department (ED). Positive findings include changes in the ureteral caliber, suspicious calcifications, stranding of perinephric fat, and dilation of the collecting system ([Fig. 56-1](#)). It has a PPV of 96% and a NPV of 93% to 97%. The greatest advantage is the speed at which it can be performed, no need for intravenous contrast administration, and the benefits of excluding other diagnoses. The disadvantages are that it does not evaluate renal function and has radiation exposure.



**FIGURE 56-1.** CT image shows 5-mm stone (arrow) at left ureterovesical junction. Other calcifications are seen in the pelvis, unrelated to the urinary

outflow system.

The use of plain kidney-ureter-bladder film (KUB) is limited by the visibility of the stone; the KUB cannot rule out a ureteral stone. The KUB is useful in following the progression of a stone, if visualized, in the outpatient setting.

Ultrasound, an anatomic rather than a functional test, is useful in patients who are not candidates for intravenous pyelogram (IVP) or computed tomography. It detects hydronephrosis and larger stones but is not sensitive for midureteral stones or small stones (<5 mm).

It is critical to consider vascular etiologies in the differential diagnosis for abdominal-flank pain, including abdominal aortic aneurysm and aortic dissections. Other concerning differentials include appendicitis, mesenteric ischemia, cholecystitis, ectopic pregnancy, gonadal torsion, renal infarction, incarcerated hernia, epididymitis, salpingitis, pyelonephritis, papillary necrosis (sickle cell disease, diabetes, nonsteroidal analgesic abuse, or infection), herpes zoster, drug-seeking behavior, and musculoskeletal strain. Patients receiving outpatient extracorporeal shock wave lithotripsy for urolithiasis may present to the ED with renal colic because the resulting “sludge” is passed in the urine.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Analgesia: nonsteroidal anti-inflammatory drugs administered parenterally are considered the primary analgesic choice. The prostaglandins promote dilation of the ureter and thus aid in the alleviation of the source of the pain. **Ketorolac** 30 milligrams IVP is the recommended starting dose. Caution should be used in the elderly, those with bleeding tendencies, and renal impairment. Opioids, in titrated doses of **morphine** 5 milligrams IV or **hydromorphone** 1 milligram IV, may also be used to assist in pain control.

2. Antiemetics: **metoclopramide**, 10 milligrams IV, the only antiemetic drug studied for the treatment of renal colic has been shown to be effective in providing relief in nausea and pain. Other antiemetics may also be used.
3. Medical expulsion therapy:  $\alpha$ -blockers demonstrate a 2- to 6-day improvement in time to expulsion. Options include **tamsulosin** (0.4 milligram daily up to 4 weeks), **terazosin** (5 to 10 milligrams daily), and **doxazosin** (4 milligrams daily).
4. Antibiotics: if the urinalysis shows evidence for an infection, the choice of antibiotic is dictated by the local resistance patterns to gram-negative rods. Patients with an infected ureteral stone without significant obstruction, fever or systemic illness, may be discharged with a fluoroquinolone such as **ciprofloxacin** (500 milligrams twice a day for 10 to 14 days) or **levofloxacin** (500 milligrams daily for 10 to 14 days) or a third-generation cephalosporin such as **cefepime** (200 milligrams twice a day for 10 to 14 days). Follow-up is essential.
5. Those patients who have a more complicated clinical situation should be admitted. Intravenous antibiotic selections to consider include **gentamicin** or **tobramycin** (1.0 milligram/kilogram/dose every 8 hours) and **ampicillin** (1 to 2 grams every 4 hours); **piperacillin-tazobactam** (3.375 grams every 6 hours); **cefepime** (2 grams every 8 hours); **ticarcillin-clavulanic acid** (3.1 grams every 6 hours), or **ciprofloxacin** (400 milligrams every 12 hours).
6. Discharge is appropriate for patients with small unilateral stones (<6 mm), no complicated infection, and pain controlled by oral analgesics. It is recommended that patients be given a urinary strainer, prescriptions for oral analgesics and medical expulsive therapy, and urologic follow-up within 7 days. If the stone is passed in the ED, no treatment is necessary other than elective urologic follow-up. Patients should be instructed to return if they develop fever, persistent vomiting, or uncontrolled pain. On average, the stone may take 7 to 20 days to pass.
7. Urologic consultation on an emergent basis is needed in those patients with a complete obstruction complicated by a solitary or transplanted kidney, fever, and/or urosepsis. Disposition should be discussed with a urologist in patients with a stone larger than 6 mm, renal insufficiency,

severe underlying disease, extravasation or complete obstruction, sloughed renal papillae, UTI, or failed outpatient management.

8. Hospitalization is absolutely indicated for those patients who have a solitary or transplanted kidney with obstruction, uncontrolled severe pain, intractable emesis, acute renal failure, hypercalcemic crisis, severe medical comorbidities, and urosepsis. Those individuals with a fever, solitary or transplanted kidney without obstruction, obstructing stone with infection, urinary extravasation, significant medical comorbidities, or large proximal ureteral stones should be considered for admission.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 97, "Urologic Stone Disease," by David E. Manthey and Bret A. Nicks.



# **CHAPTER 57**

## **Complications of Urologic Devices**

William K. Gray

### **LITHOTRIPSY**

Common post lithotripsy complications include: abdominal pain, nausea and vomiting, ureteral colic, fever, and skin ecchymosis. Supportive therapy with IV fluids, analgesics, and antiemetics may be indicated. Check complete blood count, creatinine, urinalysis, and urine output; use antibiotics if appropriate. Severe flank pain, fall in hematocrit, hypotension, and syncope, can be caused by perinephric and renal hematomas. Diagnosis is by CT or US. Acute management may include IV fluids, blood transfusions, analgesics, and antibiotics. It is important to consult urology early in the process. Rare complications include injury to abdominal viscera and surrounding structures. Consult surgery and urology for these complications.

### **COMPLICATIONS OF URINARY CATHETERS**

Complications related to the use of urinary catheters include infection, leakage, obstruction, and trauma during placement. Most catheters are made of latex, but silicone is available for the latex allergies.

#### **Catheter-Related Urinary Tract Infection**

Antibiotic treatment of asymptomatic bacteriuria in a patient with a short-term catheter is not recommended. Pyuria is universal for patients with long-term (1 month) indwelling catheters; pyuria should not be used in the

diagnosis of asymptomatic infection. Hematuria is a better indicator of infection. CA-UTI in patients with indwelling urethral, indwelling suprapubic, or intermittent catheterization is defined by the presence of symptoms or signs compatible with UTI with no other identified source of infection along with  $\geq 10^3$  colony-forming units/mL of  $\geq 1$  bacterial species in a single catheter urine specimen. Signs and symptoms compatible with CA-UTI include new onset or worsening of fever, rigors, altered mental status, malaise, or lethargy with no other identified cause; flank pain; costovertebral angle tenderness; acute hematuria; pelvic discomfort; and in those whose catheters have been removed, dysuria, urgent or frequent urination, or suprapubic pain or tenderness. In those patients with mild symptoms, treatment is **ciprofloxacin** 500 milligrams twice a day, or **levofloxacin** 500 milligrams once a day, or **cefpodoxime** (200 milligrams twice a day). Seven days is the recommended duration of antimicrobial treatment for patients with CA-UTI who have prompt resolution of symptoms (A-III), and 10 to 14 days of treatment is recommended for those with a delayed response. Pyelonephritis is the most common complication of catheter-related UTI with fever. Admission is frequently required. (See [Chapter 53](#) for further antibiotic recommendations). Check urine cultures and blood cultures if septic (see [Chapter 89](#) for septic shock). Replace the catheter if it has been in place > 7 days.

## **Nondraining Catheters**

Obstruction by blood clots is suggested when the catheter is easily flushed, but there is little or no irrigate return. If this occurs, the catheter may be replaced with a triple lumen catheter for bladder irrigation. Sometimes a larger single lumen catheter may aid in the evacuation of larger clots. If there is evidence of continual bleeding after proper irrigation, then consult urology. Long-term indwelling Foley catheters can become obstructed by intraluminal encrustations. Leakage may occur secondary to obstruction or bladder spasms.

## **Nondeflating Retention Balloon**

In a distal obstruction secondary to a crushed or defective valve, the catheter can be cut proximal to the defect. If this does not deflate the balloon a lubricated guide wire can be introduced into the cut channel in order to clean the obstruction.

## **Traumatic Complications**

Simple insertion of a Foley catheter may injure the urethra and cause bleeding, especially in the setting of prostatic hypertrophy. No specific management is required in most cases. The urethra can be injured by inflation of the balloon within the urethra. Urology should be consulted for management in these cases.

## **COMPLICATIONS OF PERCUTANEOUS NEPHROSTOMY TUBES**

Percutaneous nephrostomy is a urinary drainage procedure used for supravescical or ureteral obstruction secondary to malignancy, pyonephrosis, genitourinary stones, or ureteral strictures. You may also see it used in ureteral obstruction secondary to pregnancy. Bleeding may occur and can usually be managed with irrigation to clear the nephrostomy tube. If bleeding persists check a complete blood cell count, renal functions, and coagulation studies. Treat the patient for hemodynamic instability and consult urology.

Infectious complications from nephrostomy tubes include simple bacteruria, pyelonephritis, renal abscess, bacteremia, and urosepsis. Any wound drainage should be cultured and antibiotics should be administered after consulting urology. Mechanical complications can occur with these devices, such as catheter dislodgment and tube blockage. The urologist has several techniques available to reestablish access to an obstructed nephrostomy tube.

## **COMPLICATIONS OF ARTIFICIAL URINARY SPHINCTERS**

The artificial sphincter is a device used for urinary incontinence, neurogenic bladder with incontinence, trauma to the urethra, and congenital conditions. Infections are the most serious complication of the artificial sphincter. Infections occurring early after implantation are usually due to skin flora. Later infections are usually due to gram-negative organisms. Never introduce a urinary drainage catheter through an artificial urinary sphincter. Consult the urologists for further management and evaluation.

## **COMPLICATIONS OF URETERAL STENTS**

Symptoms such as flank pain, dysuria, urgency, urinary frequency, and incontinence are common complaints in patients with ureteral stents. The baseline discomfort in a functioning, well-positioned ureteral stent can range from minimal to debilitating, depending on the patient. Any abrupt change in the character, location, or intensity of the pain requires further evaluation for stent malposition, malfunction, and infection.

Ureteral stents can become obstructed secondary to encrustations with mineral deposits. This occurs more often in stents in place for long-term use. These patients may require urologic consultation.

## **URINARY TRACT INFECTIONS VERSUS STENT MIGRATION AND MALFUNCTION**

Changing abdominal pain or flank pain or bladder discomfort may be indicative of stent migration and stent fragmentation. A plain film x-ray is indicated with comparison to previous films to evaluate stent position. Urologic consultation and further studies to evaluate stent position may be necessary.

Most minor urinary tract infections in the presence of a ureteral stent can be treated with outpatient antibiotics. More serious infections such as pyelonephritis and sepsis will require IV antibiotics, radiologic studies to determine stent position, and urologic consultations.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 98, “Complications of Urologic Procedures and Devices” by Elaine B. Josephson and Moira McCarty.

## **SECTION 8 Gynecology and Obstetrics**

## **CHAPTER 58**

# **Vaginal Bleeding and Pelvic Pain in the Nonpregnant Patient**

Thomas W. Lukens

## **VAGINAL BLEEDING**

Vaginal bleeding is a common complaint in females presenting to the ED. Determination of pregnancy status in each patient is important in order to formulate the appropriate differential diagnosis and to guide subsequent testing and decision making.

### **Clinical Features**

Adolescents and adults should be asked about amount and duration of bleeding, reproductive history, sexual history, history of sexually transmitted infections, history of trauma, medication, possibility of foreign bodies; bleeding disorders (history of bruising, epistaxis, other abnormal bleeding), endocrine disorder, liver disease and associated GU or systemic symptoms. An abdominal and gynecologic examination, including speculum examination as well as a vaginoabdominal examination (bimanual) should be performed on nonvirginal adults and adolescents to look for structural or traumatic causes of bleeding. Skin or conjunctiva pallor or abnormal vital signs can indicate significant anemia.

### **Diagnosis and Differential**

In prepubertal children, bleeding from genital trauma and/or sexual assault needs to be considered and excluded. Vulvovaginitis is unusual but is the

most common cause of vaginal bleeding in the prepubertal female and can be associated with pain. Although nonspecific vulvovaginitis is most commonly diagnosed, specific infections with candidiasis, streptococcal infections, *Escherichia coli* and *Shigella*, viruses and pinworms also occur. Bleeding coupled with vaginal discharge raises concerns for retained foreign bodies. Less common etiologies include precocious puberty and menarche, congenital abnormalities, urethral prolapse, and tissue sensitivity to chemicals in soaps and creams.

In women of reproductive age or perimenopausal women, bleeding can arise from the uterus or cervix and is most commonly due to anovulation, pregnancy, exogenous hormone use, coagulopathy, uterine leiomyomas, cervical and endometrial polyps, pelvic infections, and thyroid dysfunction. In postmenopausal women, the most common causes of vaginal bleeding are exogenous estrogens, atrophic vaginitis, endometrial lesions including cancer, and other tumors. Primary coagulation disorders account for up to 20% of menorrhagia in teenagers, and include von Willebrand disease, myeloproliferative disorders, and immune thrombocytopenia. Skin signs such as petechiae may be absent.

*Dysfunctional uterine bleeding* (DUB) may only be diagnosed after organic and systemic causes of bleeding have been excluded. DUB may be ovulatory or anovulatory. Typically, perimenarcheal and perimenopausal DUB is anovulatory. Patients with anovulatory cycles present with prolonged menses, irregular cycles, or intermenstrual bleeding. Usually the bleeding is painless and minimal, but severe bleeding can occur, resulting in anemia and iron depletion.

A pregnancy test must be obtained on all women of reproductive age to rule out pregnancy as a cause of bleeding. Other laboratory evaluation is guided by the history and physical examination. A CBC should be checked if signs of excessive bleeding or anemia are present. A PT or INR may identify a coagulopathy. Obtain thyroid function tests in patients with symptoms and signs of thyroid dysfunction. Ultrasonography is an important imaging modality to determine uterine size, characteristics of the endometrium and to detect structural abnormalities. Ultrasound may be deferred for outpatient evaluation in a stable patient.

## **Emergency Department Care and Disposition**



Most patients with vaginal bleeding are hemodynamically stable and need no acute intervention. Patients who are unstable with persistent bleeding require resuscitation: IV crystalloids, blood products and gynecologic consultation for urgent D and C.

1. For patients with severe, vaginal bleeding/dysfunctional bleeding, hormonal manipulation with estrogens may be tried. The efficacy of PO and IV formulations are similar. **Conjugated estrogen** (eg, Premarin<sup>®</sup>) 25 milligrams IV every 2 to 6 hours until bleeding slows or conjugated **estrogen** 2.5 milligrams PO 4 times a day may be started. **Medroxyprogesterone** (eg, Provera<sup>®</sup>) 10 milligrams daily is started when bleeding subsides; both drugs are continued for 7 to 10 days.
2. In stable patients *in whom the diagnosis of DUB is clear*, short-term hormonal therapy may be prescribed. Choices include the following:
  - a. Oral contraceptive regimen: **ethinyl estradiol** 35 micrograms and **norethindrone** 1 milligram, (eg, Ortho-Novum 1/35<sup>®</sup>) 4 tablets daily for 7 days. Alternatively, a taper may be given: 4 tablets for 2 days, 3 tablets for 2 days, 2 tablets for 2 days, and 1 tablet for 3 days.
  - b. **Medroxyprogesterone** 10 milligrams daily for 10 days.
  - c. **Tranexamic acid**, (eg, Lysteda<sup>®</sup>) an antifibrinolytic agent, 600 to 1300 milligrams every 8 hours for 3 days. Withdrawal bleeding may be heavy and typically occurs 3 to 10 days after the hormonal therapy has stopped.
3. If there is any concern for malignancy, hormonal therapy is best deferred until the patient is evaluated by gynecology and decision about a biopsy is made.
4. NSAIDs, such as **naproxen** 500 milligrams twice daily PO or **ibuprofen** 400 milligrams every 6 hours PO, may reduce bleeding.
5. Stable patients may be discharged home and instructed to follow-up with their gynecologic provider.

## PELVIC PAIN

Pelvic pain generally arises from gynecologic pathology but referred pain from extrapelvic conditions, such as inflammatory bowel disease, urinary tract infections or stones, diverticulitis, leaking abdominal aneurysm or appendicitis need to be considered. Pregnancy should be excluded in all women of reproductive age. Pelvic inflammatory disease is a common cause of pelvic pain and is discussed in [Chapter 64](#).

## Clinical Features

Pelvic pain may be acute or chronic, intermittent or continuous. Attention to the characteristics of the pain will aid in determining etiologies. Sudden onset of unilateral pain suggests an ovarian cyst, adnexal torsion, obstruction, or renal lithiasis. Gradual onset suggests an infectious process or slowly enlarging mass. Other attributes, such as the relationship of the pain with the menstrual cycle, aggravating and relieving factors, and associated urinary, GI, and systemic symptoms assist in developing the differential diagnosis.

An abdominal and gynecologic examination, including speculum examination, and a vaginoabdominal examination (bimanual) should be performed. A pregnancy test should be done to rule out pregnancy. Other testing, such as urinalysis, CBC, and ultrasound are guided by the history and physical examination.

## Diagnosis and Differential

### *Primary Dysmenorrhea*

Almost 90% of menstruating women experience dysmenorrhea at some point. Symptoms include mild to severe lower abdominal cramping during menses that diminishes as menstruation tapers. The pain can radiate to the thighs or lower back and may be accompanied by nausea and vomiting. Other gynecologic, urologic, or gastrointestinal conditions should be ruled out. NSAIDs, such as **naproxen** 500 milligrams twice daily PO or **ibuprofen** 400 milligrams every 6 hours PO, may alleviate symptoms.

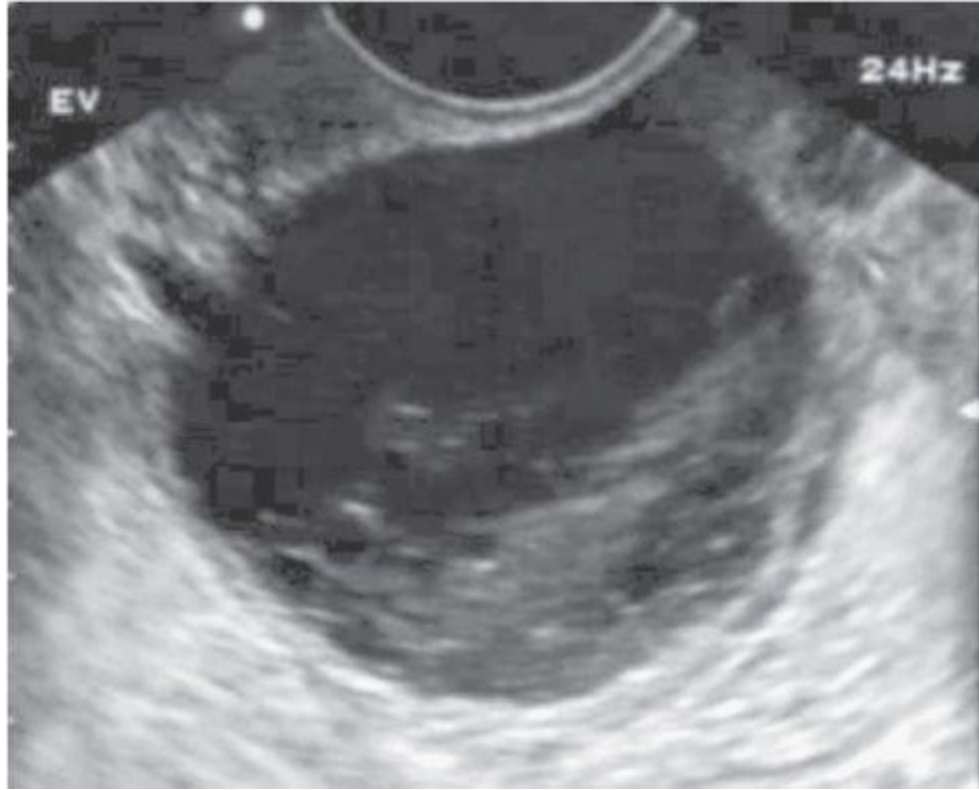
Further treatment, such as hormonal contraceptives, may be investigated at follow-up.

### *Mittelschmerz*

Mittelschmerz is a self-limited, unilateral dull, aching pain that occurs at mid cycle due to leakage of prostaglandin-containing follicular fluid. Patients frequently offer a history of previous similar pain. Treatment is symptomatic.

### *Ovarian Cysts*

Pain results from 2 mechanisms: leak of contents causing tissue irritation or mechanical pressure on adjacent organs. Sudden onset of pelvic pain in a patient with ovarian cysts suggests acute rupture. A ruptured cystmimic a ruptured ectopic pregnancy. Pelvic/transvaginal ultrasound is the diagnostic imaging modality of choice ([Fig. 58-1](#)). Patients with cyst rupture who present with hemoperitoneum and hypotension require emergent gynecological surgery intervention. Hemodynamically stable patients with pain from cyst leakage or rupture can be treated as outpatients with NSAIDs. Patients with unruptured cysts less than 5 cm in size frequently require no treatment as these cysts usually involute within 2 to 3 menstrual cycles. All patients should follow-up with their gynecologic provider for further evaluation.



**FIGURE 58-1.** A 4-cm ovarian cyst demonstrated by endovaginal ultrasonography. (Reproduced with permission from Ma OJ, Mateer JR: *Emergency Ultrasound*, 2nd ed. © 2008, McGraw-Hill Inc., New York.)

### *Ovarian Torsion*

Ovarian torsion results in the acute onset of severe adnexal pain from ischemia of the ovary. A history of intermittent pain, sometimes associated with exertion, preceding the severe symptoms may be obtained. Risk factors for torsion are pregnancy (enlarged corpus luteum), large ovarian cysts or tumors, and chemical induction of ovulation. Ultrasound with Doppler flow imaging is the diagnostic procedure of choice but is not 100% sensitive. Imaging early in the process may show congestion from venous outflow obstruction with preserved arterial flow and images obtained during a transient period of detorsion may appear normal. Analgesia, gynecologic

consultation, and preparation for surgery are warranted if the diagnosis is suspected.

### *Endometriosis*

Endometriosis results from endometrium-like stroma implanted outside of the uterus, most commonly the ovaries. Symptoms include recurrent pelvic pain associated with menstrual cycle—secondary dysmenorrhea and dyspareunia. Nonspecific pelvic pain on examination is the usual finding but if the ectopic tissue ruptures, more severe pain may be present. Ultrasound may show endometriomas. The definitive diagnosis is usually not made in the ED. Treatment consists of analgesics and gynecologic referral.

### *Leiomyomas*

Leiomyomas (uterine fibroids) are benign smooth muscle tumors, often multiple, seen most commonly in women in middle and later reproductive years. About 30% of women with leiomyomas will develop symptoms such as abnormal vaginal bleeding, dysmenorrhea, bloating, backache, urinary symptoms and dyspareunia. Severe pain can result with torsion of a pedunculated fibroid, or ischemia and infarction of a fibroid. Bimanual examination may demonstrate a mass or an enlarged uterus. Pelvic ultrasound is confirmatory. Treatment consists of NSAIDs or other analgesics for pain, hormonal manipulation for excessive bleeding, and referral to a gynecologist for definitive therapy.

## **Emergency Department Care and Disposition**

Most patients are ultimately discharged from the ED even though there may not be a specific diagnosis. Patients should receive detailed discharge instructions about signs and symptoms to expect and warnings of when to return and when to follow-up. Reevaluation in 12 to 24 hours can be scheduled if any concern persists. Analgesics, such as **NSAIDs**, provide

effective pain control for most outpatients, although some patients will require opioids, such as **oxycodone/acetaminophen (5/325)** 1 to 2 tablets every 4 to 6 hours PO for a few days.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 99, “Vaginal Bleeding in the Nonpregnant Patient” by Laurie J. Morrison and Julie M. Spence; and Chapter 100, “Abdominal Pain and Pelvic Pain in the Nonpregnant Female” by Thomas W. Lukens; and Chapter 74, “Acute Abdominal Pain” by Mary Claire O’Brien.

## **CHAPTER 59**

# **Ectopic Pregnancy and Emergencies in the First 20 Weeks of Pregnancy**

Robert Jones

## **ECTOPIC PREGNANCY**

Ectopic pregnancy (EP) is the leading cause of maternal death in the first trimester. Major risk factors include history of pelvic inflammatory disease, surgical procedures on the fallopian tubes, including tubal ligation, previous EP, diethylstilbestrol exposure, intrauterine device use, and assisted reproduction techniques. The most common extrauterine location is the fallopian tube. This diagnosis must be considered in every woman of childbearing age presenting with abdominal pain and/or vaginal bleeding.

### **Clinical Features**

The classic triad of abdominal pain, vaginal bleeding, and amenorrhea used to describe EP may be present, but many cases occur with more subtle findings. Presenting signs and symptoms may be different in ruptured versus nonruptured EP. Only 90% of women with EP complain of abdominal pain; 50% to 80% have vaginal bleeding; and only 70% give a history of amenorrhea. The pain described may be sudden, lateralized, extreme, or relatively minor and diffuse. The presence of hemoperitoneum causing diaphragmatic irritation may cause the pain to be referred to the shoulder or upper abdomen. Presenting vital signs may be entirely normal even with a ruptured ectopic pregnancy. There is poor correlation with the volume of hemoperitoneum and vital signs in EP. Relative bradycardia, as a consequence of vagal stimulation, may be present even in cases with rupture and hemoperitoneum. Physical examination findings are highly

variable. The abdominal examination may show signs of localizing or diffuse tenderness with or without peritoneal signs. The pelvic examination findings may be normal but more often shows cervical motion tenderness, adnexal tenderness with or without a mass, and possibly an enlarged uterus. Vaginal bleeding, ranging from spotting to heavier bleeding, is often present. Fetal heart tones may be heard in cases of EP beyond 12 weeks of gestation.

## **Diagnosis and Differential**

The definitive diagnosis of EP is made either by US or by direct visualization during surgery. The diagnosis of pregnancy is central to the diagnosis of possible ectopic pregnancy and needs to be confirmed first. Urine pregnancy testing (for urinary  $\beta$ -human chorionic gonadotropin [ $\beta$ -hCG]) is a qualitative screening test with a threshold for detection of  $> 20$  mIU/mL of  $\beta$ -hCG. Urine qualitative testing is 95% to 100% sensitive and specific as compared with serum testing. Dilute urine, particularly when  $\beta$ -hCG levels are  $<50$  mIU/mL, may result in a false-negative result. Qualitative serum testing for the diagnosis of pregnancy is virtually 100% sensitive for detecting  $\beta$ -hCG levels  $> 5$  mIU/mL and should be performed when the diagnosis of EP is considered but urine results are negative.

The primary goal of US in suspected EP is to determine if an IUP is present, since US cannot rule out the presence of EP. The transabdominal examination is usually performed first due to its wider field of view; the transvaginal examination is performed if the transabdominal examination is not diagnostic. When US reveals an unequivocal IUP and no other abnormalities, EP is effectively excluded unless the patient is at high risk for heterotopic pregnancy. Actual visualization of an EP with US occurs in a minority of cases. Sonographic findings of an empty uterus without an adnexal mass or free fluid in a woman with a positive pregnancy test result is considered indeterminate. In such situations, the findings must be evaluated in context with the patient's quantitative  $\beta$ -hCG level. A high  $\beta$ -hCG level ( $> 6000$  mIU/mL transabdominal or  $> 1500$  mIU/mL transvaginal) with an empty uterus is suggestive of EP. If the  $\beta$ -hCG is low ( $<1500$  mIU/ mL transvaginal), then the pregnancy may indeed be



intrauterine or ectopic but too small to be visualized on ultrasound or the patient may have already had a miscarriage. In this situation, repeat quantitative  $\beta$ -hCG testing in 2 days must be performed.  $\beta$ -hCG should increase at least 66% in that period; EP has a slower rate of increase. Since many EPs will have  $\beta$ -hCG levels  $<1500$  mIU/mL, quantitative  $\beta$ -hCG levels should not be used to determine the need for US imaging.

Differential diagnosis in the patient presenting with abdominal pain, vaginal bleeding, and early pregnancy includes threatened, incomplete, or missed abortion, recent elective abortion, implantation bleeding, molar pregnancy, heterotopic pregnancy, or corpus luteum cyst.

## **Emergency Department Care and Disposition**

Treatment of patients with suspected EP depends on the patient's vital signs, physical signs, and symptoms. Close communication with the obstetric-gynecologic consultant is essential.

1. For unstable patients, insert 2 large-bore intravenous lines and begin rapid infusion of crystalloid and/or packed red blood cells to maintain blood pressure.
2. Perform bedside urine pregnancy test.
3. Notify obstetric-gynecologic consultant immediately if the patient is unstable, even before laboratory and diagnostic tests are complete.
4. Draw blood for blood typing, and rhesus (Rh) factor determination (or cross-matching for the unstable patients), quantitative  $\beta$ -hCG determination (if indicated), and serum electrolyte determination, as required. Rh-negative women with EP should receive 50 micrograms of **anti-Rho (D) immunoglobulin**.
5. Reliable stable patients with indeterminate ultrasound results and a  $\beta$ -hCG level below 1000 mIU/mL can be discharged with ectopic precautions and follow-up in 2 days for repeat  $\beta$ -hCG determination and obstetric-gynecologic reevaluation.
6. Definitive treatment, as determined by the obstetric-gynecologic consultant, may involve laparoscopy, dilation and curettage, or medical management with methotrexate. Methotrexate therapy, even when used in

properly selected patients with EP, has a treatment failure rate in up to 36% of cases.

## **THREATENED ABORTION AND ABORTION**

According to the World Health Organization, between 20% and 40% of pregnancies will spontaneously abort. Approximately 75% of these spontaneous abortions will occur before 8 weeks of gestation.

### **Clinical Features**

Vaginal bleeding in the first 20 weeks, with a closed cervical os, benign examination, and no passage of tissue, is termed *threatened abortion*. A dilated cervix increases likelihood of abortion (*inevitable abortion*). *Incomplete abortion* is defined as partial passage of the conceptus and is more likely between 6 and 14 weeks of pregnancy. The patient may report passage of grayish white products of conception (POC), or the POC may be evident on pelvic examination. *Complete abortion* is passage of all fetal tissue before 20 weeks' gestation. All recovered POC should be sent for pathologic examination. *Missed abortion* is fetal death at less than 20 weeks without passage of any fetal tissue for 4 weeks after fetal death. *Septic abortion* implies evidence of infection during any stage of abortion, with signs and symptoms of pelvic pain, fever, cervical motion or uterine tenderness, or purulent or foul-smelling drainage.

### **Diagnosis and Differential**

Perform a pelvic examination and obtain a complete blood count (CBC), blood typing (and crossmatching for unstable patients) and rhesus (Rh) factor determination, urine pregnancy test, quantitative  $\beta$ -hCG, and urinalysis. The differential diagnosis includes ectopic pregnancy (EP), implantation bleeding, and gestational trophoblastic disease (GTD). Implantation bleeding, which is usually scant and painless, is a diagnosis of

exclusion. GTD is a proliferative disease of the trophoblast that includes complete hydatidiform mole, partial mole, trophoblastic tumor, and choriocarcinoma. Patients with GTD present with bleeding, an enlarged uterus out of proportion to menstrual age, and a significantly elevated  $\beta$ -hCG level. Also consider the possibility of a molar pregnancy in patients with hyperemesis gravidarum or pregnancy-induced hypertension before 24 weeks of gestation.

## **Emergency Department Care and Disposition**

Treatment of patients with suspected threatened abortion and abortion depends on the patient's vital signs, physical signs, and symptoms. Close communication with the obstetric-gynecologic consultant is essential.

1. Patients who are symptomatic or demonstrate signs of hemodynamic instability should receive supplemental oxygen, be placed on a cardiac monitor, and have 2 large-bore intravenous (IV) lines established. The gynecologist is consulted emergently in the unstable patient.
2. Initiate aggressive IV crystalloid and/or packed red blood cell infusion to help correct hypovolemia.
3. Rh-negative women with threatened abortion or abortion should receive **anti-Rho (D) immunoglobulin**. This is no uniform agreement on dosing. Dosing recommendations range from 50 micrograms to 300 micrograms in these patients.
4. US imaging should be performed when patient is stable.
5. Incomplete abortion or GTD requires dilation and curettage. The decision to proceed with medical treatment, such as misoprostol, 600 micrograms PO or surgical treatment, such as dilation and curettage, should be made in conjunction with the consulting obstetrician. GTD patients must receive close follow-up until quantitative  $\beta$ -hCG has returned to 0. Failure of the  $\beta$ -hCG to return to normal may indicate choriocarcinoma.
6. Septic abortion requires gynecologic consultation and broad-spectrum antibiotics such as **ampicillin/sulbactam** 3 grams IV or **clindamycin** 600 milligrams plus **gentamicin** 1 to 2 milligrams/kilogram IV.

7. Patients with threatened abortion or complete abortion may be discharged with close follow-up arranged. Discharge instructions include pelvic rest (no intercourse or tampons) and instructions to return for heavy bleeding, fever, or pain.

## **NAUSEA AND VOMITING OF PREGNANCY**

Nausea and vomiting of pregnancy generally are seen in the first 12 weeks and affect 60% and 80% of pregnant women, respectively. Cases can range from mild symptoms to hyperemesis gravidarum.

### **Clinical Features**

Findings on physical examination are usually normal except for signs of volume depletion. The presence of abdominal pain in nausea and vomiting of pregnancy or hyperemesis gravidarum is highly unusual and should suggest another diagnosis.

### **Diagnosis and Differential**

Diagnostic workup may include CBC, serum electrolytes, blood urea nitrogen, creatinine, and urinalysis. Differential diagnosis includes ruptured EP, cholelithiasis, cholecystitis, gastroenteritis, pancreatitis, appendicitis, hepatitis, peptic ulcer disease, pyelonephritis, fatty liver of pregnancy, and hemolysis-elevated liver enzymes—low platelets syndrome (known as HELLP syndrome).

### **Emergency Department Care and Disposition**

1. Rehydration should begin with IV fluid, 5% dextrose in normal saline, or 5% dextrose in lactate Ringer solution. Failure to include dextrose may result in prolonged ketosis.

2. Frequently used antiemetics are **metoclopramide** 10 milligrams IV, **promethazine** 25 milligrams IV (pregnancy class C, but widely used), and **ondansetron** 4 milligrams IV.
3. If the patient improves in the emergency department (urine ketones clearing and tolerating oral liquids), she may be discharged with a prescription for antiemetics. **Doxylamine** 25 milligrams **with pyridoxine** 25 milligrams PO every evening may be added as maintenance therapy for nausea and vomiting. Recent data suggests it does not represent an increase in fetal risk.
4. Admission is indicated for intractable vomiting, persistent ketonuria, electrolyte abnormalities, or weight loss greater than 10% of prepregnancy weight. Systemic steroids, such as **methylprednisolone**, 16 milligrams PO or IV every 8 hours for 3 days and tapered over 2 weeks to the lowest effective dose can be initiated in consultation with an obstetrician.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 101, "Ectopic Pregnancy and Emergencies in the First 20 Weeks of Pregnancy," by Richard S. Krause, David M. Janicke, and Rita K. Cydulka.

# CHAPTER 60

## Comorbid Diseases in Pregnancy

Abigail D. Hankin

Many medical conditions can present in the context of pregnancy, either as a preexisting condition or arising during pregnancy. This chapter will focus on conditions that require different management when encountered in the pregnant patient. Some disorders are covered in other chapters within this text, including hypertension ([Chapters 26](#) and [61](#)), HIV infection ([Chapter 62](#)), and cardiac rhythm disturbances ([Chapter 2](#)). Of note, when managing any acute complication of pregnancy, supplemental oxygen therapy and left lateral decubitus positioning are recommended to optimize oxygen delivery to the fetus.

### DIABETES

Diabetics are at increased risk for complications of pregnancy and acute complications of diabetes. Many patients with gestational diabetes (GD) are managed with diet alone, with a few requiring oral hypoglycemics (metformin or glyburide). Insulin therapy is necessary for some patients with GD and nearly all patients with Type I or II diabetes. Insulin requirements increase as a pregnancy progresses, from 0.7 to 1.0 units/kg/d at term.

Pregnant patients are at increased risk of diabetic ketoacidosis (DKA), especially those who are noncompliant, have hyperemesis, or are on sympathomimetic agents for tocolysis. Treatment of DKA is the same for pregnant and nonpregnant patients: isotonic fluid resuscitation to correct volume deficits, administration of continuous insulin, correction of electrolyte abnormalities (potassium and magnesium,) and treatment of the underlying cause (see [Chapter 129](#)).

Mild hypoglycemia is treated with a snack of milk and crackers, with care to avoid subsequent hyperglycemia. IV dextrose and/or IM glucagon is

used in the obtunded patient, followed by an IV 5% dextrose solution at 50 to 100 mL/h.

## **HYPERTHYROIDISM**

Hyperthyroidism in pregnancy increases the risk of preeclampsia, congenital anomalies, and neonatal morbidity. Clinical features may be subtle and may include hyperemesis gravidarum. Hyperthyroidism in pregnancy is treated with **propylthiouracil** (PTU), started at 50 milligrams PO 3 times daily (may be increased to 200 milligrams 3 times daily). Patients on PTU are at risk for purpuric rash and agranulocytosis. The use of radioactive iodine is contraindicated in pregnancy. Thyroid storm presents with fever, volume depletion, and cardiac decompensation. Management is similar to nonpregnant patients and includes PTU, sodium iodide, propranolol, cooling measures, and supportive care (see [Chapter 131](#)).

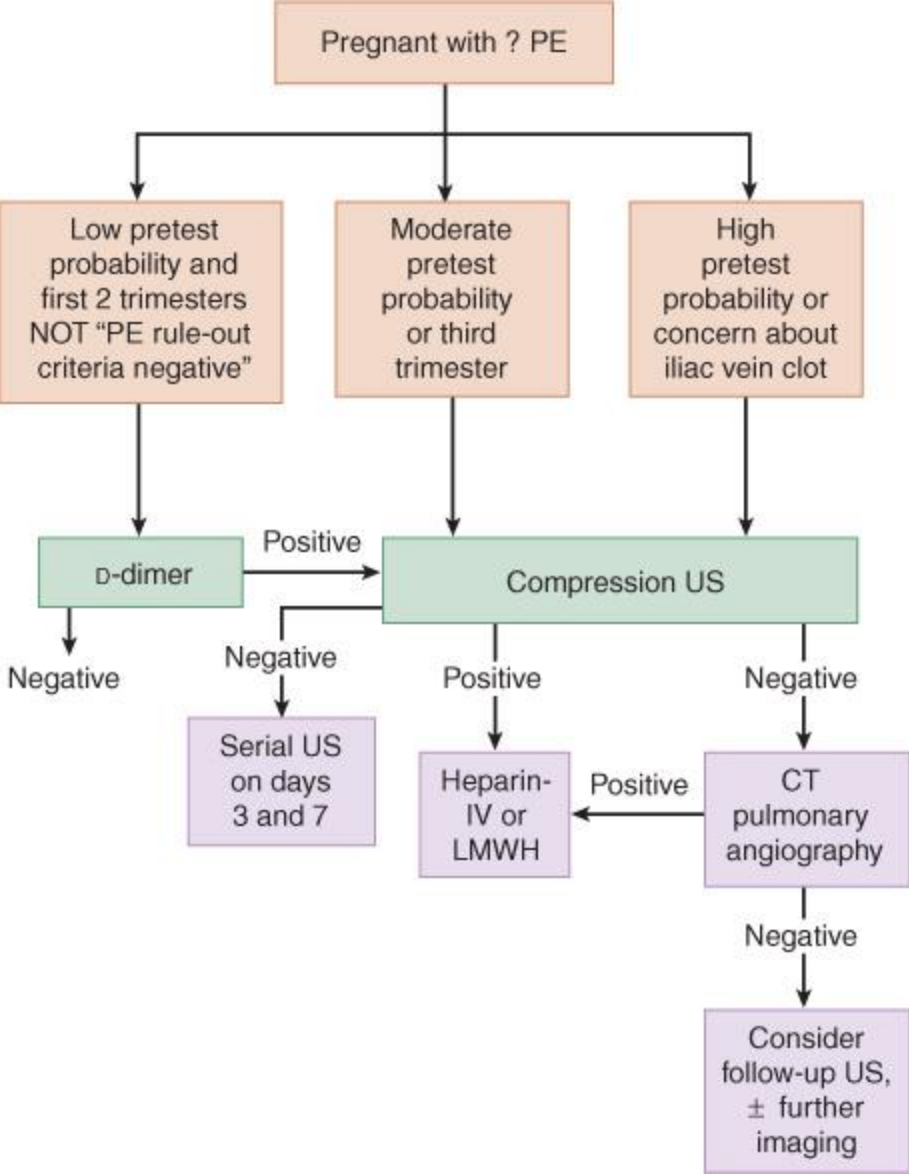
## **DYSRHYTHMIAS**

Dysrhythmias may be precipitated by pregnancy. Supraventricular tachycardias are treated with  $\beta$ -blockers, adenosine, verapamil, diltiazem, and digoxin at usual dosages. Patients with atrial fibrillation who require anticoagulation should be managed with unfractionated or low molecular weight heparin (LMWH). Electrical cardioversion may be used to treat tachyarrhythmias when indicated and have not been shown to be harmful to the fetus. Amiodarone should only be used to treat resistant, life-threatening dysrhythmias.

## **THROMBOEMBOLISM**

Factors associated with increased risk of thromboembolism include advanced maternal age, increasing parity, multiple gestations, operative

delivery, bedrest, and obesity. Symptoms of deep venous thrombosis (DVT) and pulmonary embolism (PE) may be mistaken for symptoms of normal pregnancy. Diagnosis of DVT is usually made by compression ultrasonography. CT angiography may be used to diagnose PE, and exposes the fetus to less radiation than ventilation-perfusion scan. MRI may also be used, but is less well studied in pregnancy. The utility of D-dimer testing in pregnancy is controversial. (See [Fig. 60-1](#) for a suggested diagnostic algorithm.) DVT and PE are treated with unfractionated or low molecular weight heparin at usual doses. Warfarin is contraindicated (see [Chapter 25](#)).





**FIGURE 60-1.** Algorithm for diagnosis of pulmonary embolism (PE) in pregnancy.

## **ASTHMA**

The presentation and management of acute asthma are similar in pregnant and nonpregnant patients. Acute therapy includes maintenance of oxygen saturation above 95%, administration of inhaled  $\beta_2$ -agonists, and early administration of systemic corticosteroids. Aerosolized ipratropium may be added in severely obstructed patients. Terbutaline sulfate, 0.25 milligram SC every 20 min, may also be used. Subcutaneous epinephrine should be avoided, if possible. Continuous fetal monitoring should be performed after 20 weeks' gestation. Criteria for intubation or admission are similar in pregnant and nonpregnant patients; standard agents for rapid sequence intubation are used.

## **CYSTITIS AND PYELONEPHRITIS**

Urinary tract infection is the most common bacterial infection in pregnancy. Clinical features are similar in pregnant and nonpregnant women. Simple cystitis may be treated for 7 to 10 days with **nitrofurantoin** sustained release 100 milligrams PO twice daily, **amoxicillin** 500 PO 3 times daily, or **cephalexin** 500 milligrams PO 4 times daily. Pregnant patients with pyelonephritis are treated aggressively because of increased risk of preterm labor and sepsis, and should be admitted for IV hydration and antibiotics (**cefazolin** 1 to 2 grams IV, or **ampicillin** 1 gram IV plus **gentamicin** 1 milligram/kilogram IV). Many providers continue antibiotic suppression for the remainder of the pregnancy. Quinolones are contraindicated in pregnancy. Avoid sulfonamides during the third trimester.

## **SICKLE CELL DISEASE**

Women with sickle cell disease are at higher risk for miscarriage, preterm labor, and vaso-occlusive crises. Clinical features, evaluation, and treatment are similar to nonpregnant patients. Management of vaso-occlusive crisis includes aggressive hydration, analgesia, and fetal monitoring (if the fetus is viable). Opiates can be used; NSAIDs should be avoided, particularly after 32 weeks' gestation. Blood transfusion should be considered when conservative measures have failed, or with severe anemia, preeclampsia, hypoxemia, acute chest syndrome, or a new-onset neurological event. Hydroxyurea should be discontinued in pregnancy.

## **HEADACHES**

Headaches can be a presenting symptom of a wide variety of benign and life-threatening disorders, including intracranial hemorrhage, mass, infection, and preeclampsia/eclampsia. Warning symptoms of potentially life-threatening disease include acute onset, postpartum headaches (increased risk of thrombosis), neurological deficits, and papilledema or retinal hemorrhages. If indicated, CT scan of the brain should be performed with appropriate shielding of the uterus. Migraine headaches should be treated with acetaminophen, narcotics, and antiemetics. Ergot alkaloids and triptans should not be used.

## **SEIZURE DISORDERS**

Seizure frequency may increase in patients with epilepsy due to altered drug pharmacokinetics. Acute seizure management is similar to that in nonpregnant patients (see [Chapter 146](#)). Status epilepticus with prolonged maternal hypoxia and acidosis has a high mortality rate for the mother and infant, and should be treated aggressively with early intubation and ventilation. Chronic seizure control is best managed by the patient's physician. Valproic acid, carbamazepine, lamotrigine, and phenytoin should be avoided due to teratogenicity.

## **SUBSTANCE ABUSE**

Cocaine use is associated with increased incidence of fetal death in utero, placental abruption, preterm labor, premature rupture of membranes, spontaneous abortion, intrauterine growth restriction, and fetal cerebral infarcts. Treatment of acute cocaine intoxication is unchanged in pregnancy. Opiate withdrawal in pregnant women is treated with methadone or buprenorphine in conjunction with an obstetrician and drug addiction specialist. Clonidine may be used acutely to blunt symptoms. Alcohol use contributes to increased rates of spontaneous abortion, low birthweight, preterm deliveries, and fetal alcohol syndrome. Benzodiazepines, a category D class, are best avoided in early pregnancy, but can be used in the context of severe alcohol withdrawal or severe cocaine intoxication.

## **DRUG USE IN PREGNANCY**

[Table 60-1](#) provides general recommendations regarding drug use in pregnancy. For any drug not listed in the table, check the manufacturer's recommendations before administration and check your hospital formulary or drug databases for the most current information.

Drug	Category <sup>†</sup>	Comments
<b>Antibiotic</b>		
Cephalosporins	B	
Penicillins	B	
Erythromycin estolate	B	Maternal hepatotoxicity
Azithromycin	B	
Clarithromycin	C	
Nitrofurantoin	B	
Clindamycin	B	
Metronidazole	B	Fetal facial defects (first trimester)
Ethambutol	B	
Quinolones	C, D	Toxicity to fetal cartilage
Aminoglycosides	C, D	Some of this class cause ototoxicity
Isoniazid	C	In TB, benefit may outweigh risk
Clavulanate combos	B	
Sulfonamides	C	Kernicterus (near term)
Tetracycline	D	Fetal bone/teeth anomalies
Trimethoprin	C	Folate antagoist (first trimester)
<b>Antivirals</b>		
Acyclovir	B	
Zidovudine	C	Recommended in HIV+ mothers

Antihypertensives		
α-Methyldopa	B	
β-Blockers	C	
Calcium channel blockers	C	
Hydralazine	C	Frequently used
ACE inhibitors	D, X	Discontinue use at first sign of pregnancy
Anticonvulsants		
Valproic acid	X	Neural tube defects
Phenytoin	C, D	Fetal anomalies (benefit may outweigh risk)
Carbamazepine	C, D	Fetal anomalies
Corticosteroids	C	May exacerbate hyperglycemia
Anticoagulants		
Heparin	C	
Enoxaparin	B	
Warfarin	X	Nasal hypoplasia, optic atrophy
Analgesics		
Acetaminophen	A	
Propoxyphene	C	Avoid close to term, neonatal withdrawal may occur
Opiates	C	
NSAIDs	B, C, D	Should not be used after 32 wk
Sumatriptan	C	
Ergot alkaloids	X	Potential for fetal death and abortion
Antiemetics		
Meclizine	B	

Diphenhydramine	B	
Metoclopramide	B	
Phenothiazine	C	Widely used
Ondansetron	B	
<b>Vaccines</b>		
Live vaccines (measles/ mumps/rubella)	X	Potential exists for fetal transmission
Inactivated viral vaccines—rabies, hepatitis B, influenza	C	Commonly given in pregnancy
Pneumococcal vaccine	C	Avoid using in first trimester
Tetanus and diphtheria	C	Commonly given in pregnancy
Tetanus immune globulin	C	Commonly given in pregnancy

\*Few studies have been done on pregnant women, therefore any medication should be used only if clearly necessary. Consult drug reference for more information.

!A = safe, B = presumed safe, C = possible adverse effect, D = unsafe but use may be justifiable in certain circumstances, X = contraindicated.

Key: ACE = angiotensin-converting enzyme, HIV = human immunodeficiency virus, NSAIDs = nonsteroidal anti-inflammatory drugs, TB = tuberculosis.

**TABLE 60-1 Use of Medications in Pregnancy\***

## DOMESTIC VIOLENCE

Between 4% to 20% of pregnant women are victims of intimate partner violence. They are at risk for placental abruption, uterine rupture, preterm labor, and fetal fractures. Injured pregnant women should be treated according to usual trauma protocols. Rh immunoglobulin, 300 micrograms intramuscularly, should be considered after blunt abdominal trauma in Rh-negative patients.

## DIAGNOSTIC IMAGING IN PREGNANCY

The threshold for teratogenesis from ionizing radiation is 10 rads, with the first trimester being the most vulnerable period. The effects of radiation exposure change with gestational age. The second to the eighth week post

conception is the period of organogenesis, when the fetus is most at risk for birth defects. Mental retardation and other problems may occur with significant x-ray exposure between 8 and 25 weeks. No single test exceeds the teratogenic threshold, but the effects are cumulative, and multiple tests may exceed the threshold. Ultrasound and magnetic resonance imaging have not been associated with teratogenic effects.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 102, "Comorbid Diseases in Pregnancy," by Pamela L. Dyne and Matthew A. Waxman.

## **CHAPTER 61**

# **Emergencies After 20 Weeks of Pregnancy and the Postpartum Period**

Howard Roemer

Continuous fetal monitoring is essential for the evaluation of a mother and fetus during the second half of a woman's pregnancy and should be implemented when assessing all the conditions discussed in this chapter.

## **VAGINAL BLEEDING DURING THE SECOND HALF OF PREGNANCY**

Abruptio placentae, placenta previa, and preterm labor are the most common causes of vaginal bleeding during the second half of pregnancy.

### **Clinical Features**

#### *Abruptio Placentae*

*Abruptio placentae*, the premature separation of the placenta from the uterine wall, must be considered in all pregnant females near term who present with painful vaginal bleeding. Clinical features include vaginal bleeding, abdominal pain, uterine tenderness, hypertonic contractions, increased uterine tone, fetal distress, and, in severe cases, disseminated intravascular coagulation (DIC), and fetal and/or maternal death. Vaginal bleeding may be mild or severe, depending on whether the area of abruption communicates to the cervical os. Abruption of greater than 50% of the placenta usually results in fetal demise.

#### *Placenta Previa*



*Placenta previa* is the implantation of the placenta over the cervical os. Clinical features include painless, bright red vaginal bleeding. The amount of bleeding is frequently large as opposed to normal “bloody show,” when a small amount of bright red blood and mucous are passed.

## **Diagnosis and Differential**

Transabdominal ultrasound should be obtained prior to performing speculum or digital pelvic examination to differentiate abruption placenta from placenta previa as it is contraindicated in previa. Ultrasound is very sensitive in detecting placenta previa but has limited sensitivity in diagnosing abruption placenta.

## **Emergency Department Care and Disposition**

1. Hemodynamic instability is managed with IV normal saline or leukoreduced packed red blood cells.
2. Obtain emergent obstetric consultation, CBC, type and crossmatching, baseline coagulation studies, electrolyte studies on all patients.
3. Obtain a DIC profile on patients with suspected abruptio placentae.
4. Give **Rh (D) immune globulin** 300 micrograms IM to Rh-negative patients.
5. Patients with abruptio placentae or placenta previa may need emergent caesarean delivery.
6. Tocolytics should not be used in patients with suspected abruption.

### ***Premature Rupture of Membranes***

*Premature rupture of membranes* (PROM) is rupture of membranes before the onset of labor. Clinical presentation is a rush of fluid or continuous leakage of fluid from the vagina. Diagnosis is confirmed by finding a pool of fluid in the posterior fornix with pH greater than 7.0 (dark blue on Nitrazine paper) and ferning pattern on smear. Sterile speculum

examination may be done; however, **digital pelvic examination should be avoided because it increases the rate of infection.** Tests for chlamydia, gonorrhea, bacterial vaginosis, and group B *Streptococcus* should be performed. Management of PROM depends on gestational age and maturity of the fetus, condition of the fetus, concern for infection, and presence of other complicating factors. An obstetrics consultation should be obtained to assist with treatment and admission decisions.

## **PRETERM LABOR**

### **Clinical Features and Diagnosis**

Preterm labor is defined as labor before 37 weeks' gestation. Clinical features include regular uterine contractions with effacement of the cervix. The diagnosis is made by fetal monitoring and sterile speculum examination. Cervical fluid should be examined for possible PROM. Only after premature rupture of membranes and placenta previa have been excluded, should digital examination be performed; use sterile gloves. Tests for chlamydia, gonorrhea, bacterial vaginosis, and group B streptococci are obtained. Ultrasound should be obtained for fetal age, weight, anatomy, amniotic fluid level, but obstetric consultation should not be delayed awaiting results.

### **Emergency Department Care and Disposition**

1. Mother and fetus are monitored.
2. An obstetrician is consulted for admission and decision regarding tocolytics.
3. If tocolytics are initiated, the mother should receive glucocorticoids to hasten fetal lung maturity. Dexamethasone 6 milligrams IM is commonly used.
4. Tocolytics are not used if abruptio placenta is suspected.
5. Gestational age younger than 34 weeks is associated with poorer outcomes; if possible, the patient should be transferred to a tertiary care

center with a high-risk intensive care unit.

## **HYPERTENSION, PREECLAMPSIA, AND RELATED DISORDERS**

Hypertension in pregnancy may be chronic due to preexisting hypertension, transient (gestational), or preeclampsia. Hypertension with pregnancy is associated with preeclampsia, eclampsia, HELLP (*Hemolysis, Elevated Liver enzymes, and Low Platelets*) syndrome, abruptio placenta, preterm birth, and low-birthweight infants.

### **Clinical Features**

Hypertension in pregnancy is defined as a systolic blood pressure  $\geq 140$  mm Hg or diastolic  $\geq 90$  mm Hg based on the average of at least two measurements in a woman who was normotensive prior to 20 weeks' of gestation. Preeclampsia is characterized by hypertension (greater than 140/90 mm Hg) and proteinuria ( $\geq$  milligrams/24 h) in patients 20 weeks' gestation until 4 to 6 weeks after delivery. Edema may or may not be present. Symptoms of severe preeclampsia reflect end-organ involvement and may include headache, visual disturbances, mental status changes, edema, oliguria, dyspnea (pulmonary edema) and abdominal pain. Eclampsia is preeclampsia with seizures. HELLP syndrome is probably a clinical variant of preeclampsia. Patients usually complain of abdominal pain, especially epigastric and right upper quadrant pain. Because the blood pressure is not always elevated, HELLP syndrome should be considered in the evaluation of all pregnant women ( $> 20$  weeks' gestation) with abdominal pain.

### **Diagnosis and Differential**

Preeclampsia is a clinical diagnosis. The following laboratory abnormalities may be seen in severe preeclampsia: anemia, thrombocytopenia, elevated creatinine, elevated liver enzymes, elevated LDH. The HELLP variant is diagnosed by laboratory tests: schistocytes on peripheral smear, platelet count lower than 150,000/mL, elevated aspartate aminotransferase and alanine aminotransferase levels. The differential diagnosis of preeclampsia includes worsening of preexisting hypertension, transient hypertension, renal disease, fatty liver disease of pregnancy, and coagulation disorders. A CT scan of the pelvis and abdomen should be done if concerns for subcapsular hematoma exist.

## **Emergency Department Care and Disposition**

1. Severe preeclampsia or eclampsia is treated with a **magnesium sulfate** loading dose of 4 to 6 grams over 20 min, followed by a maintenance infusion of 1 to 2 grams/h to prevent seizure. Serum magnesium and reflexes must be monitored.
2. Severe hypertension ( $> 160/110$  mm Hg) is treated with **labetalol** 20 milligrams IV initial bolus, followed by repeat boluses of 40 to 80 milligrams, if needed, to a maximum of 300 milligrams for blood pressure control or **hydralazine** 5.0 milligrams initially, followed by 5 to 10 milligrams IV every 10 min.
3. Consult an obstetrician emergently for severe preeclampsia or eclampsia.
4. All patients with a sustained systolic blood pressure  $\geq 140$  mm Hg or diastolic  $\geq 90$  mm Hg plus any symptoms of preeclampsia should be hospitalized.
5. Definitive treatment requires delivery of the fetus.

## **POSTPARTUM HEMORRHAGE**

The differential diagnosis of hemorrhage in the first postpartum day includes uterine atony (most common), uterine rupture, laceration of the lower genital tract, retained placental tissue, uterine inversion, and coagulopathy. After the first 24 hours, retained products of conception,

uterine polyps, or coagulopathy such as von Willebrand disease are more likely causes. An enlarged and “doughy” uterus suggests uterine atony; a vaginal mass suggests an inverted uterus. Bleeding despite good uterine tone and size may indicate retained products of conception or uterine rupture. The vagina and cervix must be inspected for lacerations. The first priority of ED management is stabilization of the patient with crystalloid IV fluids and/or leukoreduced packed red blood cells, if needed. CBC, clotting studies, and type and crossmatching must be obtained. Uterine atony is treated with uterine massage and **oxytocin** 20 units/L NS at 200 mL/h. Minor lacerations may be repaired in the ED. Extensive lacerations, retained products of conception, uterine inversion, or uterine rupture require emergency operative treatment by the obstetrician.

## **POSTPARTUM ENDOMETRITIS**

Postpartum endometritis is a polymicrobial infection with symptoms that usually begin several days after delivery. Clinical features include fever, lower abdominal pain, and foul-smelling lochia. Physical examination reveals uterine or cervical motion tenderness and discharge. CBC, urinalysis, and cervical cultures should be obtained. Admission for antibiotic treatment is indicated for most patients. Antibiotic regimens include **clindamycin** 450 to 900 milligrams IV every 8 hours plus **gentamicin** 1.5 milligrams/ kilogram IV every 8 hours or **cefoxitin** 1 to 2 gram IV every 6 hours.

## **MASTITIS**

Mastitis is cellulitis of the periglandular breast tissue. Clinical features include swelling, redness, and tender engorgement of the involved portion of the breast, with or without fever and chills. Milk stasis presents similarly, except it lacks erythema, fever, or chills. For cellulitis, initiate treatment with **dicloxacillin** 500 milligrams orally 4 times daily or **cephalexin** 500 milligrams orally 4 times daily. **Clindamycin** 300 milligrams PO every 6

hours may be used in patients with penicillin allergy or if concerns about MRSA exist. Oral analgesics may be needed. Patients should continue nursing on the affected breast, however, in cases of purulent discharge, the mother should pump and discard the milk rather than nurse. Differentiate mastitis from breast abscess using bedside ultrasound.

## **AMNIOTIC FLUID EMBOLISM**

Amniotic fluid embolism is a sudden, catastrophic illness with mortality rates of 60% to 80%. Clinical features include sudden cardiovascular collapse with hypoxemia, seizures (often a presenting sign), and DIC. Intensive management for cardiovascular collapse and DIC is indicated.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 104, “Emergencies after 20 Weeks of Pregnancy and the Postpartum Period,” by Michelle A. Echevarria and Gloria J. Kuhn.

# **CHAPTER 62**

## **Emergency Delivery**

Stacie Zelman

Precipitous delivery in an emergency setting can be a source of significant anxiety for an emergency physician. While a relatively uncommon occurrence, when a patient in active labor does present to the ED, careful preparation and education can help avoid serious complications of labor and delivery.

### **CLINICAL FEATURES**

Any pregnant woman who is beyond 20 weeks' gestation and appears to be in active labor should be evaluated expeditiously. Evaluation includes maternal vital signs, especially blood pressure, and fetal heart monitoring. A persistently slow fetal heart rate (fewer than 100 beats/ min) is an indicator of fetal distress. History includes time of onset of contractions, leakage of fluid, vaginal bleeding, and prenatal care. A focused physical examination should include an abdominal examination evaluating fundal height, abdominal or uterine tenderness, and fetal position. A bimanual or sterile speculum examination should be performed if no contraindications exist.

False labor is characterized by irregular, brief contractions usually confined to the lower abdomen. These contractions, commonly called Braxton-Hicks contractions, are irregular in intensity and duration. True labor is characterized by painful, regular contractions of steadily increasing intensity and duration leading to progressive cervical dilatation. True labor typically begins in the fundal region and upper abdomen and radiates into the pelvis and lower back.

### **DIAGNOSIS AND DIFFERENTIAL**

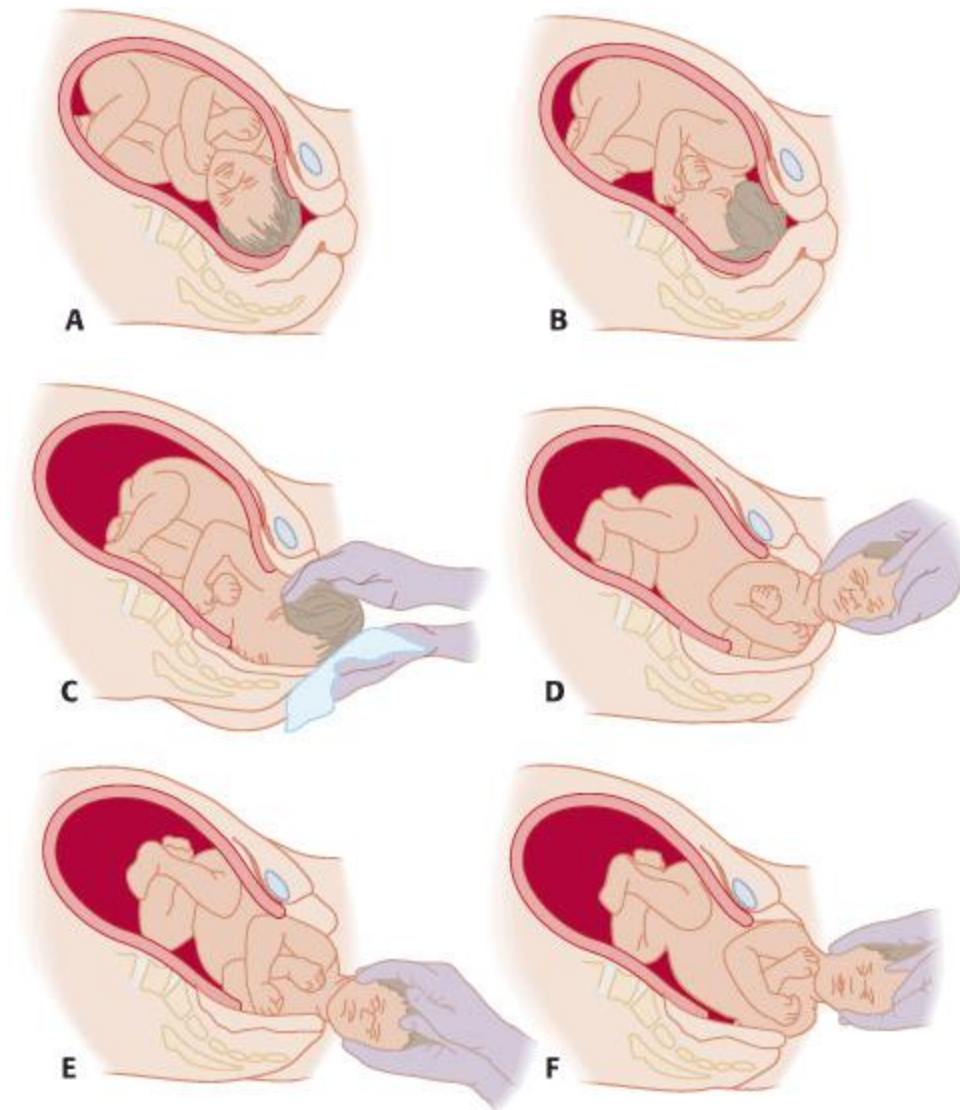
Patients without vaginal bleeding should be assessed with sterile speculum and bimanual examinations to evaluate the progression of labor, cervical dilation, and rupture of membranes. Patients with active vaginal bleeding require initial evaluation with ultrasound to rule out placenta previa. Abruptio placentae should be considered in patients with a tender, firm uterus, and marked bleeding (see [Chapter 60](#)). Spontaneous rupture of membranes typically occurs with a gush of clear or blood-tinged fluid. If ruptured membranes are suspected, a sterile speculum examination should be performed and amniotic fluid obtained from the fornix or vaginal vault. Amniotic fluid is alkaline, will stain Nitrazine paper dark blue and will “fern” if dried on a slide. The presence of meconium in amniotic fluid should be noted. Avoid digital examinations in the preterm patient in whom prolongation of gestation is desired as even one examination increases the chance of infection.

## **EMERGENCY DEPARTMENT CARE**

If the cervix is dilated in a woman experiencing active contractions, further transport, even short distances, may be hazardous. Preparations should be made for emergency delivery. Assess fetal position by physical examination, and confirm by ultrasound, if possible. Place the patient in the dorsal lithotomy position. Notify an obstetrician, if one is available.

**Emergency Delivery Procedure ([Fig. 62-1](#))**





**FIGURE 62-1.** Movements of normal delivery. Mechanism of labor and delivery for vertex presentations. A. Engagement, flexion, and descent. B. Internal rotation. C. Extension and delivery of the head. After delivery of the head, the neck is checked for encirclement by the umbilical cord. D. External rotation, bringing the thorax into the anteroposterior diameter of the pelvis. E. Delivery of the anterior shoulder. F. Delivery of the posterior shoulder. Note that after delivery, the head is supported and used to gently guide delivery of the shoulder. Traction should be minimized.

1. **Control of the delivery** of the neonate is the major challenge.

- a. As the **infant's head** emerges from the introitus, support the perineum with a sterile towel placed along the inferior portion of the perineum with one hand while supporting the fetal head with the other.
  - b. Exert mild counterpressure to prevent the rapid expulsion of the fetal head, which may lead to third- or fourth-degree perineal tears.
  - c. As the infant's head presents, use the inferior hand to control the fetal chin while keeping the superior hand on the crown of the head, supporting the delivery.
  - d. This controlled extension of the fetal head will aid in the atraumatic delivery.
  - e. Ask the mother to breathe through contractions rather than bearing down and attempting to push the baby out rapidly.
2. After delivery of the head, palpate the neck for the presence of a **nuchal cord**.
    - a. A nuchal cord is present in up to 35% of all cephalad-presenting deliveries.
    - b. If the cord is loose, reduce it over the infant's head; the delivery may then proceed as usual.
    - c. If the cord is tightly wound, clamp it in the most accessible area using two clamps in close proximity and cut to allow delivery of the infant.
  3. After delivery of the head, the head will reconstitute or turn to one side or the other.
    - a. As the head rotates, hands are placed on either side, providing gentle downward traction to deliver the anterior shoulder.
    - b. Then guide the fetus upward, delivering the posterior shoulder and allowing the remainder of the infant to be delivered.
  4. Place the posterior (left) hand underneath the infant's axilla before delivering the rest of the body. Use the anterior hand to grasp the infant's ankles and ensure a firm grip.
  5. Wrap the infant in a towel and stimulate it while drying.
  6. Double clamp the umbilical cord and cut with sterile scissors.
  7. Finish drying. Place the infant in a warm incubator, where postnatal care may be provided and **Apgar scores** calculated at 1 and 5 min after delivery.

- a. Scoring includes: General color, tone, heart rate, respiratory effort, reflexes.
8. Use of routine **episiotomy** for a normal spontaneous vaginal delivery is discouraged since it increases the incidence of third- and fourth-degree lacerations at time of delivery.
9. If an episiotomy is necessary (eg, with a breech presentation), it may be performed as follows:
  - a. Inject a solution of 5 to 10 mL of 1% **lidocaine** with a small-gauge needle into the posterior fourchette and perineum.
  - b. While protecting the infant's head, make a 2- to 3-cm cut with scissors to extend the vaginal opening.
  - c. Support the incision with manual pressure from below, taking care not to allow the incision to extend into the rectum.

## **CORD PROLAPSE**

1. If bimanual examination shows a **palpable, pulsating cord**:
  - a. Do not remove the examining hand; use the hand to elevate the presenting fetal part to reduce compression of the cord.
  - b. Immediate obstetric assistance is necessary, as a **cesarean section is indicated**.
  - c. Keep the examining hand in the vagina while the patient is transported and prepped for surgery to prevent further compression of the cord by the fetal head. Do not attempt to reduce cord.

## **SHOULDER DYSTOCIA**

1. Is first recognized after the delivery of the fetal head, when routine downward traction is insufficient to deliver the anterior shoulder. The anterior shoulder is trapped behind the pubic symphysis.
2. After delivery of the infant's head, the head retracts tightly against the perineum ("Turtle sign").

3. Upon recognizing shoulder dystocia, suction the infant's nose and mouth and call for assistance to position the mother in the extreme lithotomy position, with legs sharply flexed up to the abdomen (McRoberts maneuver) and held by the mother or an assistant.
4. Drain the bladder.
5. A generous episiotomy also may facilitate delivery.
6. Next, an assistant should apply suprapubic pressure to disimpact the anterior shoulder from the pubic symphysis.
7. Do not apply fundal pressure because this will further force the shoulder against the pelvic rim.
8. A Woods Corkscrew maneuver may be attempted—place a hand behind the posterior shoulder of the infant, and rotate the shoulder girdle 180°.

## **BREECH PRESENTATION**

1. The primary concern with breech presentation is head entrapment.
2. Breech presentations may be classified as **frank, complete, incomplete, or footling**.
3. In any breech delivery, immediate obstetric consultation should be requested.
4. **Frank and complete breech presentations:**
  - a. Serve as a dilating wedge nearly as well as the fetal head, and delivery may proceed in an uncomplicated fashion.
  - b. Main point is to allow the delivery to progress spontaneously. This lets the presenting portion of the fetus to dilate the cervix maximally.
  - c. Consult obstetrical texts for a detailed description of maneuvers for breech delivery.
5. **Footling and incomplete breech positions:** are not considered safe for vaginal delivery because of the possibility of cord prolapse or incomplete dilatation of the cervix.

## **POSTPARTUM CARE**

1. The placenta should be allowed to separate spontaneously and assisted with gentle traction.
2. Aggressive traction on the cord risks uterine inversion, tearing of the cord, or disruption of the placenta, which can result in severe vaginal bleeding.
3. After removal of the placenta, gently massage the uterus should to promote contraction.
4. Infuse **oxytocin** 10 to 40 U/1000 mL NS at a moderate rate to maintain uterine contraction. Oxytocin may also be given as 10 U IM.
5. Episiotomy or laceration repair may be delayed until an experienced obstetrician is able to close the laceration and inspect the patient for fourth-degree (rectovaginal) tears.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 105, “Emergency Delivery,” by Michael J. VanRooyen and Jennifer A. Scott.

# CHAPTER 63

## Vulvovaginitis

Stacie Zelman

Causes of vulvovaginitis include infections, irritants, allergies, reaction to foreign bodies, and atrophy. The normal vaginal flora helps maintain an acidic pH between 3.8 and 4.5, which decreases pathogen growth.

## BACTERIAL VAGINOSIS

Bacterial vaginosis (BV) is the most common cause of malodorous vaginal discharge. However, many infected women are asymptomatic. BV occurs when vaginal lactobacilli are replaced by anaerobes, *Gardnerella vaginalis*, and *Mycoplasma hominis*.

### Clinical Features

The most common symptom is malodorous or “fishy smelling” vaginal discharge. Vaginal irritation, excoriation, and fissures are less common. Examination findings range from mild vaginal redness to a frothy gray-white or yellow discharge.

### Diagnosis and Differential

The diagnosis can be made if 3 of the following 4 criteria are present: (a) vaginal discharge, (b) vaginal pH greater than 4.5, (c) positive amine test (fishy odor when 10% KOH is added to the discharge), (d) clue cells seen on saline wet preparation. Often, the diagnosis of BV is suspected from the history.

## **Emergency Department Care and Disposition**

Treat with **metronidazole** 500 milligrams PO twice daily for 7 days. **Clindamycin** 300 milligrams PO twice daily for 7 days is an alternative. Treatment is not recommended for male partners or asymptomatic women. All patients treated with metronidazole should refrain from alcohol use during treatment and for 24 hours after ending treatment, to avoid a disulfiram-like reaction.

Pregnant women at high risk of preterm labor should be considered for treatment, and all symptomatic pregnant women should be treated. The recommended treatment in pregnancy is **metronidazole** 250 milligrams PO twice daily for 7 days. Routine treatment of asymptomatic pregnant women with BV is not recommended.

## **CANDIDA VAGINITIS**

*Candida albicans* is a common cause of vaginitis. Conditions that promote *Candida* vaginitis include systemic antibiotics, diabetes, pregnancy, and birth control pills. Incidence is decreased in postmenopausal patients. Candidiasis is not considered a sexually transmitted disease, though it can be transmitted sexually.

### **Clinical Features**

The most common symptom of *Candida* vaginitis is pruritus. Other symptoms include vaginal discharge, external dysuria, and dyspareunia. Signs include vulvar and vaginal edema, erythema, and a thick “cottage cheese” discharge.

### **Diagnosis and Differential**

Examine vaginal secretions microscopically in a few drops of saline solution or make a KOH preparation. Ten percent KOH dissolves vaginal epithelial cells, leaving yeast buds and pseudohyphae intact and easier to see. The sensitivity of the KOH technique is 80%, with a specificity approaching 100%.

## **Emergency Department Care and Disposition**

Almost all topically applied azoles are equally efficacious. Treatment options include **clotrimazole** 100 milligram intravaginal tablet (2 tablets for 3 days), **butoconazole** 2% cream 1 vaginal applicator daily for 3 days, **miconazole** 200 milligrams vaginal suppository for 3 days. Pregnant patients are treated with intravaginal agents for 7 days. Single-dose treatment with **fluconazole**, 150 milligrams PO, is as effective as topical treatments but oral fluconazole may not be used in pregnancy. For nonpregnant patients with complicated candidiasis, **fluconazole** 150 milligrams PO is given on days 1 and 3.

## **TRICHOMONAS VAGINITIS**

Trichomoniasis is a common sexually transmitted disease caused by the protozoan *T vaginalis*.

### **Clinical Features**

Presenting symptoms include a frothy, malodorous vaginal discharge, vaginal erythema and vulvar irritation. However, up to 50% of women harboring the organism are asymptomatic.

### **Diagnosis and Differential**



Saline wet prep shows motile, pear-shaped, flagellated trichomonads. Microscopy should be performed within 20 min of obtaining the sample or the organisms may lose motility. The sensitivity of microscopy is 60% to 70%. The sensitivity of culture is 95% but results are not readily available in the ED.

## **Emergency Department Care and Disposition**

The treatment of choice for trichomoniasis is **metronidazole** 2 grams single oral dose or **tinidazole** 2 grams single oral dose. **Metronidazole** 500 milligrams twice daily for 7 days is recommended for patients who fail single-dose therapy. Metronidazole gel is much less efficacious and thus not recommended for use. Male partners should be treated to avoid retransmission of disease. Patients should be advised to abstain from alcohol intake until 24 hours after completing metronidazole and 72 hours after completing tinidazole therapy. They should also be counseled to abstain from sexual activity until treatment course is completed and they are asymptomatic.

## **CONTACT VULVOVAGINITIS**

Common causes of contact vulvovaginitis include douches, soaps, bubble baths, deodorants, perfumes, feminine hygiene products, topical antibiotics, and tight undergarments. Patients complain of perineal burning, itching, swelling, and often dysuria. The examination shows a red and swollen vulvovaginal area. In severe cases, vesicles and ulceration may be present. Vaginal pH changes may promote overgrowth of *Candida*, obscuring the primary problem.

Try to identify the precipitating agent and rule out infectious causes. Most cases resolve spontaneously when the precipitant is withdrawn. For more severe reactions, cool sitz baths, compresses with Burow's solution, and topical corticosteroids may help. Oral antihistamines are drying but may be helpful if a true allergy is identified. Concomitant *Candida* infections should be treated as previously discussed.

## **VAGINAL FOREIGN BODIES**

In younger girls, common items include toilet paper, toys, and small household objects. Later, a forgotten or irretrievable tampon or items used for sexual stimulation are more often seen. Patients present with a foul-smelling or bloody discharge. Removal of the object is usually curative.

## **ATROPHIC VAGINITIS**

After menopause, the lack of estrogen stimulation leads to vaginal mucosal atrophy. The epithelium becomes pale, thin, and less resistant to minor trauma or infection. Bleeding can occur. The vaginal pH also increases, and subsequent changes in the vaginal flora can predispose to bacterial infection with purulent discharge. Treatment consists primarily of topical estrogen creams. Estrogen creams should not be prescribed in the emergency department for women with prior reproductive tract cancer or postmenopausal bleeding.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 106, "Vulvovaginitis," by Gloria J. Kuhn and Robert P. Wahl.

## CHAPTER 64

# Pelvic Inflammatory Disease and Tubo-Ovarian Abscess

Paul W. Beck

Pelvic inflammatory disease (PID) comprises a spectrum of infections of the female upper reproductive tract. Most cases originate as lower genital tract infections that ascend to cause salpingitis, endometritis, myometritis, parametritis, tubo-ovarian abscess (TOA), perihepatitis, or focal pelvic peritonitis. *Neisseria gonorrhoea* or *Chlamydia trachomatis* are common pathogens; however, 30% to 40% of infections are polymicrobial. Risk factors include multiple sexual partners, sexual abuse, adolescence, presence of other sexually transmitted diseases, douching, lack of condom use, delay in seeking care, and intrauterine device use. PID occurs less commonly in pregnancy, but first trimester infections can lead to fetal loss. Long-term sequelae include ectopic pregnancy, infertility, and chronic pain.

## CLINICAL FEATURES

Lower abdominal pain is usually present. Other symptoms include vaginal discharge, vaginal bleeding, dyspareunia, urinary discomfort, fever, nausea, and vomiting. Peritoneal signs may be present. Occasionally, symptoms are minimal. An exquisitely tender unilateral mass may suggest TOA. The presence of right upper quadrant tenderness, especially with associated jaundice, may indicate Fitz-Hugh-Curtis syndrome (perihepatitis).

## DIAGNOSIS AND DIFFERENTIAL

The clinical diagnosis of PID is imprecise. Diagnostic criteria for empiric treatment are listed in [Table 64-1](#). Obtain a pregnancy test, wet prep, and endocervical swabs for gonorrhea and chlamydia. A pelvic ultrasound will

help detect TOA and may differentiate PID from surgical conditions such as appendicitis, cholecystitis, and ovarian torsion. The differential diagnosis includes gastroenteritis, diverticulitis, ectopic pregnancy, spontaneous or septic abortion, ovarian cyst, pyelonephritis, and renal colic.

<p><b>1. Minimal criteria for diagnosis and empiric treatment:</b>  Lower abdominal or pelvic pain without another identifiable cause PLUS  Uterine tenderness or  Adnexal tenderness or  Cervical motion tenderness</p>
<p><b>2. Additional criteria improving diagnostic specificity:</b>  Oral temperature &gt;101°F (38.3°C)  Abnormal cervical or vaginal mucopurulent discharge  Abundant numbers of WBC on saline microscopy of vaginal fluid  Elevated erythrocyte sedimentation rate  Elevated C-reactive protein  Laboratory evidence of cervical infection with <i>Neisseria gonorrhoea</i> or <i>Chlamydia trachomatis</i> (ie, culture or DNA probe techniques)</p>
<p><b>3. Most specific criteria:</b>  Transvaginal ultrasound (or MRI) showing thickened, fluid-filled tubes with or without free pelvic fluid or tuboovarian complex  Laparoscopic confirmation*  Endometrial biopsy showing endometritis*</p>

Key: MRI = magnetic resonance imaging, PID = pelvic inflammatory disease.

\*These diagnostic procedures are not usually performed during ED visit.

Source: Adapted from *MMWR*. 2010;59 (RR-12):65.

**TABLE 64-1** Diagnostic Criteria for PID

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Treatment guidelines of the Centers for Disease and Control Prevention are outlined in [Tables 64-2](#) and [64-3](#). Patients with mild to moderate symptoms may be treated with oral therapy as outpatients. Adequate analgesia and hydration should be provided.

**Recommended**

1. Cefotetan 2 grams IV q12h *or* ceftioxin 2 grams IV q6h  
Plus  
Doxycycline 100 milligrams IV or PO q12h
2. Clindamycin 900 milligrams IV q8h  
Plus  
Gentamicin 2 milligrams/kilogram IV loading dose followed by 1.5 milligrams/kilogram q8h

**Alternative**

- Ampicillin/Sulbactam 3 grams IV q6h  
Plus  
Doxycycline 100 milligrams IV/PO q12h

Key: IV = intravenously, PO = orally

Source: Adapted from *MMWR*. 2010; 59 (RR-12):65.

**TABLE 64-2** Parenteral Treatment Regimens for Pelvic Inflammatory Disease

1. Ceftriaxone 250 milligrams IM once; *or* ceftioxin 2 grams IM once *and* probenecid 1 gram PO × 1 (or other parenteral third generation cephalosporin)  
+  
Doxycycline 100 milligrams PO bid for 14 d  
±  
Metronidazole 500 milligrams PO bid for 14 d
2. Alternative oral regimens, if parenteral cephalosporin therapy is not feasible *and* community prevalence of fluoroquinolone resistance is low:  
Levaquin 500 milligrams PO daily or Ofloxacin 400 milligrams PO bid for 14 d  
±  
Metronidazole 500 milligrams PO bid for 14 d

Key: bid = twice daily, IM = intramuscularly, PO = orally.

Source: Adapted from 2010 Centers for Disease Control and Prevention Guidelines <http://www.cdc.gov/std/treatment/2010/pid.htm>

**TABLE 64-3** Oral and Outpatient Treatment Regimens for Pelvic Inflammatory Disease

2. Suggested criteria for admission include: toxic appearance, inability to tolerate oral medication, nausea and vomiting, inability to exclude alternative diagnoses or surgical emergency, failure to respond to outpatient management, pregnancy, immunosuppression, concern for noncompliance, and TOA.

3. Sixty percent to 80% of TOAs respond to antibiotics alone; the remainder require drainage.
4. Outpatients should be reevaluated within 72 hours.
5. Provide preventative counseling and test or refer for HIV testing. The patient and the sexual partner(s) must complete the full treatment course before resuming sexual activity to prevent reinfection.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 109, "Pelvic Inflammatory Disease," by Suzanne M. Shepherd, William Shoff and Amy J. Behrman.

# CHAPTER 65

## Complications of Gynecologic Procedures

Anitha Mathew

The most common reasons for emergency department visits during the postoperative period after gynecologic procedures are pain, fever, and vaginal bleeding. A focused but thorough evaluation should be performed, including sterile speculum and bimanual examination and consultation with the gynecologist who performed the procedure is indicated. (Complications common to gynecologic and abdominal surgeries are covered in [Chapter 49](#).)

### COMPLICATIONS OF ENDOSCOPIC PROCEDURES

#### Laparoscopy

The major complications associated with laparoscopy are thermal injury of the bowel, viscus perforation, hemorrhage, vascular injury, ureteral or bladder injuries, incisional hernia, and wound dehiscence. Patients with thermal injury may not develop symptoms for several days to weeks postoperatively and typically present with bilateral lower abdominal pain, fever, elevated white blood cell count, and peritonitis. X-rays can show an ileus or free air under the diaphragm. Patients with greater than expected pain after laparoscopy have a bowel injury until proven otherwise, and early gynecology consultation should be obtained.

#### Hysteroscopy

Complications of hysteroscopy include cervical and uterine perforation, postoperative bleeding, fluid overload from absorption of distention media, infection. Consultation with a gynecologist is required. Gas embolism and anesthesia reaction are intraoperative complications. Postoperative bleeding requires hemodynamic stabilization; the gynecologist may choose to insert a Foley or balloon catheter into the uterus to tamponade the bleeding. Vasopressin or misoprostol are alternative treatments. Patients with uterine perforation who present with peritoneal signs require surgical exploration. Patients with fluid overload are likely to be hyponatremic. Infection as a result of hysteroscopy is uncommon and is treated with antibiotics.

## **OTHER COMPLICATIONS OF GYNECOLOGIC PROCEDURES**

### **Vaginal Cuff Cellulitis**

*Cuff cellulitis*, a common complication after hysterectomy, is an infection of the contiguous retroperitoneal space immediately above the vaginal apex and the surrounding soft tissue. Patients typically present between postoperative days 3 and 5 with fever, abdominal pain, pelvic pain, back pain, and abnormal vaginal discharge. Cuff tenderness and induration are prominent during the bimanual examination, and a vaginal cuff abscess may be palpable. Treat with broad spectrum antibiotics. One suggested regimen is **ampicillin**, 2 grams IV every 6 hours plus **gentamicin**, 1 milligram/kilogram IV loading dose followed by 1 milligram/kilogram IV every 8 hours, plus **clindamycin**, 900 milligrams IV every 6 hours. Admit for continuation of antibiotics and possible abscess drainage.

### **Postoperative Ovarian Abscess**

Patients with ovarian abscesses typically present shortly after hospital discharge with fever and abdominal and pelvic pain. A CT scan or US can help identify and localize the abscess. A sudden increase in pain can signal



possible abscess rupture, which requires emergent laparotomy. Patients with ovarian abscesses should be admitted for IV antibiotics and possible drainage.

## **Ureteral Injury**

Ureteral injury can occur during abdominal hysterectomy, resulting from crushing, transecting, or ligating trauma. These patients present soon after surgery with flank pain, fever, and costovertebral angle tenderness. The work-up includes a urinalysis and a CT scan with IV contrast or an IVP to evaluate for obstruction. These patients should be admitted for ureteral catheterization and possible repair.

## **Vesicovaginal Fistula**

Vesicovaginal fistulas can occur after abdominal hysterectomy. Patients typically present 10 to 14 days following surgery with a watery vaginal discharge, and should receive prompt gynecologic consultation. Patients are treated with Foley catheter drainage after the diagnosis is confirmed.

## **Postconization Bleeding**

The most common complication associated with loop electrocautery, laser ablation, and cold-knife conization of the cervix is bleeding, which can be rapid and excessive. Delayed hemorrhage can occur 1 to 2 weeks postoperatively. Direct visualization of the bleeding site is required. Applying Monsel solution, direct pressure for 5 min with a large cotton swab, or cauterization with silver nitrate is a reasonable first step. If unsuccessful, the bleeding site may be better visualized and treated in the OR.

## **Septic Pelvic Thrombophlebitis**

Patients with ovarian vein thrombosis present within a week after delivery or surgery with fever, tachycardia, GI distress, and unilateral abdominal pain. Patients with deep septic pelvic thrombophlebitis present a few days after delivery or surgery with spiking fevers that are unresponsive to antibiotics; these patients may have abdominal pain. Ultrasound, CT and MRI are frequently nondiagnostic making this a diagnosis of exclusion. Patients are admitted for anticoagulation (heparin or enoxaparin) and IV antibiotics, such as **ampicillin/sulbactam** 3 grams IV every 6 hours, **piperacillin/ tazobactam** 4.5 grams IV every 8 hours, or **ticarcillin/clavulonate** 3.1 grams IV every 4 hours. Monotherapy with a carbapenem, such as imipenam 500 milligrams every 6 hours, may be used for patients with  $\beta$ -lactam intolerance.

## **Induced Abortion**

Complications associated with induced abortion include uterine perforation, cervical lacerations, retained products of conception, and postabortal endometritis ([Table 65-1](#)). Patients with retained products of conception usually present with excessive bleeding and abdominal pain. Pelvic examination reveals an enlarged and tender uterus with an open cervical os. A pelvic ultrasound should be obtained to confirm the diagnosis. Treatment is dilatation and curettage. Endometritis can occur with or without retained products of conception and is treated with antibiotics, as previously discussed in Vaginal Cuff Cellulitis. Women who are Rh negative require **Rh<sub>0</sub> immunoglobulin**, 300 micrograms IM, after spontaneous or induced abortion.

Timing	Complication	Possible Etiologies
Immediate complications: within 24 h postprocedure	Bleeding, pain	Uterine perforation, cervical lacerations
Delayed complications: between 24 h and 4 wk postprocedure	Bleeding	Retained products of conception, postabortive endometritis
Late complications: >4 wk postprocedure	Amenorrhea, psychological problems, Rh isoimmunization	—

**TABLE 65-1** Complications Associated with Induced Abortion

## Assisted Reproductive Technology

Complications related to ultrasound-guided aspiration of oocytes include ovarian hyperstimulation syndrome, pelvic infection, intraperitoneal bleeding, and adnexal torsion. Ovarian hyperstimulation syndrome can be a life-threatening complication of assisted reproduction. Mild cases present with abdominal distention, ovarian enlargement, and weight gain. In severe cases, patients have rapid weight gain, tense ascites from third spacing of fluid into the abdomen, pleural effusions, hemodynamic instability, oliguria, electrolyte abnormalities, and increased coagulability. Bimanual pelvic exam is contraindicated to avoid rupturing the ovaries. Initiate IV volume replacement, obtain CBC, electrolytes, liver function tests and coagulation studies and consult with gynecology for admission.

## Postembolization Syndrome

*Postembolization syndrome* consists of postprocedure pelvic pain, nausea, vomiting and fever lasting 2 to 10 days caused by myometrial and fibroid ischemia after uterine fibroid embolization. Evaluate patients for other causes of fever and provide pain control. Patients with inadequate pain control or those in whom an infection is present may require admission.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 109, “Complications of Gynecologic Procedures,” by Michael A. Silverman.

## **SECTION 9 Pediatrics**

## **CHAPTER 66**

# **Fever and Serious Bacterial Illness in Children**

Milan D. Nadkarni

## **FEVER**

Fever is the most common chief complaint presenting to an emergency department and accounts for 30% of outpatient visits each year. Early studies suggested that infants younger than 3 months were at high risk of a serious bacterial illness (SBI), which included sepsis, pyelonephritis, pneumonia, and meningitis. Current practice guidelines vary in their cut-offs for evaluation and treatment strategies. Neonates are clearly at the highest risk, while infants in their second and third months of life gradually transition to the lower risk profile of older infants and children. The incidence of bacteremia falls from around 10% among febrile neonates to approximately 0.2% in immunized infants and children older than 4 months; meningitis risk decreases from about 1% in the first month of life to <0.1% later in infancy; the risk for pyelonephritis remains relatively constant among young girls with fever, and gradually decreases among boys over the first year of life. The individual practitioner must weigh these risks against the invasiveness of their ED evaluation and make shared decisions with the family on the best approach.

### **Clinical Features**

In the neonate or infant <2 to 3 months of age, the threshold for concerning fever is 38°C (100.4°F); in infants and children 3 to 36 months old, the threshold is 39°C (102.2°F). In general, higher temperatures are associated with a higher incidence of serious bacterial illness.

Young infants are especially problematic in assessing severity of illness. Immature development and immature immunity make reliable examination findings difficult. Persistent crying, inability to console, poor feeding, or temperature instability may be the only findings suggestive of an SBI.

## **Diagnosis and Differential**

### *Infants up to 3 Months*

History and physical examination are rarely helpful in diagnosing or excluding SBI in this age group as symptoms are typically vague, and physical exam findings are unreliable: meningismus is present in <15% of bacterial meningitis; rales may not be appreciated in the absence of ability to generate negative inspiratory forces; and bacteremia can occur in the well-appearing infant. A history of cough, tachypnea, or hypoxia (by pulse oximetry), however, should alert the examiner to a possible lower respiratory tract infection and prompt chest radiograph.

**The safest course for 0 to 28 day old infants is full sepsis testing, admission, and empiric antibiotic treatment.** Antibiotic coverage in this age group includes ampicillin for *Listeria monocytogenes* (see [Table 66-1](#)). Sepsis testing includes complete blood count (CBC), blood culture, urinalysis and urine culture, chest radiograph, and lumbar puncture.

Age Group	Bacteremia	Sepsis*	Meningitis
Neonates (age 0 to 28 days)	Not applicable	Ampicillin, 100 milligrams/kilogram <i>plus</i> Cefotaxime, 50 milligrams/kilogram <sup>†</sup>	Ampicillin, 100 milligrams/kilogram <i>plus</i> Cefotaxime, 50 milligrams/kilogram <sup>†</sup>
Young infants (29 to 90 days)	Ceftriaxone, 50 milligrams/kilogram	Ampicillin, 100 milligrams/kilogram <i>plus</i> Cefotaxime, 50 milligrams/kilogram <i>or</i> Ceftriaxone 50 milligrams/kilogram <i>plus consider</i> Vancomycin, 15 milligrams/kilogram <sup>‡</sup>	Ampicillin, 100 milligrams/kilogram <i>plus</i> Cefotaxime, 100 milligrams/kilogram <i>or</i> Ceftriaxone, 100 milligrams/kilogram <i>plus</i> Vancomycin, 15 milligrams/kilogram <sup>§</sup>
Older infants and children (age >90 days)	Ceftriaxone, 50 milligrams/kilogram*	Cefotaxime, 50 milligrams/kilogram <i>or</i> Ceftriaxone, 50 milligrams/kilogram <i>plus consider</i> Vancomycin, 15 milligrams/kilogram <sup>§</sup>	Cefotaxime, 100 milligrams/kilogram <i>or</i> Ceftriaxone, 100 milligrams/kilogram <i>plus</i> Vancomycin, 15 milligrams/kilogram <sup>†</sup>

\*Use meningitis doses if the patient is considered too unstable for lumbar puncture in the emergency department.

<sup>†</sup>Cefotaxime is used rather than ceftriaxone for neonates ≤28 days old because ceftriaxone may displace bilirubin and worsen hyperbilirubinemia.

<sup>‡</sup>Consider addition of vancomycin in sepsis with critical illness.

\*May be given IM.

<sup>§</sup>Add vancomycin only if there is evidence of bacterial meningitis in the cerebrospinal fluid.

**TABLE 66-1** Initial Intravenous Antibiotic Dosages for Bacteremia, Sepsis, and Meningitis

Criteria used to define infants at low risk for SBI in the 31 to 90 days age group include well appearance without a history of prematurity or other comorbidity, and a normal urinalysis. Infants with a suggestion of lower respiratory tract disease should have a chest radiograph. The Boston and, Philadelphia, criteria (which include normal CSF and CBC) should only be applied if the child's presentation warrants the sepsis testing listed above.



Obtaining these laboratory tests is no longer considered routine for infants in the 31 to 90 days age group because of the lower incidence of bacteremia since the advent of the Hib (*Haemophilus influenzae* type B) and Prevnar (*Streptococcus pneumoniae*) vaccinations.

All ill appearing infants should receive parenteral antibiotic therapy (see [Table 66-1](#)) and be admitted to the hospital. Management of low-risk infants remains a subject of significant debate. Infants older than 28 days at low risk may be managed conservatively as inpatients with **ceftriaxone** (see [Table 66-1](#)) pending cultures; as inpatients without antibiotics; as outpatients with **ceftriaxone** 50 milligrams/kilogram IM; or as outpatients without antibiotics. The key deciding factor should be the physician's comfort level and the ability for close follow-up, typically within 12 hours. *If antibiotics are administered (inpatient or outpatient), CSF and blood cultures should be obtained prior to administration of antibiotics.*

Well appearing febrile children between the ages of 29 and 90 days with an identifiable viral source of infection (eg, respiratory syncytial virus [RSV] or influenza) should have urinary tract infection (UTI) ruled out before being discharged from the emergency department. Chest radiographs should be obtained at the discretion of the clinician, but are not indicated for infants with RSV. Lumbar puncture in this group of children may be deferred in those who are well appearing and test positive for a viral source of infection.

### *Infants 3 to 36 Months*

Physical examination findings become more reliable with increasing age, though meningeal signs remain unreliable throughout the first year of life. Viral illnesses including pneumonia account for most febrile illnesses in this age group; patients with clinical findings suggesting pneumonia should have a chest radiograph. One infection that may present with fever only in this age group is UTI. UTI is a significant source of bacterial illness in females prior to toilet training, circumcised boys younger than 6 months of age and uncircumcised boys under 1 year of age; these patients should have urinalysis and urine culture (by catheterization) if a source for the fever is not otherwise identified.

### *Older Febrile Children*

The risk for bacteremia in children older than 3 years is <0.2% since the introduction of Prevnar. CBC and blood cultures are no longer recommended in immunized older children with fever. Etiologies to consider in older febrile children include streptococcal pharyngitis, pneumonia, and EBV infection. Testing is directed by clinical presentation.

## **Emergency Department Care and Disposition**

For the management of pneumonia, see [Chapter 71](#); for the management of UTI, see [Chapter 75](#); infections of the ears, nose, and throat are covered in [Chapter 68](#). Although fever makes children uncomfortable and may potentiate seizures, it typically is not harmful to children, though it does lower the seizure threshold. The physician can use several methods to reduce fever:

1. Remove excessive clothing and blankets to increase heat loss through radiation.
2. Administer **acetaminophen** 15 milligrams/kilogram PO/PR every 4 to 6 hours (maximum dose, 80 milligrams/kilogram in 24 hours).
3. Consider **ibuprofen** 10 milligrams/kilogram PO in children older than 1 year of age; the dose can be repeated every 6 to 8 hours (maximum of 40 milligrams/kilogram in 24 hours), and can be given concurrently with acetaminophen.

The disposition of young infants is discussed above. Patients who are called to return to the ED for evaluation of positive blood cultures require repeat evaluation. Patients with cultures positive for *Neisseria meningitidis* or methicillin-resistant *Staphylococcus aureus*, should be hospitalized and treated with parenteral antibiotics. Otherwise, well appearing afebrile children already on antibiotics should complete the course of therapy. If the patient is afebrile, clinically well, without a focus of infection, and not currently on antibiotics, controversy exists as to the need for repeat cultures

and antibiotics; in general, neither repeat testing or treatment is necessary. If the child with a positive blood culture remains febrile or continues to appear ill, a full septic workup (complete blood cell count, repeat blood culture, lumbar puncture, urinalysis, urine culture, and chest radiograph) should be performed. The patient should be hospitalized and receive parenteral antibiotics (see [Table 66-1](#)).

## **SEPSIS**

Sepsis (bacteremia with clinical evidence of systemic infection) can rapidly progress to multiorgan failure and death. Risk factors include prematurity, immunocompromise, recent invasive procedures, and indwelling foreign objects such as catheters.

### **Clinical Features**

Clinical signs may be vague and subtle in the young infant, including lethargy, poor feeding, irritability, or hypotonia. Fever is common; however, very young infants may be hypothermic. Tachypnea and tachycardia are usually present as a result of fever but also may be secondary to hypoxia and metabolic acidosis. Sepsis can rapidly progress to shock, manifest as prolonged capillary refill, decreased peripheral pulses, altered mental status, and decreased urinary output. Hypotension is usually a very late sign of septic shock in children and, in conjunction with respiratory failure and bradycardia, indicates a grave prognosis.

### **Diagnosis and Differential**

Diagnosis is based on clinical findings and confirmed by positive blood culture results. Though international criteria for sepsis have been published, all infants who appear toxic should be considered septic. The laboratory evaluation of a child with presumed sepsis should include a CBC, blood culture, complete metabolic panel, catheterized urinalysis with culture and

sensitivities, chest radiograph, lumbar puncture, and stool studies in the presence of diarrhea. A serum glucose level should be performed on any critically ill child with cardiorespiratory instability. Serum lactate may be useful for predicting severity of the clinical course.

## **Emergency Department Care and Disposition**

1. Administer high-flow oxygen, institute cardiac monitoring, and secure IV or IO access immediately. Endotracheal intubation should be performed in the presence of respiratory failure.
2. Treat shock with 20 mL/kg boluses of **normal saline** solution. Repeat boluses until vital signs, perfusion, and mental status and urine output improve, up to 100 mL/kg total volume.
3. Treat hypoglycemia with 4 to 5 mL/kg 10% dextrose in neonates and young infants and 2 mL/kg 25% dextrose in older infants and children.
4. Initiate antibiotic therapy promptly, as soon as IV access is achieved. Do not delay due to difficulty with procedures such as lumbar puncture. Empiric antibiotic choices are listed in [Table 66-1](#).
5. Treat volume-refractory shock with **dopamine** 5 to 20 micrograms/kilogram/min or **norepinephrine** 0.1 to 0.2 microgram/kilogram/min.

Consider the presence of drug-resistant organisms or immunoincompetence and infection with unusual or opportunistic organisms.

## **MENINGITIS**

Meningitis is usually a complication of a primary bacteremia and has a peak incidence in children between birth and 2 years of age. Prematurity and immunoincompetence put children at higher risk.

### **Clinical Features**

Meningitis may present with the subtle signs that accompany less serious infections, such as otitis media or sinusitis. Irritability, inconsolability, hypotonia, and lethargy are most common in infants. Older children may complain of headache, photophobia, nausea, and vomiting and exhibit the classic signs of meningismus with complaints of neck pain. Occasionally, meningitis presents as a rapidly progressive, fulminant disease characterized by shock, seizures, or coma, or with febrile status epilepticus.

## **Diagnosis and Differential**

Diagnosis is made by lumbar puncture and analysis of the cerebrospinal fluid (CSF). The CSF should be examined for white blood cells, glucose, and protein and undergo Gram stain and culture. Herpes encephalitis should be considered in the seizing neonate and any child with CSF pleocytosis. In the presence of immunocompromise, infections with opportunistic or unusual organisms should be considered. Cranial computed tomography should be performed before lumbar puncture in the presence of focal neurologic signs or increased intracranial pressure.

## **Emergency Department Care and Disposition**

1. Treatment should always begin with the ABCs and restoration of oxygenation and perfusion (see specific treatment recommendations under Sepsis, above).
2. Empiric antibiotic therapy is based on the patient's age and listed in [Table 66-1](#). Antibiotics should not be deferred or delayed when meningitis is strongly suspected.
3. The role of steroids in the management of meningitis is highly controversial.

For any patient suspected of having meningitis for whom efforts at lumbar puncture fail, the patient should be admitted, hydrated, given meningitis doses of antibiotics, and blood/ urine cultures obtained. Lumbar puncture may be successful after hydration.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 113, “Fever and Serious Bacterial Illness,” by Vincent J. Wang.

# CHAPTER 67

## Common Neonatal Problems

Lance Brown

In general, the signs and symptoms of illness are vague and nonspecific in neonates making the identification of specific diagnoses challenging ([Table 67-1](#)). The survival of premature infants has produced a population of children whose corrected gestational age (chronological age since birth in weeks minus the number of weeks of prematurity) makes them, in some ways, similar to neonates. Neonates present to the emergency department with a range of conditions that span from normal to critical.

Fever or hypothermia
Abnormal tone (limp or stiff)
Altered mental status (lethargy or irritability)
Weak suck
Poor feeding
Jaundice
Abnormal respirations
Cyanosis or mottling
Vomiting

**TABLE 67-1** Nonspecific Signs and Symptoms of Neonatal Emergencies

## NORMAL VEGETATIVE FUNCTIONS

Bottle-fed infants generally take 6 to 9 feedings (2 to 4 oz) in a 24-hour period, with a relatively stable pattern developing by the end of the first month of life. Breast-fed infants generally prefer feedings every 1 to 3 hours. Infants typically lose up to 12% of their birth weight during the first 3 to 7 days of life. After this time, infants are expected to gain about 1 oz/d (20 to 30 grams) during the first 3 months of life. The number, color, and consistency of stool in the same infant changes from day to day and differs among infants. Normal breast-fed infants may go 5 to 7 days without

stooling or have 6 to 7 stools per day. Color has no significance unless blood is present or the stool is acholic (ie, white).

A normal respiratory rate for a neonate is from 30 to 60 breaths/min. Periodic breathing (alternating episodes of rapid breathing with brief (<5 to 10 seconds) pauses in respiration) is usually normal. Normal newborns awaken at variable intervals that can range from about 20 minutes to 6 hours. Neonates and young infants tend to have no differentiation between day and night until approximately 3 months of age.

## **ACUTE, UNEXPLAINED, EXCESSIVE CRYING (INCONSOLABILITY)**

There are benign to life-threatening causes of prolonged crying in infants ([Table 67-2](#)). True inconsolability represents a serious condition in most infants and requires investigation for injury (accidental or inflicted), infection, supraventricular tachycardia (SVT), corneal abrasion, hair tourniquet, hernia or testicular torsion, or abdominal emergency. If, after a thorough emergency department evaluation, a cause for excessive crying has not been identified and the child continues to be inconsolable, admission to the hospital is warranted.

Corneal abrasion
Hair tourniquet (finger, toe, penis)
Stomatitis
Intracranial hemorrhage
Fracture (nonaccidental trauma)
Nasal obstruction/congestion
Inborn error of metabolism
Acute infection (sepsis, urinary tract infection, meningitis)
Congenital heart disease (including supraventricular tachycardia)
Abdominal emergency (incarcerated hernia, volvulus, intussusception)
Testicular torsion
Encephalitis (herpes)

**TABLE 67-2** Conditions Associated With Acute, Unexplained, Excessive Crying in Neonates



## **INTESTINAL COLIC**

Intestinal colic is the most common cause of excessive (but not inconsolable) crying. The cause is unknown. The incidence is about 13% of all neonates. The formal definition includes crying for at least 3 hours per day for at least 3 days per week over a 3-week period. Intestinal colic seldom lasts beyond age 3 months. No effective treatment has been identified. In general, the initial diagnosis of colic is not made in the emergency department and it is a diagnosis of exclusion.

## **NONACCIDENTAL TRAUMA (CHILD ABUSE)**

A battered child may present with unexplained bruises at different ages, skull fractures, intracranial injuries identifiable on computed tomography of the head, extremity fractures, cigarette burns, retinal hemorrhages, unexplained irritability, lethargy, or coma. See [Chapter 187](#) for further discussion of child abuse.

## **FEVER AND SEPSIS**

Fever in the neonate (age 28 days or younger) is defined as the history of documented fever by a parent or presence of a rectal temperature of 38°C (100.4°F) or higher in the ED. Fever in the neonate must be taken seriously, and at this point in time the proper management includes a complete septic work-up, administration of parenteral antibiotics, and admission. See [Chapter 66](#) for appropriate ED therapy of the febrile neonate.

## **GASTROINTESTINAL SYMPTOMS**

### **Surgical Lesions**

Surgically correctable abdominal emergencies in neonates are uncommon, may present with nonspecific symptomatology, and, when suspected, require prompt consultation with an experienced pediatric surgeon. [Chapter 74](#) discusses the most important abdominal emergencies in children. Common signs and symptoms include irritability and crying, poor feeding, vomiting, constipation, and abdominal distention. Bilious vomiting is suggestive of malrotation with midgut volvulus or intussusception and requires emergent surgical consultation and radiologic evaluation (upper GI for malrotation, air-contrast enema for intussusception). Projectile vomiting following feeds suggests pyloric stenosis, which is evaluated with ultrasound. A groin mass may represent an incarcerated hernia; inguinal hernias are common among premature infants.

## **Feeding Difficulties**

Parental perception that an infant's food intake is inadequate may prompt an ED visit. If the patient's weight gain is adequate (see Normal Vegetative Functions above) and the infant appears satisfied after feeding, parental reassurance is appropriate. A successful trial of feeding in the emergency department can reassure parents, emergency department nurses, and physicians alike. When there is an underlying anatomic abnormality interfering with feeding or swallowing (eg, esophageal stenosis, esophageal stricture, laryngeal cleft, or compression of the esophagus or trachea by a double aortic arch), the infant typically has had trouble feeding from birth and usually presents malnourished and dehydrated.

## **Regurgitation**

Regurgitation is due to reduced lower esophageal sphincter tone and relatively increased intragastric pressure in neonates and is ubiquitous in young infants. Gastric contents are effortlessly expelled, typically within 30 min of feeding, and, though potentially large in volume, are never projectile or bilious.

Gastroesophageal reflux (GERD) is typically a self-limited condition and, if an infant is thriving and gaining weight appropriately, reassurance is appropriate.

## **Vomiting**

Vomiting is differentiated from regurgitation by forceful contraction of the diaphragm and abdominal muscles. Vomiting has a variety of causes and is rarely an isolated symptom. Vomiting from birth is usually due to an anatomic anomaly and is often diagnosed in the newborn nursery. Vomiting is a nonspecific but serious symptom in neonates. Etiologies are diverse and include increased intracranial pressure (eg, shaken-baby syndrome), infections (eg, urinary tract infections, sepsis, or gastroenteritis), hepatobiliary disease (usually accompanied by jaundice), and inborn errors of metabolism (usually accompanied by hypoglycemia and metabolic acidosis). Bilious vomiting in a neonate or infant should be considered a surgical emergency (malrotation in the neonate or infant, intussusception in the older infant).

## **Diarrhea**

Although bacterial infection may cause bloody diarrhea, this is rare in neonates. The most common causes of blood in the stool in infants younger than 6 months are cow's milk intolerance and anal fissures. Breast-fed infants may have heme-positive stool from swallowed maternal blood due to bleeding nipples. Necrotizing enterocolitis may present as bloody diarrhea and usually presents with other signs of sepsis (eg, jaundice, lethargy, fever, poor feeding, or abdominal distention). Abdominal radiography may demonstrate pneumatosis intestinalis or free air. Dehydrated neonates (and neonates with impending dehydration from rotavirus) should be admitted for parenteral rehydration.

## **Abdominal Distention**

Abdominal distention can be normal in the neonate and is usually due to lax abdominal muscles, relatively large intraabdominal organs, and swallowed air. In general, if the neonate appears comfortable, is feeding well, and the abdomen is soft, there is no need for concern.

## **Constipation**

Infrequent bowel movements in neonates do not necessarily mean that the infant is constipated. Stool patterns can be quite variable and breast-fed infants may go 1 week without passing stool and then pass a normal stool. Inquire about the passage of meconium in the first 24 to 48 hours of life; infants without normal stooling in the first 2 days of life may have anatomic anomalies (eg, intestinal stenosis or atresias), cystic fibrosis, Hirschsprung disease, or meconium ileus or plug. Constipation that develops later in the first month of life suggests Hirschsprung disease, hypothyroidism, anal stenosis, or anterior anus. Rarely, botulism can present with constipation that precedes neurologic symptoms (cranial nerve deficits, hypotonia, weak cry). Laxatives and enemas are contraindicated in neonates.

## **CARDIORESPIRATORY SYMPTOMS**

### **Noisy Breathing and Stridor**

Noisy breathing in a neonate is usually benign. Infectious causes of stridor seen commonly in older infants and young children (eg, croup) are rare in neonates. Stridor in a neonate is often due to a congenital anomaly, most commonly, laryngomalacia. Other causes include webs, cysts, atresias, stenoses, clefts, and airway hemangiomas. [Chapter 69](#) reviews upper airway emergencies in infants and children.

Nasal congestion from a mild upper respiratory tract infection may cause significant respiratory problems in a neonate. Neonates are obligate nasal breathers and feed for relatively prolonged periods while breathing only

through their noses. The use of **saline drops** and nasal suctioning is typically effective.

## **Apnea and Periodic Breathing**

Periodic breathing may be normal in neonates. *Apnea* is formally defined as a cessation of respiration for longer than 10 to 20 seconds with or without accompanying bradycardia and cyanosis. Apnea generally signifies critical illness including infection, CNS injury, and metabolic disease, and prompt investigation and admission for monitoring and treatment should be initiated. Apnea may be the first sign of bronchiolitis with respiratory syncytial virus in neonates and can occur before wheezing. Chlamydia and pertussis can also cause apnea in the young infant.

## **Cyanosis and Blue Spells**

Many disorders can present with cyanosis, and differentiating among them can be a diagnostic challenge. However, symptom patterns may help differentiate various causes and assist in suggesting the correct diagnosis and course of action. Rapid, unlabored respirations and cyanosis suggest cyanotic heart disease with right-to-left shunting (see [Chapter 72](#)). Irregular or shallow breathing and cyanosis suggest sepsis, CNS disease, or metabolic disorders (see [Chapter 79](#)). Labored breathing with grunting and retractions is suggestive of pulmonary disease such as pneumonia or bronchiolitis (see [Chapter 70](#)). All cyanotic neonates should be admitted to the hospital for monitoring, therapy, and further investigation.

## **JAUNDICE**

There are multiple causes of jaundice, and the likelihood of any specific cause is based on the age of onset. Jaundice that occurs within the first 24 hours of life tends to be serious in nature and usually is addressed while the patient is in the newborn nursery. Jaundice that develops during the second

or third day of life is usually physiologic; if the neonate is gaining weight, feeding and stooling well, is not anemic, does not have an elevated direct (conjugated) bilirubin level, and does not have a total bilirubin level indicating the need for phototherapy (see parent chapter of *Tintinalli's Emergency Medicine*, or <http://aappolicy.aappublications.org/cgi/content/full/pediatrics;114/1/297.full>), reassurance and close follow-up are appropriate. Jaundice that develops after the third day of life is generally serious. Causes include sepsis, UTI, congenital TORCH infections, hemolytic anemia, biliary atresia, breast milk jaundice, and hypothyroidism. Workup of these infants usually includes a complete septic evaluation, including a lumbar puncture, a peripheral blood smear, complete blood count, total and direct bilirubin levels, liver function tests, reticulocyte count, and a Coombs test. Empiric antibiotics (see [Chapter 66](#)) are generally administered when sepsis is suspected.

## ORAL THRUSH

Intraoral lesions due to *Candida* are typically white and pasty and cover the tongue, lips, gingiva, and buccal mucosa. The presence of oral thrush may prompt a visit to the emergency department because the parent notices “something white” in the mouth or because the discomfort of extensive lesions may interfere with feeding. Treatment consists of topical application of oral **nystatin** suspension 4 times a day.

## DIAPER RASH

Two main types of diaper rash are common in neonates: contact dermatitis and candidal diaper dermatitis. Contact dermatitis is macular, erythematous, and has sharply demarcated edges. Treatment consists of frequent diaper changes, air drying, and application of a barrier cream such as **zinc oxide**. Candidal dermatitis presents with erythematous plaques with a scalloped

border and satellite lesions. Treatment consists of frequent diaper changes and application of **nystatin** cream 4 times a day.

## **APPARENT LIFE-THREATENING EVENTS**

An *apparent life-threatening event* (ALTE) is defined as an episode that is frightening to the observer and involves a period of apnea, transient color change (usually pale or cyanotic), and a transient change in tone (limp or stiff). According to the conventional use of ALTE, these infants appear well on presentation to the emergency department. Once it is determined that an ALTE has occurred, the workup typically includes a thorough history and physical exam; vital signs including temperature, HR, RR, and pulse oximetry; and a bedside glucose. Additional laboratory investigations are rarely helpful in the ED and should be directed by the initial history and vital signs but may include: a complete blood count; electrolytes, calcium, phosphorous, magnesium, and ammonia levels; urine toxicological screen; chest radiograph; electrocardiogram; and a septic workup, including blood, urine, and cerebrospinal fluid. Unless the history clearly suggests a benign etiology such as reflux, infants with ALTE are typically admitted to the hospital for further workup and apnea monitoring. The utility of apnea monitoring (particularly in the home) has been recently questioned. At the conclusion of hospitalization, diagnoses of infants with ALTE often remain elusive. When identified, etiologies range from GERD to seizures, inborn errors of metabolism, lower respiratory tract infection, pertussis, gastroenteritis, asthma, head injury, feeding difficulties, and urinary tract infections. There is no relation between ALTEs and sudden infant death syndrome which are now considered distinct entities.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 111, “Neonatal Emergencies and Common Neonatal Problems and Common Neonatal Problems,” by Quynh H. Doan and Niranjan Kissoon; and Chapter 112, “Sudden Infant Death Syndrome and Apparent Life-Threatening Event,” by Carol D. Berkowitz, Ilene Claudius, and Joel S. Tieder.

## CHAPTER 68

# Common Infections of the Ears, Nose, Neck, and Throat

David M. Spiro

This chapter is limited to infections of the ears, nose, neck, and throat. Further information can be found in [Chapter 69](#) “Upper Respiratory Emergencies-Stridor and Drooling,” as well as [Chapter 151](#) “Ear, Nose Emergencies,” and [Chapter 153](#) “Neck and Upper Airway Disorders.”

## ACUTE OTITIS MEDIA

Acute otitis media (AOM) accounts for 13% of all visits to emergency departments in the United States. AOM is an infection of the middle ear space that commonly affects young children because of relative immaturity of the upper respiratory tract, especially the eustachian tube. The most common pathogens in the post-pneumococcal vaccine era are *Streptococcus pneumoniae* (31%) and nontypeable *Haemophilus influenzae* (56%).

### Clinical Features

Peak age is 6 to 36 months. Symptoms include fever, poor feeding, irritability, vomiting, ear pulling, and earache. Signs include bulging, pus behind the tympanic membrane ([Fig. 68-1](#)), an immobile tympanic membrane (TM), loss of visualization of bony landmarks within the middle ear, and bullae on the TM (bullous myringitis). Mastoiditis is the most common suppurative complication of AOM. The primary symptoms of mastoiditis include fever, protrusion of the auricle, and tenderness over the mastoid.





**FIGURE 68-1.** Acute otitis media in a 3-year-old child with an outward bulge of the tympanic membrane and an exudative process in the middle ear space. (Courtesy of Dr. Shelagh Cofer, Department of Otolaryngology, Mayo Clinic.)

## **Diagnosis and Differential**

*Making an accurate diagnosis is the most important first step.* The definition of acute otitis media requires three equally important components: (a) acute onset (<48 hours) of signs and symptoms, (b) middle ear effusion (see [Fig. 68-1](#)), and (c) signs and symptoms of middle ear inflammation. A red TM alone does not indicate the presence of an ear infection. Fever and prolonged crying can cause hyperemia of the TM alone. Pneumatic otoscopy can be a helpful diagnostic tool; however, a retracted drum for whatever reason will demonstrate decreased mobility. Other common causes of acute otalgia are a foreign body in the external ear canal or otitis externa.

## Emergency Department Care and Disposition

1. *Treatment of pain is essential for all children diagnosed with AOM.* Topical analgesics such as benzocaine-antipyrine are recommended for routine use, unless there is a known perforation of the TM. Acetaminophen 15 milligrams/kilogram or ibuprofen 10 milligrams/kilogram can be used.
2. Consider the use of a wait-and-see prescription for the treatment of uncomplicated AOM. Parents are given a prescription and told to wait and see for 48 to 72 hours, and if the child is not better or becomes worse, to fill the prescription. Contraindications to the use of a wait-and-see prescription are: age <6 months, an immunocompromised state, ill-appearance, recent use of antibiotics or the diagnosis of another bacterial infection. If any of these conditions are met, the child should be prescribed an immediate antibiotic.
3. **Amoxicillin** 40-50 milligrams/kilogram/dose PO given twice daily (or 30 milligrams/kilogram/dose three times daily) remains the first drug of choice for uncomplicated AOM.
4. Second line antibiotics include **amoxicillin/clavulanate** 40–50 milligrams/ kilogram/dose given twice daily. **Cefpodoxime** 5 milligrams/kilogram/ dose PO twice daily, **cefuroxime axetil** 15 milligrams/kilogram/dose twice daily, **cefdinir** 7 milligrams/kilogram/dose PO once or twice daily, and **ceftriaxone** 50 milligrams/kilogram/dose IM for 3 daily doses are alternatives. For patients allergic to the previously mentioned antibiotics, **azithromycin** 10 milligrams/kilogram/dose PO on the first day followed by 5 milligrams/kilogram/dose PO for 4 more days can be used.
5. Infants younger than 60 days with AOM are at risk for infection with group B *Streptococcus*, *Staphylococcus aureus*, and gram-negative bacilli and should undergo evaluation and treatment for presumed sepsis.
6. In uncomplicated AOM, symptoms resolve within 48 to 72 hours; however, the middle ear effusion may persist as long as 8 to 12 weeks. Routine follow-up is not necessary unless the symptoms persist or worsen.

7. If mastoiditis is suspected, obtain a CT scan of the mastoid. If the diagnosis is confirmed, obtain consultation with an otolaryngologist and start parenteral antibiotics.

Uncomplicated AOM is treated as an outpatient while mastoiditis typically requires inpatient treatment.

## **OTITIS EXTERNA**

Otitis externa (OE) is an inflammatory process involving the auricle, external auditory canal (EAC), and surface of the TM. It is commonly caused by *Pseudomonas aeruginosa*, *Staphylococcus epidermidis*, and *Staphylococcus aureus*, which often coexist.

### **Clinical Features**

Peak seasons for OE are spring and summer, and the peak age is 9 to 19 years. Symptoms include earache, itching, and, less commonly, fever. Signs include erythema, edema of EAC, white exudate on EAC and TM, pain with motion of the tragus or auricle, and periauricular or cervical adenopathy.

### **Diagnosis and Differential**

Diagnosis for OE is based on clinical signs and symptoms. A foreign body within the external canal should be excluded by carefully removing any debris that may be present.

### **Emergency Department Care and Disposition**

1. Cleaning the ear canal with a small tuft of cotton attached to a wire applicator is the first step. Place a wick in the canal if significant edema obstructs the EAC.

2. Treat mild OE with acidifying agents alone, such as 2% acetic acid (VoSol).
3. Consider oral analgesics, such as **ibuprofen** at 10 milligrams/kilogram/dose every 6 hours.
4. Fluoroquinolone otic drops are now considered the preferred agents over neomycin containing drops. **Ciprofloxacin with hydrocortisone**, 0.2% and 1% suspension (Cipro HC), 3 drops twice daily or **ofloxacin** 0.3% solution 10 drops twice daily can be used. Ofloxacin is used when TM rupture is found or suspected.
5. Oral antibiotics are indicated if auricular cellulitis is present.

Follow-up should be advised if improvement does not occur within 48 hours; otherwise routine follow-up is not recommended. Malignant OE is characterized by systemic symptoms and auricular cellulitis. This condition can result in serious complications and requires hospitalization with parenteral antibiotics.

## **ACUTE BACTERIAL SINUSITIS**

Sinusitis is an inflammation of the paranasal sinuses that may be secondary to infection or allergy and may be acute, sub-acute, or chronic. Acute bacterial sinusitis is defined as an infection of the paranasal sinuses with complete resolution in <30 days. The major pathogens in childhood are *Streptococcus pneumoniae*, *Moraxella catarrhalis*, and nontypeable *Haemophilus influenzae*.

### **Clinical Features**

Two major types of sinusitis may be differentiated on clinical grounds: acute severe sinusitis and mild sub-acute sinusitis. Acute severe sinusitis is associated with elevated temperature, headaches, and localized swelling and tenderness or erythema in the facial area corresponding to the sinuses. Such localized findings are seen most often in older adolescents. Mild sub-acute sinusitis is manifest in childhood as a protracted upper respiratory infection

associated with purulent nasal discharge persisting in excess of 2 weeks. Fever is infrequent. Chronic sinusitis may be confused with allergies or upper respiratory infections.

## **Diagnosis and Differential**

The diagnosis is made on clinical grounds without laboratory or radiographic studies. Transillumination of the maxillary or frontal sinuses is seldom helpful. Nasal congestion lasting 3 to 7 days often accompanies viral upper respiratory infections and should not be diagnosed as acute sinusitis and does not need treatment with antibiotics. Similarly, colored drainage from the nose as a solitary symptom does not suggest a diagnosis of sinusitis and should not be treated with antibiotics. Imaging studies are not needed to confirm a diagnosis of acute bacterial sinusitis in children <6 years of age with persistent symptoms.

## **Emergency Department Care and Disposition**

Patients with mild symptoms suggestive of a viral infection can be observed for 7 to 10 days, with no antibiotics prescribed. Suspect acute bacterial sinusitis if symptoms persist or are severe: fever > 39°C, purulent nasal drainage for > 3 days and ill-appearance.

1. For children with mild to moderate sinusitis, treat with **amoxicillin** (40–50 milligrams/kilogram/dose PO twice daily) for 10 to 14 days.
2. For children who present with severe symptoms, are in day care or have recently been treated with antibiotics, prescribe oral second- and third-generation cephalosporins such as **cefprozil** (7.5 to 15 milligrams/kilogram PO twice a day), **cefuroxime** (15 milligrams/kilogram PO twice a day), and **cefpodoxime** (5 milligrams/kilogram PO twice a day).
3. Intranasal steroids have shown modest benefits and are recommended if antibiotics do not result in improvement in the first 3 to 4 days of treatment.

# STOMATITIS AND PHARYNGITIS

Herpangina, hand, foot, and mouth disease (HFMD), and herpes simplex gingivostomatitis are the primary infections that cause stomatitis in children and are all viral. The vast majority of pharyngitis is caused by viral infections, however, Group A  $\beta$ -hemolytic *Streptococcus* (GABHS) and *Neisseria gonorrhoea* are bacterial infections that require accurate diagnoses. The identification and treatment of GABHS pharyngitis is important to prevent the suppurative complications and the sequelae of acute rheumatic fever.

## Clinical Features

Herpangina causes a vesicular enanthem of the tonsils and soft palate, affecting children 6 months to 10 years of age during late summer and early fall. The vesicles are painful and can be associated with fever and dysphagia. HFMD disease usually begins as macules which progress to vesicles of the palate, buccal mucosa, gingiva, and tongue. Similar lesions may present on the palms of hands, soles of feet, and buttocks. Herpes simplex gingivostomatitis often presents with abrupt onset of fever, irritability, and decreased oral intake with edematous and friable gingiva. Vesicular lesions often with ulcerations are seen in the anterior oral cavity.

Peak seasons for GABHS are late winter or early spring, the peak age is 5 to 15 years, and it is *rare before the age of 2*. Symptoms (sudden onset) include sore throat, fever, headache, abdominal pain, enlarged anterior cervical nodes, palatal petechiae, and hypertrophy of the tonsils. With GABHS there is usually the absence of cough, coryza, laryngitis, stridor, conjunctivitis, and diarrhea. A scarlatinaform rash associated with pharyngitis almost always indicates GABHS and is commonly referred to as *scarlet fever*.

Ebstein Barr Virus (EBV) is a herpes virus and often presents much like streptococcal pharyngitis. Common symptoms are fever, sore throat, and malaise. Cervical adenopathy may be prominent and often is posterior and

anterior. Hepatosplenomegaly may be present. EBV should be suspected in the child with pharyngitis nonresponsive to antibiotics in the presence of a negative throat culture.

Gonococcal pharyngitis in children and nonsexually active adolescents should alert one to the possibility of sexual abuse. Gonococcal pharyngitis may be associated with infection elsewhere including proctitis, vaginitis, urethritis, or arthritis.

## **Diagnosis and Differential**

The diagnoses of herpangina, HFM disease and herpes simplex gingivostomatitis are based on clinical findings. To diagnose GABHS, current guidelines recommend the use of **Centor criteria** to determine which patients require testing: (a) tonsillar exudates, (b) tender anterior cervical lymph-adenopathy, (c) absence of cough, (d) history of fever. With 2 or more criteria, testing should be performed with a rapid antigen detection test and/ or culture. If the rapid antigen test is negative, a confirmatory throat culture is recommended.

Diagnosis of EBV is often clinical. A heterophile antibody (monospot) can aid in the diagnosis. The monospot may be insensitive in children <2 years of age and is often negative in the first week of illness. If obtained, the white blood cell count may show a lymphocytosis with a preponderance of atypical lymphocytes. Diagnosis of gonococcal pharyngitis is made by culture on Thayer-Martin medium. Vaginal, cervical, urethral, and rectal cultures also should be obtained if gonococcal pharyngitis is suspected.

## **Emergency Department Care and Disposition**

1. Treatment of herpangina, HFM disease and herpes simplex gingivostomatitis is primarily supportive. Systemic analgesics such as a combination of ibuprofen and tylenol should be considered. Parenteral hydration may be necessary if the child cannot tolerate oral fluids. Occasionally oral narcotics may be required.
2. Antibiotics for the treatment of GABHS pharyngitis should be reserved for patients with a positive rapid antigen test or culture. Antibiotic

choices for GABHS include **penicillin V** (children 250 milligrams PO twice daily, adolescent/adult 500 milligrams PO twice daily); **benzathine penicillin G** 1.2 million units IM (600,000 units IM for patients weighing less than 27 kg); and **erythromycin ethylsuccinate** 10 to 20 milligrams/kilogram/dose PO given twice daily for 10 days. Antipyretics and analgesics should be routinely prescribed until symptoms resolve.

3. Treat gonococcal pharyngitis with **ceftriaxone** 250 milligrams IM. When gonococcal pharyngitis is suspected, empiric treatment of chlamydia is recommended with **azithromycin** 1 gram PO given in the emergency department. Appropriate follow-up should be encouraged for treatment failure and symptomatic contacts. Follow-up for suspected gonococcal pharyngitis should include local reporting agencies and social service investigations.
4. EBV is usually self-limited and requires only supportive treatment including antipyretics, fluids, and rest. A dose of **dexamethasone** 0.5 milligrams/kilogram PO to a maximum of 10 milligrams once may be given for more severe disease presentations.

## CERVICAL LYMPHADENITIS

Acute, unilateral cervical lymphadenitis is commonly caused by *Staphylococcus aureus* or *Streptococcus pyogenes*. Bilateral cervical lymphadenitis is often caused by viral entities such as EBV and adenovirus. Chronic cervical lymphadenitis is less common but may be caused by *Bartonella henselae* (also called oculoglandular fever) or *Mycobacterium* species.

### Clinical Features

Acute cervical lymphadenitis presents with tender, 2 to 6 cm nodes often with overlying erythema. Bilateral cervical lymphadenitis presents with small, rubbery lymph nodes and usually self-resolves. Bartonella results from the scratch of a kitten with ipsilateral cervical lymphadenitis and often concurrent conjunctivitis.



## Diagnosis and Differential

Most cases are diagnosed clinically, although culture may guide effective antimicrobial treatment. Differential may also include sialoadenitis (infection of the salivary glands), which is usually caused by *Staphylococcus aureus* or *Streptococcus pyogenes*, as well as gram-negative and anaerobic bacteria.

## Emergency Department Care and Disposition

1. **Either amoxicillin plus clavulanic acid**, 30 to 40 milligrams/kilogram/dose given twice daily or **clindamycin** 10 to 15 milligrams/kilogram/dose given three times daily are recommended first line antibiotics for the treatment of acute cervical lymphadenitis.
2. The presence of a fluctuant mass may require incision and drainage in addition to antimicrobial therapy.

Most cases of acute bilateral cervical lymphadenitis resolve without antibiotics, as they often represent viral infection or reactive enlargement. Chronic cases of lymphadenitis are often treated surgically, with directed antimicrobial therapy in some cases depending on clinical diagnosis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 114, “Ear and Mastoid Disorders in Infants and Children” by David M. Spiro and Donald H. Arnold; Chapter 116, “The Nose and Sinuses” by Joanna S. Cohen and Dewesh Agrawal; Chapter 117 “The Mouth and Throat” by Derya Caglar and Richard Kwun; and Chapter 118, “Neck Masses in Children” by Osama Y. Kentab and Nadeemuddin Qureshi.

## **CHAPTER 69**

# **Upper Respiratory Emergencies—Stridor and Drooling**

Kathleen M. Adalgais

The physical sign common to all causes of upper respiratory tract obstruction is stridor. Laryngomalacia, due to a developmentally weak larynx, accounts for 60% of stridor in the neonatal period, but is self-limited and rarely requires treatment. Common causes of stridor in children > 6 months of age discussed here include viral croup, epiglottitis, bacterial tracheitis, airway foreign body, retropharyngeal abscess, and peritonsillar abscess. Other etiologies including Ludwig's angina and oropharyngeal trauma are covered in [Chapter 153](#).

## **VIRAL CROUP (LARYNGOTRACHEOBRONCHITIS)**

Viral croup is responsible for most cases of stridor after the neonatal period. It is usually a benign, self-limited disease caused by edema and inflammation of the subglottic area. Children ages 6 months to 3 years are most commonly affected, with a peak at an age of 12 to 24 months.

### **Clinical Features**

Croup occurs mainly in late fall and early winter, typically, beginning with a 1- to 5-day prodrome of cough and coryza, followed by a 3- to 4-day period of classic barking cough, though cough and stridor may be abrupt in onset. Symptoms peak on days 3 to 4 and are often perceived as more severe at night. Physical examination classically shows a biphasic stridor, although the inspiratory component usually is much greater.

## Diagnosis and Differential

The diagnosis of croup is clinical: a barking, seal-like cough and history or finding of stridor in the appropriate setting is diagnostic. The differential diagnosis includes epiglottitis, bacterial tracheitis, or foreign body aspiration. Radiographs are not necessary, unless other causes are being considered. Lateral neck and chest radiographs may demonstrate the normally squared shoulders of the subglottic tracheal air shadow as a pencil tip, hourglass, or “steeple sign” though this sign is neither sensitive nor specific for croup.

## Emergency Department Care and Disposition

1. Patients with significant stridor should be kept in a position of comfort with minimal disturbance; monitor pulse oximetry and provide oxygen as needed.
2. Administer **dexamethasone** 0.15 to 0.6 milligrams/kilogram (10 milligrams max) PO or IM (may use the IV formulation orally). Nebulized **budesonide** (2 milligrams) may be clinically useful in moderate to severe cases. Even patients with very mild croup symptoms benefit from steroids, therefore most ED patients diagnosed with croup should be treated with corticosteroids.
3. Nebulized **racemic epinephrine**, 0.05 mL/kg/dose up to 0.5 mL of a 2.25% solution, should be used to treat moderate to severe cases (significant stridor at rest). Alternatively **L-epinephrine** (1:1000), 0.5 mL/ kg (to a maximum of 5 mL) can be used. Children with stridor associated only with agitation do not need epinephrine.
4. Although intubation should be performed when clinically indicated, aggressive treatment with epinephrine results in less than a 1% intubation rate. When necessary, consider a smaller endotracheal tube than estimated by age to avoid trauma to the inflamed mucosa.
5. Helium plus oxygen (**Heliox**), typically in a 70:30 mixture, may prevent the need for intubation in the most severe cases. Heliox can be effectively

given with a maximum oxygen concentration of 40%, therefore, patients requiring higher  $\text{FiO}_2$  are not candidates for Heliox.

6. Children with persistent stridor at rest, tachypnea, retractions, and hypoxia or those who require more than two treatments of epinephrine should be admitted to the hospital.
7. Discharge criteria include the following: at least 3 hours since the last dose of epinephrine, nontoxic appearance, no clinical signs of dehydration, room air oxygen saturation greater than 90%, parents able to recognize changes in the patient's condition, and no social concerns with access to telephone and relatively short transit time to the hospital.

## **EPIGLOTTITIS**

Epiglottitis is life threatening and can occur at any age. Historically caused by *Haemophilus influenzae*, vaccination has decreased the occurrence of epiglottitis and shifted the median age of presentation toward older children and adults. In immunized children, most cases are caused by strep and staph species.

### **Clinical Features**

Classically, there is abrupt onset of high fever, sore throat, and drooling. Symptoms may progress rapidly to stridor and respiratory distress. Cough may be absent and the voice muffled. The patient is toxic in appearance and may assume a tripod or sniffing position to maintain the airway. The presentation in older children and adults can be subtler. The only complaint may be severe sore throat, with or without stridor. The diagnosis is suggested by severe sore throat, normal-appearing oropharynx, and a striking tenderness with gentle movement of the hyoid.

### **Diagnosis and Differential**

Radiographs are usually unnecessary to make the diagnosis in patients with a classic presentation. If the diagnosis is uncertain, then lateral neck films should be taken at the bedside in extension and during inspiration with a minimum of disturbance. If it is necessary for the patient to be moved to the radiology suite, a physician trained in airway management should be present at all times. The epiglottis is normally tall and thin, but in epiglottitis, it is very swollen and appears squat and fat like a thumbprint (called the “thumb sign”) at the base of the hypopharynx ([Fig. 69-1](#)). False negative radiographic evaluations do occur, and, if suspicion remains, gentle direct visualization of the epiglottis is necessary to exclude the diagnosis. Blood cultures are positive in up to 90% of patients, whereas cultures from the epiglottis are less sensitive.



**FIGURE 69-1.** Lateral neck view of a child with epiglottitis. (Courtesy of W. McAlister, MD, Washington University School of Medicine, St. Louis, MO.)

## **Emergency Department Care and Disposition**

1. Keep the patient seated and upright. Provide oxygen and administer nebulized **racemic or L-epinephrine**. Heliox also can be attempted.
2. In the event of total airway loss, attempt bag-valve-mask ventilation.
3. Alert a referral center or pediatric otolaryngologist to coordinate decisions regarding definitive management.
4. The most experienced individual should perform intubation as soon as the diagnosis is made. Use sedation, paralytics, and vagolytics as indicated. Multiple endotracheal tube sizes must be immediately available. For the patient who is able to maintain their airway, use of paralytics must be accompanied by the certainty that intubation will be successful or that a surgical airway can immediately be performed if unsuccessful.
5. Steroids may be employed to decrease mucosal edema of the epiglottis. Use **methylprednisolone** 1 milligram/kilogram IV every 6 hours or **dexamethasone** 0.15 to 0.6 milligrams/kilogram IV.
6. Administer antibiotics only after airway management: **cefuroxime** 50 milligrams/kilogram IV per dose, **cefotaxime** 50 milligrams/kilogram IV per dose, or **ceftriaxone** 50 milligrams/kilogram IV per dose are appropriate empiric options. In regions with increased cephalosporin resistance, **vancomycin** 10 milligrams/kilogram/dose should be added.

## **BACTERIAL TRACHEITIS**

Bacterial tracheitis (membranous laryngotracheobronchitis or “bacterial croup”) is uncommon and can present as either a primary or secondary infection. The mean age of presentation is between 4 and 8 years of age, compared with younger ages as had been previously described. It is usually caused by *S aureus*, *S pneumoniae*, or  $\beta$ -lactamase-producing gram-negative organisms (*Haemophilus influenza* and *Moraxella catarrhalis*).

### **Clinical Features**

Patients with bacterial tracheobronchitis have more respiratory distress than do patients with croup. Children appear septic and present similarly to those

with epiglottitis, with the following exceptions: severe inspiratory and expiratory stridor, occasionally with thick sputum production, and a raspy hoarse voice but no dysphagia.

## **Diagnosis and Differential**

Radiographs of the lateral neck and chest usually demonstrate subglottic narrowing of the trachea with irregular densities and ragged and indistinct borders.

## **Emergency Department Care and Disposition**

1. Manage patients as above for epiglottitis; more than 85% require intubation. Ideally, perform intubation and bronchoscopy in the operating room where cultures and Gram stain may be obtained to guide antibiotic therapy.
2. Administer empiric parenteral antibiotics: ampicillin/sulbactam 50 milligrams/kilogram/dose or **ceftriaxone** 50 milligrams/kilogram IV per dose **plus** clindamycin 10 milligrams/kilogram/dose. In areas with increasing *S. aureus* resistance, consider the addition of **vancomycin** 10 milligrams/kilogram IV every 6 hours.

## **AIRWAY FOREIGN BODY**

Foreign body (FB) aspirations cause more than 3000 deaths each year and have a peak incidence between ages 1 and 3 years. In children younger than 6 months, the cause is usually secondary to a feeding by a well-meaning sibling. The most common FB aspirations fall into 2 groups: food and toys. Commonly aspirated foods include peanuts, sunflower seeds, raisins, grapes, hot dogs, but almost any object may be aspirated. Unlike small round metal objects, aspirated vegetable matter commonly causes intense pneumonitis and subsequent pneumonia and suppurative bronchitis. A FB aspiration should be suspected if there is a history of sudden onset of

coughing or choking and should be considered in all children with unilateral wheezing.

## **Clinical Features**

At presentation many patients are asymptomatic. There may or may not be a witnessed aspiration. The primary symptom is cough, which is classically abrupt in onset, and may be associated with gagging, choking, stridor, or cyanosis. Signs depend on the location of the FB and the degree of obstruction: stridor with a FB in the laryngotracheal area; wheezing with a bronchial FB. Symptoms are unreliable in localizing the level of FB, however: wheeze is present in 30% of laryngotracheal FB aspirations and stridor is found in up to 10% of bronchial aspirations. Eighty percent to 90% of FBs are located in the bronchus. Patients with immediate onset of severe stridor and cardiac arrest usually have laryngotracheal aspirations.

## **Diagnosis and Differential**

FB aspiration is easily confused with more common causes of upper respiratory diseases because 36% of patients have fever, 35% have wheezes, and 38% have rales. Plain chest radiographs can be normal in > 50% of tracheal FB and 25% of bronchial FB; more than 75% of FB in children <3 years of age are radiolucent. In cases of complete obstruction, atelectasis may be found. In partial obstructions, a ball valve effect occurs, with air trapping caused by the FB leading to hyperinflation of the obstructed lung. Thus, in a stable cooperative child, inspiratory and expiratory posteroanterior chest radiographs may be helpful. In a stable but noncooperative child, decubitus films may be used but are less sensitive than fluoroscopy. FB aspiration is definitively diagnosed preoperatively in only one-third of cases; thus, if clinically suspected, laryngoscopy is indicated.

Upper esophageal FB are usually radiopaque and can impinge on the posterior aspect of the trachea. Patients may present with stridor, and typically have dysphagia. Radiographically, flat FBs such as coins are



usually oriented in the sagittal plane when located in the trachea (which appear as a thick line in an anteroposterior chest radiograph) and in the coronal plane when in the esophagus (which appear round on an anteroposterior chest radiograph).

## **Emergency Department Care and Disposition**

1. If FB aspiration or airway obstruction is clearly present, perform BLS procedures to relieve airway obstruction (see [Chapter 3](#)).
2. If BLS maneuvers fail, undertake direct laryngoscopy with Magill forceps to remove the FB. If the FB cannot be seen, orotracheal intubation with dislodgement of the FB distally may be lifesaving.
3. Consider racemic epinephrine or Heliox as symptomatic palliatives.
4. Definitive treatment usually requires rigid bronchoscopy in the operating room under general anesthesia.

## **RETROPHARYNGEAL ABSCESS**

### **Clinical Features**

Retropharyngeal abscess is the second most commonly seen deep neck infection, and usually occurs in children between 6 months and 4 years of age. Patients classically appear toxic and present with fever, drooling, dysphagia, and inspiratory stridor. Patients may hold their neck in an unusual position with torticollis, hyperextension, or stiffness. Dysphagia and refusal to feed occur before significant respiratory distress. Patients may develop rapidly fatal airway obstruction from sudden rupture of the abscess pocket. Aspiration pneumonia, empyema, mediastinitis, and erosion into the jugular vein and carotid artery are reported complications.

### **Diagnosis and Differential**

Physical examination of the pharynx may show a retropharyngeal mass. Although palpation commonly will demonstrate fluctuance, this could lead to rupture of the abscess. Lateral neck radiograph performed during inspiration may show a widened retropharyngeal space. The diagnosis is suggested when the retropharyngeal space at C2 is twice the diameter of the vertebral body or greater than one half the width of C4. CT of the neck with IV contrast is thought to be almost 100% sensitive and very helpful in differentiation between cellulitis and abscess.

## **Emergency Department Care and Disposition**

1. Immediate airway stabilization is the first priority. Intubate unstable patients before performing CT.
2. Antibiotic choice is controversial because most retropharyngeal abscesses contain mixed flora. Consider **ampicillin/sulbactam** 50 milligrams/kilogram/dose IV hours and/or **clindamycin** 10 milligrams/kilogram IV hours. Substitute **ceftriaxone** 50 milligrams/kilogram/ dose for ampicillin/sulbactam in the penicillin-allergic patient.
3. Consider adjunctive treatment with parenteral steroids (eg, **dexamethasone** 0.15 to 0.6 milligrams/kilogram IV to a maximum of 10 milligrams).
4. Consult otolaryngology for operative incision and drainage as indicated. Although cellulitis and some very small abscesses may do well with antibiotics alone, most require surgery.

## **PERITONSILLAR ABSCESS**

### **Clinical Features**

Peritonsillar abscess in children most commonly presents in adolescents with an antecedent sore throat. Patients usually appear acutely ill with fevers, chills, dysphagia/odynophagia, trismus, drooling, and a muffled “hot potato” voice.

The uvula is displaced away from the affected side. As a rule, the affected tonsil is anteriorly and medially displaced.

## **Diagnosis and Differential**

The diagnosis can typically be made through careful visualization of the oral cavity. Classic findings include uvular deviation away from the abscess, soft palate displacement, trismus, and localized fluctuance; airway compromise may occur. In typical cases, imaging studies are unnecessary, though in patients with toxic appearance or atypical exam findings, computed tomography (CT) with contrast or ultrasound is indicated.

## **Emergency Department Care and Disposition**

1. Treat most cases with needle aspiration, antibiotics, and pain control. Administer topical (eg, benzocaine or endocaine spray), oral (eg, oxycodone or vicodin), or parenteral (eg, fentanyl, morphine) analgesics, then aspirate the abscess using a large gauge needle. Avoid deep penetration which could injure adjacent vascular structures and result in significant bleeding. The last centimeter of the tip of a needle guard can be cut off, and carefully reattached to the aspirating syringe, covering all but the end of the needle, to limit the depth of penetration.
2. Consider **clindamycin** 10 milligrams/kilogram IV hours or **ampicillin/sulbactam** 50 milligrams/kilogram/dose IV hours. Definitive follow-up is essential in all cases. Oral antibiotics for outpatient treatment include **amoxicillin/clavulanate** 22.5 milligrams/kilogram/dose given twice daily.
3. Formal incision and drainage in the operating room is sometimes necessary, especially in young or uncooperative patients. Most patients can be discharged safely on oral antibiotics following drainage.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 69, “Stridor and Drooling” by Joseph D. Gunn III.

## **CHAPTER 70**

# **Wheezing in Infants and Children**

Donald H. Arnold

## **ASTHMA**

Asthma is the most common chronic disease and the most frequent reason for hospitalization of children in the United States. The primary pathologic event is airway inflammation causing recurrent episodes of wheezing, dyspnea and cough associated with airflow obstruction that is variably reversible. The most common triggers are viral infections (often with fever), allergens (animals, dust, mold, pollen), environmental irritants (tobacco smoke, ozone), cold air, and exercise. Acute exacerbations may progress to unresponsive airway obstruction (status asthmaticus), respiratory failure and death and demand immediate treatment calibrated to severity.

### **Clinical Features**

The patient with an acute exacerbation may present with cough, wheezing, shortness of breath, chest tightness and/or chest pain. Cough is as frequent a manifestation as wheezing, and wheezing may be absent if airway obstruction is severe. Rales or rhonchi may be present but are usually due to atelectasis and thus are localized and clear with bronchodilator treatment. Tachypnea is a sensitive sign, and together with accessory muscle use is an accurate measure of severity. Elevation of pulsus paradoxus, decreased aeration on chest auscultation, and patient fatigue are potential signs of impending respiratory failure.

Hypoxemia is usually mild ( $SpO_2 > 92\%$ ) and due to V/Q mismatch, which may worsen during initial treatment with bronchodilators for a period

of 1 to 2 hours requiring oxygen therapy. If available, end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) by capnometry should be monitored during severe exacerbations. Hypocapnia is expected early in the course of an asthma exacerbation, thus a normal or minimally elevated ETCO<sub>2</sub> may be a sign of impending ventilatory failure.

## **Diagnosis and Differential**

The diagnosis of bronchospasm is made clinically. The chronic diagnosis of asthma is rarely made in the ED, as spirometry, the criterion standard, is not routinely available in the ED. Although peak expiratory flow is often recommended for children > 5 years, it frequently underestimates the severity of airway obstruction. If available, FEV<sub>1</sub> is the preferred measure of severity, with percent predicted level defining severity:  $\geq 40\%$  correlates with mild-moderate airway obstruction, and  $<40\%$  correlates to a severe exacerbation.

Because viral infections are the most common precipitant of asthma exacerbations in children, and because fever is a common associated sign, fever alone does not indicate the need for a chest radiograph. It may be considered for infants and young children with a first episode of wheezing to exclude anatomic abnormalities or foreign body. For others, a radiograph is indicated when localized findings (rales or decreased breath sounds) do not resolve with bronchodilator treatment or when there is concern for possible pneumothorax (pain or significant hypoxia) or foreign body. The pediatric respiratory assessment measure (PRAM) is one of the few severity scores that has been validated, and each ED should have a preferred score to facilitate severity assessment and communication amongst providers.

The differential diagnosis of wheezing in infants and children is extensive and should consider patient age, presenting signs and symptoms, overall clinical course, and results of ancillary testing, if indicated.

## **Emergency Department Care and Disposition**

An inhaled  $\beta_2$ -agonist, most often albuterol, is the mainstay of acute asthma therapy.

1. Administer **oxygen** for saturations below 95%.
2. Give **albuterol** by metered-dose inhaler with spacer (4 to 8 puffs every 20 min  $\times$  3 doses then Q 1 to 4 hours); or by intermittent nebulization (0.15 milligram/kilogram, minimum 2.5 milligrams, every 20 min  $\times$  3 doses, then 0.15-0.3 milligram/kilogram up to 10 milligrams every 1 to 4 hours); or continuously (0.5 milligram/kilogram/h).
3. Administer **systemic corticosteroids** in all but the mildest cases that respond immediately to albuterol. Early administration, even at the time of triage, decreases hospital admission rates. **Prednisone or prednisolone** (2 milligrams/kilogram/d, maximum 60 milligrams/d) are the preferred agents. A 3 to 5 day course is usually sufficient and does not require tapering. These medications are generally contraindicated in varicella-susceptible patients who have or might have exposure to varicella. **Dexamethasone** (0.6 milligram/kilogram to a maximum of 10 milligrams) may be used as a single dose in lieu of a short burst of shorter acting steroid. The IV formulation may be given orally and may be associated with less vomiting than prednisolone.
4. Give **ipratropium** with nebulized or meter-dosed albuterol (0.25 to 0.5 milligram Q 20 min  $\times$  3 doses).
5. **Systemic  $\beta$ -agonists** have no advantage over inhaled albuterol except in the patient with minimal ventilation. **Terbutaline** has selective  $\beta_2$  activity and can be administered SQ (0.01 milligram/kilogram, maximum 0.4 milligram, Q 20 min  $\times$  3 doses) or IV (0.01 milligram/ikilogram load over 5 to 10 min then 0.001 to 0.01 milligram/kilogram/min). **Epinephrine** continues to be used by some clinicians for its  $\alpha$ -agonist activity that may shrink edematous mucosa (SQ: 0.01 milligram/ kilogram, maximum 0.5 milligram, Q 15 min).
6. Consider **magnesium sulfate** (50 to 75 milligram/kilogram, maximum 2 grams, IV over 10 to 20 min) in the patient with poor ventilation.
7. Consider **ketamine** (2 milligrams/kilogram IV followed by 2 to 3 milligrams/kilogram/h) in severe disease to delay or prevent respiratory failure and the need for assisted ventilation.

8. **Helium-oxygen (Heliox)** as a 60:40 or 70:30 (helium:oxygen) mix may restore laminar airflow and improve alveolar ventilation. Nebulized albuterol may be administered with this treatment. A maximum FiO<sub>2</sub> of 40% can be administered with helium so patients requiring high concentrations of oxygen are not candidates for this therapy.
9. Administer **IV fluids** (normal saline) to patients in status asthmaticus who have decreased oral intake or are NPO due to the severity of the episode.
10. Admit children to the hospital who do not respond adequately to treatment (eg, persistent hypoxemia or failure to normalize aeration over 2 to 4 hours) or whose caretaker may not be able to provide necessary ongoing care.
11. **Respiratory failure** may be avoided by rapid escalation of the above treatments. However, patient fatigue or persistent bronchospasm may nonetheless occur, and for this reason the need for endotracheal intubation and mechanical ventilation should be anticipated and planned for. Because laryngoscopy may precipitate severe laryngo- or bronchospasm, the decision to intubate should be carefully considered. The most experienced operator available should perform the procedure, and a carefully considered sequence of rapid-sequence intubation medications chosen. These often include premedication with atropine (0.02 milligram/kilogram, minimum 0.5 milligram; maximum 1 milligram) and lidocaine (1.5 milligrams/kilogram) and sedation with ketamine (2 milligrams/kilogram), followed by paralysis using succinylcholine (2 milligrams/kilogram) or rocuronium (1 milligram/kilogram) to provide optimal intubating conditions.
12. **Discharge planning** should include an “action plan” (available at [http://www.nhlbi.nih.gov/health/public/lung/asthma/actionplan\\_text.htm](http://www.nhlbi.nih.gov/health/public/lung/asthma/actionplan_text.htm)), albuterol as MDI or nebulizer, oral steroids, and follow-up with the primary care provider.

## **BRONCHIOLITIS**

Bronchiolitis is the most frequent lower respiratory infection in the first 2 years of life and is most commonly caused by *respiratory syncytial virus* (RSV). Infection causes acute airway inflammation and edema, small airway epithelial cell necrosis and sloughing, increased mucus production and mucus plugs, and bronchospasm, all of which can vary considerably between patients and during the course of the illness.

## **Clinical Features**

Most patients have rhinorrhea typical of an upper respiratory infection (URI) in addition to variable signs and symptoms of lower respiratory infection, including fever, wheezing, tachypnea, cough, rales, use of accessory muscles and nasal flaring. Apnea is of great concern in young infants (see below), as is dehydration. Hypoxemia, cyanosis or altered mental status or fatigue are ominous signs and may portend respiratory failure. Management decisions should consider signs of severity as well as the typical time course for illness: severity increases over the first 3 to 5 days with total duration of illness 7 to 14 days. Sustained immunity does reliably occur, and reinfection with recurrence of illness is not uncommon.

## **Diagnosis and Differential**

Diagnosis is clinical and does not require laboratory or radiologic studies. The above signs and symptoms occurring from November to March are sufficient for diagnosis. Serial observations (including pulse oximetry) and reassessment is key to determining disposition, and patients with risk factors for apnea and/or severe disease with possible respiratory failure should be identified early, including: (a) young developmental age (<6–12 weeks) or: prematurity (<37 weeks); (b) witnessed apnea, (c) hemodynamically significant congenital heart disease (on medication for CHF; moderate to severe pulmonary hypertension; cyanotic CHD), (d) chronic lung disease (bronchopulmonary dysplasia; congenital malformations; cystic fibrosis), and (e) immunocompromise.



Routine testing for RSV or other pathogens is indicated only if the information is necessary for placement decisions for hospitalized patients. Routine performance of radiographs increases inappropriate antibiotic use and does not change time to recovery. Radiographs are indicated only if other diseases or foreign body are suspected, or if the patient does not improve or whose disease is more severe than expected.

## **Emergency Department Care and Disposition**

1. **Nasal suctioning and saline drops:** suctioning of the nasal passages after saline instillation may substantially decrease work of breathing, correct hypoxemia, and enable the patient to feed normally. Nasal vasoconstrictors are not indicated and have resulted in tachydysrhythmia.
2. Provide **oxygen** to maintain saturations > 90% to 92%.
3. **Nebulized  $\alpha$ - and  $\beta$ -agonists** should not be routinely used. However, use of a  $\beta_2$ -agonist (albuterol) might be considered, particularly if there is a personal or family history of asthma. Epinephrine (0.5 mL of 1:1000 in 2.5 mL saline) may be beneficial due to  $\alpha$ -agonist mediated mucosal vasoconstriction with reduction of edema. If these medications are used, an objective measure (eg, respiratory rate or bronchiolitis score) should be used to assess response.
4. Consider **nebulized hypertonic saline** (3% or 5%, 3 to 5 mL by nebulizer) to decrease mucus production and viscosity.
5. Provide **isotonic IV fluids (NS)** if necessary. Patients with bronchiolitis may not be able to feed normally, and when respiratory rates exceed 60 to 70 breaths/min there is increased risk of aspiration of feedings.
6. **Helium-oxygen** (Heliox, see above) may delay or avoid respiratory failure and need for invasive ventilation in severe cases.
7. **Corticosteroids** should not be used routinely for patients with bronchiolitis. A trial of dexamethasone and epinephrine published in 2009 concluded that this *combination* was beneficial, however, the benefit was marginal and the doses of dexamethasone were large. Potential adverse effects of these medications and lack of sufficient evidence for benefit preclude their routine use.

8. **Ventilatory support:** noninvasive measures (CPAP or BiPAP) may improve oxygenation and ventilation, decrease work of breathing, and delay or obviate the need for endotracheal intubation. Additionally, application of CPAP may prevent further apnea in affected infants.
9. **Decision for hospitalization :** consider whether risk factors for *apnea and/or severe disease* are present (see Diagnosis above). Additional indications for hospitalization are persistent hypoxia, tachypnea, or abnormal work of breathing, and inability to feed or maintain hydration. Decision-making must also consider the time-point of disease progression (severity increases over the first 3 to 5 days of illness), the ability of caretakers to manage the illness, and the availability of follow-up.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 120, “Wheezing in Infants and Children,” by Donald H. Arnold, David M. Spiro and Melissa L. Langan.

# CHAPTER 71

## Pneumonia in Infants and Children

Lance Brown

Most cases of pediatric pneumonia develop from inhalation of infective bacteria or viruses. The clinical presentation, likely etiologic agents, severity of illness, and disposition, vary with age. In the neonate, group B *Streptococci*, gram-negative bacteria, and *Listeria monocytogenes*, are important pathogens. In the 1 to 3 month old age group, infants may be afebrile with pneumonitis syndrome secondary to *Chlamydia trachomatis*, respiratory syncytial virus (RSV), other respiratory viruses, and *Bordetella pertussis*. In the 1 to 24 month age group, mild to moderate pneumonia can be caused by respiratory viruses as well as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Mycoplasma pneumoniae*. Although viral pathogens dominate during years 2 to 5, the above bacterial pathogens are common. By the sixth year through 18 years of age, influenza virus A or B and adenovirus are common. At any age, severe pneumonia may be caused by *S aureus*, *S pneumoniae*, *M pneumoniae*, *H influenzae B*, and group A streptococci.

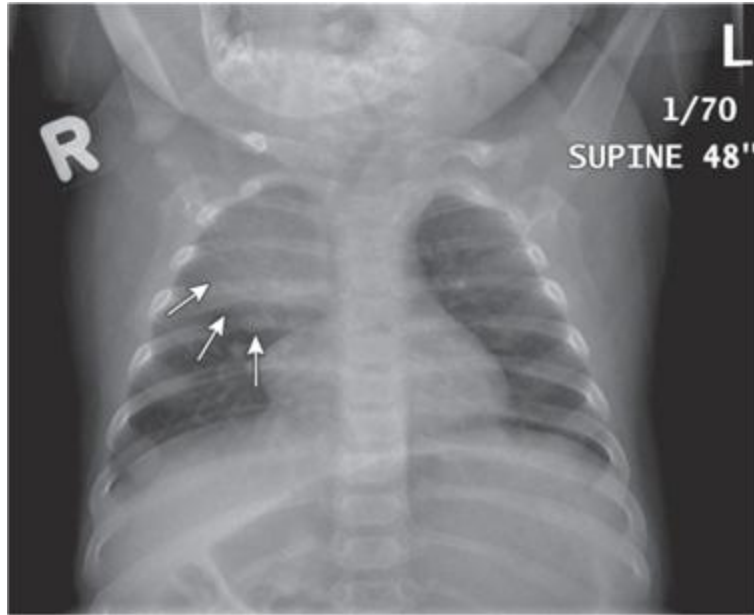
### CLINICAL FEATURES

Clinical features of pneumonia are quite variable. In addition to the age of the patient, factors that affect the clinical presentation of pediatric pneumonia include the specific respiratory pathogen, the severity of the disease, and underlying illnesses. Tachypnea is the most commonly physical sign; other signs and symptoms of pneumonia include respiratory distress, rales, or decreased breath sounds. The absence of these findings in a well appearing child makes pneumonia unlikely. Neonates and young infants with pneumonia may present with a sepsis syndrome, and signs and symptoms can be nonspecific: fever or hypothermia, apnea, tachypnea, poor feeding, vomiting, diarrhea, lethargy, grunting, bradycardia, and shock. In

older children, signs and symptoms of pneumonia are similar to adults and include fever, abnormal lung sounds, cough, and pleuritic chest pain. Possible associated findings may include headache, malaise, wheezing, rhinitis, conjunctivitis, pharyngitis, and rash. The clinical manifestations of bacterial and viral pneumonias overlap, making the clinical distinction problematic. Lower lobe pneumonias may cause significant abdominal pain and distention mimicking acute appendicitis.

## **DIAGNOSIS AND DIFFERENTIAL**

Though chest radiography is the gold standard for the diagnosis of pneumonia, clinical diagnosis is reasonable: fever, cough, and focal findings on the lung exam along with tachypnea and possibly hypoxemia comprise the classic clinical diagnostic criteria. If obtained, chest radiography may demonstrate a segmental or lobar infiltrate suggestive of bacterial pneumonia; diffuse air-space disease, hyperinflation, peribronchial thickening or cuffing and atelectasis seen with viral and atypical pathogens; or pleural fluid suggestive of empyema. However, there is overlap in the radiographic appearance of bacterial and viral pneumonias, making this distinction problematic at times. In children, the thymus can be mistaken for a mediastinal mass or lobar pneumonia and this normal finding must be differentiated from pulmonary pathology ([Fig. 71-1](#)).



**FIGURE 71-1.** Arrows indicate a normal thymus. Rotation, apparent from the location of the heart, trachea, and clavicles, makes this thymus appear to be far right of midline. (Courtesy of BC Children’s Hospital, Vancouver, BC, Canada.)

Rapid viral antigen tests are available for RSV and influenza, and may be helpful in identifying a viral etiology in the emergency department, sparing unnecessary antibiotics. A complete blood count (CBC) may reveal lymphocytosis in viral infections or leukocytosis with a left shift in bacterial pneumonia, but is not usually required for diagnosis; *S pneumoniae* can cause hemolytic uremic syndrome with signs of hemolysis and thrombocytopenia on the CBC and renal failure on basic metabolic evaluation. Blood cultures are rarely helpful or positive in pediatric bacterial pneumonia.

Other conditions that may be confused with infectious pneumonia include: foreign body aspiration, congestive heart failure, atelectasis, tumors, congenital pulmonary anomalies, aspiration pneumonitis, poor inspiration or technical difficulties with the chest radiograph, allergic alveolitis, and chronic pulmonary diseases.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

The ED care of pediatric pneumonia includes supportive care (management of hypoxia, dehydration, and fever), in addition to antibiotic therapy for suspected bacterial causes.

1. Provide oxygen for patients with significant respiratory distress or oxygen saturations below 92% on room air.
2. Administer 20 mL/kg normal saline for dehydration associated with increased fluid losses associated with increased respiratory rate and fever.
3. Administer empiric antibiotics based on the likely etiologic agents given the patient's age and whether the patient is admitted to the hospital or discharged home ([Table 71-1](#)).

Age Group	Inpatient Treatment	Outpatient Treatment
Neonates	Ampicillin 50 milligrams/kilogram IV AND cefotaxime 50 milligrams/kilogram IV OR gentamicin 2.5 to 5.0 milligrams/kilogram IV	Not recommended
1-3 months	Afebrile: erythromycin 10 milligrams/kilogram IV Febrile: cefuroxime 50 milligrams/kilogram IV +/- erythromycin IV	Not recommended
3 months-5 years	Ampicillin 50 milligrams/kilogram IV OR cefuroxime 50 milligrams/kilogram IV +/- erythromycin IV	Amoxicillin 40 to 50 milligrams/kilogram/dose twice daily OR amoxicillin-clavulanate 22.5 milligrams/kilogram /dose twice daily OR cefuroxime axetil 15 milligrams/kilogram/dose twice daily
5-18 years	Ampicillin 50 milligrams/kilogram IV AND erythromycin 10 milligrams/kilogram IV Alternative: Cefuroxime 50 milligrams/kilogram IV +/- erythromycin	Azithromycin 10 milligrams/kilogram × 1 on day 1 then 5 milligrams/kilogram daily for 4 days OR amoxicillin-clavulanate OR cefuroxime axetil

**TABLE 71-1** Empiric Treatment for Pneumonia in Healthy Children

4. Consider treatment with **albuterol** (2.5 to 5.0 milligrams by nebulizer or 4 to 8 puffs of MDI with a spacer) for pneumonia associated with wheezing and prolonged expiratory phase. For bronchiolitis, racemic epinephrine is more likely to be of benefit than  $\beta$ -agonists (see [Chapter 70](#)).

Most previously healthy infants over 3 months of age and children with pneumonia are treated as outpatients. The exact pulse oximetry threshold at which an otherwise well appearing young child with pneumonia should be admitted to the hospital is unknown, though commonly  $<92\%$  on room air. Indications for admission include age younger than 3 months, hypoxia, a history of apnea or cyanosis, toxic appearance, significant respiratory distress, dehydration, vomiting, failed outpatient therapy, immunocompromised state, associated pleural effusion or pneumatocele, or an unreliable care taker. Pediatric intensive care unit admission is appropriate for children with severe respiratory distress or impending respiratory failure.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 121, "Pneumonia in Infants and Children," by Joseph E. Copeland.

# CHAPTER 72

## Pediatric Heart Disease

Garth D. Meckler

There are 6 common clinical presentations of pediatric heart disease: cyanosis, shock, congestive heart failure (CHF), pathologic murmur, hypertension, and syncope. [Table 72-1](#) lists the most common lesions in each category. While cyanosis and shock typically appear in the first weeks of life and are often dramatic in their presentation, the symptoms of CHF may be subtle and include respiratory distress or feeding intolerance, which may be misdiagnosed as viral upper respiratory tract illness, especially in winter months. A high index of suspicion must therefore be maintained in order to make the correct diagnosis. This chapter focuses on conditions producing cardiovascular symptoms seen in the emergency department (ED) that require immediate recognition, therapeutic intervention, and prompt referral to a pediatric cardiologist.

Cyanosis	TGA, TOF, TA, Tat, TAVR
Shock	Coarctation, HPLHS
Congestive heart failure	Coarctation, PDA, ASD, VSD (See Table 72-2 for other causes)
Murmur	Shunts: VSD, PDA, ASD Obstructions (eg, valvular stenosis) Valvular incompetence
Hypertension	Coarctation
Syncope	
Cyanotic	TOF
Acyanotic	Critical AS

*Key:* AI = aortic insufficiency, AS = aortic stenosis, ASD = atrial septal defect, HPLHS = hypoplastic left heart syndrome, PDA = patent ductus arteriosus, TA = truncus arteriosus, Tat = tricuspid atresia, TAPVR = total anomalous pulmonary venous return, TGA = transposition of the great arteries, TOF = tetralogy of Fallot, VSD = ventricular septal defect.

**TABLE 72-1** Clinical Presentation of Pediatric Heart Disease

The evaluation of an asymptomatic murmur is a nonemergent diagnostic workup that can be done on an outpatient basis. Innocent murmurs, often



described as flow murmurs, are of low intensity, are brief, and occur during systole. In general, common pathologic murmurs in children are typically harsh, holosystolic, continuous, or diastolic in timing and often radiate. They may be associated with abnormal pulses or symptoms such as syncope or CHF.

The treatment of dysrhythmias is discussed in [Chapter 3](#), pediatric hypertension is discussed in [Chapter 26](#), and syncope is discussed in [Chapter 78](#). Chest pain is usually of benign etiology in children, though may occasionally represent congenital (eg, aberrant left coronary artery) or acquired (eg, Kawasaki disease, myocarditis, pericarditis, cardiomyopathy) heart disease. Myocarditis and cardiomyopathy are covered in [Chapter 24](#), chest pain and acute coronary syndrome in [Chapters 17](#) and [18](#), and Kawasaki disease in [Chapter 83](#).

## CYANOSIS AND SHOCK

Cardiac causes of cyanosis and shock typically present in the first 2 weeks of life and present in the critically ill neonate. The differential diagnosis, however, is broad at this age, and, in addition to congenital heart disease, the clinician should consider infection (sepsis, pneumonia), metabolic disease (see [Chapter 79](#)) and nonaccidental trauma. For the neonate presenting with cyanosis, the hyperoxia test helps to differentiate respiratory disease from cyanotic congenital heart disease (although imperfectly). When placed on 100% oxygen, the infant with cyanotic congenital heart disease will fail to demonstrate an increase in PaO<sub>2</sub>, while those with respiratory causes will often respond with an improvement in pulse oximetry.

### Clinical Features

Acral cyanosis (blue discoloration of the distal extremities) can be normal in the neonate, but central cyanosis (including the mucus membranes of the mouth) is the cardinal feature of cyanotic congenital heart disease.

Appreciation of cyanosis in dark-skinned neonates may be difficult, and an accurate set of vital signs including pulse oximetry and 4-extremity blood pressures is essential. Cyanosis associated with a heart murmur strongly suggests congenital heart disease, but the absence of a murmur does not exclude a structural heart lesion. The cyanotic infant may be tachypnic, as well, though the increased respiratory rate in cyanotic heart disease is often effortless and shallow unless associated with congestive heart failure, which is rare in the first weeks of life.

Shock with or without cyanosis, especially during the first 2 weeks of life, should alert the clinician to the possibility of ductal-dependent congenital heart disease in which systemic (shock) or pulmonary (cyanosis) blood flow depends on patency of the fetal ductus arteriosus. Shock in the neonate is recognized by inspection of the patient's skin for pallor (or, more often, an "ashen grey" appearance), mottling, and cyanosis, and assessment of the mental status appropriate for age. Mental status changes include apathy, irritability, or frank lethargy. Tachycardia and tachypnea may be the initial signs of impending cardiovascular collapse. Distal pulses should be assessed for quality, amplitude, and duration, and a differential between preductal (right brachial) and postductal (femoral) pulses or blood pressure is classic for ductal-dependent lesions such as coarctation of the aorta.

## **Diagnosis and Differential**

The workup for congenital heart disease begins with chest radiograph and electrocardiogram (ECG) with pediatric analysis. Chest radiographs are assessed for heart size, shape, and pulmonary blood flow. An abnormal right position of the aortic arch may be a clue to the diagnosis of congenital cardiac lesion. Increased pulmonary vascularity may be seen with significant left-to-right shunting or left-sided failure. Decreased pulmonary blood flow is seen with right-sided outflow lesions such as pulmonic stenosis. Cyanotic heart lesions often demonstrate right axis deviation and right ventricular hypertrophy on ECG while left outflow obstruction (eg, coarctation of the aorta) may show left ventricular hypertrophy. Echocardiography is generally required to define the diagnosis.

The differential diagnosis for cyanosis or shock due to congenital heart disease typically includes cyanotic lesions: transposition of the great vessels, tetralogy of Fallot, and other forms of right ventricular outflow tract obstruction or abnormalities of right heart formation. Acyanotic lesions that can present with shock include severe coarctation of the aorta, critical aortic stenosis, and hypoplastic left ventricle. It should be noted that cyanosis may accompany shock of any cause.

*Transposition of the great vessels* represents the most common cyanotic defect presenting in the first week of life. This entity is easily missed due to the absence of cardiomegaly or murmur (unless there is a coexistent ventricular septal defect [VSD]). Symptoms (before shock) include central cyanosis, increase respiratory rate, and/or feeding difficulty. There is usually a loud and single S2. Chest radiographs may show an “egg on a string” shaped heart with a narrow mediastinum and increased pulmonary vascular markings. ECG may show right-axis deviation and right ventricular hypertrophy.

*Tetralogy of Fallot* is the most common cyanotic congenital heart disease overall, and can present with cyanosis later in infancy or childhood. Physical examination reveals a holosystolic murmur of ventricular septal defect, a diamond-shape murmur of pulmonary stenosis, and cyanosis. Cyanotic spells in the toddler may be relieved by squatting. Chest radiograph may show a boot-shape heart with decreased pulmonary vascular markings or a right-sided aortic arch. The ECG often demonstrates right ventricular hypertrophy and right axis deviation.

*Hypercyanotic episodes*, or “*tet spells*,” may bring children with tetralogy of Fallot to the ED in dramatic fashion. Symptoms include paroxysmal dyspnea, labored respirations, increased cyanosis, and syncope. Episodes frequently follow exertion due to feeding, crying, or straining with stools and last from minutes to hours.

*Left ventricular outflow obstruction syndromes* may present with shock, with or without cyanosis. Several congenital lesions fall into this category, but in all these disorders, systemic blood flow is dependent on a large contribution of shunted blood through a patent ductus arteriosus. When the ductus closes, infants present with decreased or absent perfusion, pallor or an ashen appearance, hypotension, tachypnea, and severe lactic acidosis.

Diminished lower extremity pulses and BP, particularly compared to right brachial pulse and BP, is classic for coarctation of the aorta.

## **Emergency Department Care and Disposition**

1. Cyanosis and respiratory distress are first managed with high-flow oxygen, cardiac and oxygen monitoring, and a stable intravenous or intraosseous line. *Caveat:* Neonates tolerate low oxygen saturations well due to oxygen-avid fetal hemoglobin; oxygen is a potent pulmonary vasodilator and may lead to “pulmonary steal” of systemic blood flow, worsening systemic shock in ductal-dependent systemic blood flow such as coarctation of the aorta. Treatment with prostaglandins (see below) is critical in these instances.
2. For severe shock in infants suspected of having shunt-dependent lesions, **prostaglandin E<sub>1</sub>** should be given in an attempt to reopen the ductus. Treatment begins with 0.05 to 0.1 microgram/kilogram/min; this may be increased to 0.2 microgram/kilogram/min if there is no improvement. Side effects include fever, skin flushing, diarrhea, and periodic apnea.
3. Immediate consultation should be obtained with a pediatric cardiologist and, if the patient is in shock, a pediatric intensivist.
4. Management of hypercyanotic spells consists of positioning the patient in the knee-to-chest position and administration of **morphine** sulfate 0.2 milligram/kilogram SC, IM, or IO. Resistant cases should prompt immediate consultation with a pediatric cardiologist for consideration of phenylephrine for hypotension or propranolol for tachycardia.
5. Noncardiac causes of symptoms should be considered and treated appropriately, including a fluid challenge of 10 to 20 mL/kg of **normal saline** solution, and empiric administration of antibiotics as indicated. Fluids should be administered more judiciously to neonates with congenital heart disease, typically in 10 mL/kg boluses.
6. Epinephrine is the initial drug of choice for hypotension. An infusion is started at 0.05 to 0.5 microgram/kilogram/min and titrated to the desired blood pressure.

By definition, these children are critically ill and require admission, usually to the neonatal or pediatric intensive care unit.

## **CONGESTIVE HEART FAILURE**

### **Clinical Features**

Congestive heart failure from congenital or acquired heart disease typically presents after the neonatal period, typically in the second or third month of life (congenital) or later in childhood (acquired causes). The distinction between pneumonia and CHF in infants requires a high index of clinical suspicion and is often difficult. Pneumonia can cause a previously stable cardiac condition to decompensate; thus, both problems can present simultaneously. Presenting symptoms include poor feeding, diaphoresis, irritability or lethargy with feeding, weak cry, and, in severe cases, grunting, nasal flaring, and respiratory distress. Note that the early tachypnea of CHF in infants is typically “effortless” and the first manifestation of decompensation, followed by increased work of breathing and rales on examination.

### **Diagnosis and Differential**

Cardiomegaly evident on chest radiograph is universally present except in constrictive pericarditis. A cardiothoracic index greater than 0.6 is abnormal. The primary radiographic signs of cardiomegaly on the lateral chest radiograph are an abnormal cardiothoracic index and lack of retrosternal air space due to the direct abutment of the heart against the sternum.

Once CHF is recognized, age-related categories simplify further differential diagnosis ([Table 72-2](#)). Congenital cardiac causes of CHF are best categorized by age of onset. Early-onset CHF is associated with ductal-dependent lesions such as coarctation of the aorta and may be abrupt in onset; persistent patent ductus arteriosus (PDA) may also present in the

neonatal period with CHF. Rarely, sustained tachyarrhythmias may present with CHF in the neonatal period. By contrast, lesions that result in pulmonary over-circulation such as VSD or atrial septal defect (ASD) present with gradual development of failure in the second or third month of life. Onset of CHF after age 3 months usually signifies acquired heart disease such as cardiomyopathy or myocarditis. The exception is when pneumonia, endocarditis, or another complication causes a congenital lesion to decompensate.

Age	Spectrum		
1 min	Noncardiac origin: anemia (isoimmunization), meconium aspiration, acidosis, asphyxia, hypoglycemia, supraventricular tachycardia (intra-uterine)	} Congenital or acquired	
1 h			Hypocalcemia, sepsis
1 d	PDA in premature infants	} Congenital	
1 wk	HPLHS		
2 wk	Coarctation		
1 mo	Ventricular septal defect		
3 mo	Large atrial septal defect, supraventricular tachycardia		
1 y	Myocarditis Cardiomyopathy Severe anemia	} Acquired	
10 y			Rheumatic fever

Key: HPLHS = hypoplastic left heart syndrome, PDA = patent ductus arteriosus.

**TABLE 72-2** Differential Diagnosis of Congestive Heart Failure Based on Age at Presentation

Cardiomyopathy presents with respiratory distress and feeding difficulties. A pathologic gallop (S3 and or S4) is key to recognition. Rales and organomegaly are often present and cardiomegaly and pulmonary vascular congestion are noted on chest radiography.

Myocarditis is often preceded by a viral respiratory illness and needs to be differentiated from pneumonia. As with pneumonia, the infant usually presents in distress with fever, tachypnea, and tachycardia. ECG may show diffuse ST changes, dysrhythmias, or ectopy, which is associated with an increased risk of sudden death. Chest radiograph shows cloudy lung fields from inflammation or pulmonary edema. Cardiomegaly with poor distal pulses and prolonged capillary refill, however, distinguish it from common

pneumonia. Once cardiomegaly is discovered, hospital admission and an echocardiogram are indicated.

Usually pericarditis presents with pleuritic and positional chest pain. Muffled heart sounds and a friction rub may be present on physical examination. Cardiomegaly is seen on a chest radiograph. An echocardiogram is performed urgently to distinguish a pericardial effusion from dilated or hypertrophic cardiomyopathy and to determine the need for pericardiocentesis.

If an infant presents in pure right-side CHF, the primary problem is most likely to be pulmonary, such as cor pulmonale. In early stages, periorbital edema is often the first noticeable sign. This may progress to hepatomegaly, jugular venous distention, peripheral edema, and anasarca.

## **Emergency Department Care and Disposition**

1. The infant who presents with mild tachypnea, hepatomegaly, and cardiomegaly should be seated upright in a comfortable position, oxygen should be given, and the child should be kept in a neutral thermal environment to avoid metabolic stresses imposed by hypothermia or hyperthermia.
2. If the work of breathing is increased or CHF is apparent on chest radiograph, 1 to 2 milligrams/kilogram **furosemide** should be administered parenterally.
3. Hypoxemia is usually corrected by administration of oxygen, fluid restriction, and diuresis, although continuous positive airway pressure is sometimes necessary.
4. Stabilization and improvement of left ventricular function is often first accomplished with inotropic agents. **Digoxin** is used in milder forms of CHF. The appropriate first digitalizing dose to be given in the ED is 0.02 to 0.03 milligram/kilogram.
5. When CHF progresses to cardiogenic shock (absent distal pulses and decreased end-organ perfusion), continuous infusions of inotropic agents, such as **dopamine** or **dobutamine**, are indicated instead of digoxin. The initial starting range is 2 to 10 micrograms/kilogram/min.

6. Aggressive management of secondary derangements, including respiratory insufficiency, acute renal failure, lactic acidosis, disseminated intravascular coagulation, hypoglycemia, and hypocalcaemia should be implemented.
7. Definitive diagnosis and treatment of congenital defects presenting with CHF often requires cardiac catheterization followed by surgical repair. See the previous section for recommendations regarding administration of prostaglandin E<sub>1</sub> as a temporizing measure before surgery.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see [Chapter 122A](#), “Pediatric Heart Disease: Congenital Heart Defects,” by Linton L. Yee and Garth D. Meckler, and [Chapter 122B](#) Pediatric Heart Disease: Acquired Heart Disease by Linton L. Yee and Garth D. Meckler.



## CHAPTER 73

# Vomiting and Diarrhea in Infants and Children

Stephen B. Freedman

Gastroenteritis is a major public health problem, accounting for up to 20% of all acute care outpatient visits to hospitals. Most children who come to the emergency department because of vomiting and/or diarrhea have a self-limited viral disorder. Nevertheless, loss of water and electrolytes can lead to clinical dehydration and may result in hypovolemic shock or life-threatening electrolyte disturbances.

## CLINICAL FEATURES

The evaluation of the child's hydration status is the cornerstone to clinical management, regardless of whether the presenting complaint is vomiting or diarrhea ([Table 73-1](#)). Viral, bacterial, and other infectious organisms can cause gastroenteritis, and spread most commonly occurs by the fecal-to-oral route. Viral pathogens cause disease by invading tissue and altering the intestine's ability to absorb water and electrolytes. Bacterial pathogens cause diarrhea by producing enterotoxins and cytotoxins and by invading the intestine's mucosal absorptive surface. Dysentery occurs when bacteria invade the mucosa of the terminal ileum and colon, producing diarrhea with blood, mucus, or pus. [Table 73-2](#) lists common infectious agents, clinical features, and treatments of diarrhea in children. Infants are at greater risk for rapid dehydration and hypoglycemia as are those with chronic illnesses, high-risk social situations, or malnutrition.

Sign	Score = 0	Score = 1	Score = 2
General appearance	Normal	Thirsty, restless, lethargic, or irritable	Drowsy or not responsive, limp, cold sweaty
Eyes	Normal	Slightly sunken	Very sunken
Tongue	Moist	Sticky	Dry
Tears	Normal	Decreased	None

Score: 0 = no dehydration, 1 to 4 some dehydration, 5 to 8 severe dehydration.

**TABLE 73-1** Clinical Dehydration Score

Organism	Clinical Features	Antimicrobial Therapy
<i>Campylobacter</i>	Diarrhea, abdominal pain, fever, malaise Often hematochezia in infants	Typically self limited; 20% have relapse or prolonged symptoms Treat if: moderate-severe symptoms, relapse, immunocompromised, day care and institutions Options: erythromycin, azithromycin, ciprofloxacin
<i>Escherichia coli</i> -Shiga toxin producing	Bloody or nonbloody diarrhea, severe abdominal pain	None indicated; debated risk of increased incidence of hemolytic uremic syndrome with treatment
<i>E coli</i> -enteropathogenic	Severe watery diarrhea	Options: trimethoprim-sulfamethoxazole, azithromycin, ciprofloxacin
<i>E coli</i> -enterotoxigenic	Moderate watery diarrhea, abdominal cramps	Treat if severe symptoms Options: trimethoprim-sulfamethoxazole, azithromycin, ciprofloxacin
<i>E coli</i> -enteroinvasive	Fever, bloody or nonbloody dysentery	Treat if dysentery Options: trimethoprim-sulfamethoxazole, azithromycin, ciprofloxacin
<i>E coli</i> -enteroaggregative	Watery, occasionally bloody diarrhea	Options: trimethoprim-sulfamethoxazole, azithromycin, ciprofloxacin

<i>Salmonella</i>	Mild: watery diarrhea, mild fever, abdominal cramps Typhoid fever: high fever, constitutional symptoms, abdominal pain, hepatosplenomegaly, rose spots, altered mental status	Typically self limited Treat if: <3 mo of age, hemoglobinopathy, immunodeficiency, chronic GI tract disease, malignancy, severe colitis, bacteremia, sepsis Gastroenteritis: ampicillin, amoxicillin, trimethoprim-sulfamethoxazole, cefotaxime, ceftriaxone, fluoroquinolone Invasive disease: cefotaxime, ceftriaxone
<i>Shigella</i>	Mild: watery stools without constitutional symptoms Severe: fever, abdominal pain, tenesmus, mucoid stools, hematochezia	Typically self limited (48 to 72 h) Treat if: immunocompromised, severe disease, dysentery or systemic symptoms Options: azithromycin, trimethoprim-sulfamethoxazole, ceftriaxone, ciprofloxacin
<i>Yersinia</i>	Bloody diarrhea with mucus, fever, abdominal pain Pseudoappendicitis syndrome: fever, right lower quadrant pain, leukocytosis	Typically self limited Treat if: sepsis, non-GI infections, immunocompromised, excess iron storage condition (desferrioxamine use, sickle cell anemia, thalassemia) Options: trimethoprim-sulfamethoxazole, aminoglycosides, cefotaxime, fluoroquinolones, tetracycline, doxycycline

**TABLE 73-2** Clinical Features and Treatment of Bacterial Gastroenteritis

## DIAGNOSIS AND DIFFERENTIAL

Acute gastroenteritis is a clinical diagnosis and is typically defined by the presence of three or more diarrheal stools in a 24-hour period. Because gastroenteritis induced dehydration is usually isotonic, serum electrolytes are not routinely helpful unless signs of severe dehydration are present or intravenous rehydration fluids will be administered. The exception is infants in the first 6 months of life, in whom significant sodium abnormalities may develop. Bedside glucose should be checked in all patients with altered mental status; hypoglycemia can develop rapidly in the

setting of protracted vomiting or diarrhea in infants and toddlers. Stool cultures are reserved for cases in which the child has travelled to a high-risk country, is highly or persistently febrile, has > 10 stools in the previous 24 hours, or has blood in the stool. In the setting of a known outbreak of *Escherichia coli* O157:H7 consider stool cultures and blood tests to check for evidence of hemolysis, thrombocytopenia, and acute renal failure. The fecal leukocyte and guaiac tests have poor sensitivity and limited use.

Although diarrhea is the most prominent symptom of acute gastroenteritis in infants and children, other etiologies of diarrhea that may result in significant morbidity must be considered: bacterial colitis, Hirschsprung disease, partial obstruction, inflammatory bowel disease and hemolytic uremic syndrome. Acute appendicitis typically manifests with abdominal pain followed by vomiting associated with constipation, however, it may also cause diarrhea, particularly once the appendix has perforated (see [Chapter 74](#) “Pediatric Abdominal Emergencies”).

Similarly, vomiting is a common and nonspecific presentation for other disease processes, such as otitis media, urinary tract infection, sepsis, malrotation, intussusception, increased intracranial pressure, metabolic acidosis, and drug or toxin ingestions. Consequently, *isolated vomiting*, though most often of viral origin, requires a careful and thoughtful evaluation before being diagnosed as acute gastroenteritis. Specific clinical findings, such as bilious or bloody vomitus, hematochezia, or abdominal pain, should trigger concerns for a disease process other than simple viral gastroenteritis.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

### **Dehydration (Vomiting and/or Diarrhea)**

1. Because most cases are self-limited, oral rehydration is generally all that is necessary. Vomiting is not a contraindication to oral rehydration; the key is to give small amounts of the solution frequently. Use of a commercially available **oral rehydration solution** (ORS) containing 45 to 60 mmol/L of sodium is recommended. Many other beverages

traditionally suggested for children with vomiting and diarrhea, such as tea, juice, or sports drinks, are deficient in sodium and may provide excessive sugar, resulting in amplified fluid losses. Give 50 to 100 mL of ORS/kilogram of body weight, plus additional ORS to compensate for ongoing losses. Aim for about 1 ounce (30 mL) of ORS per kilogram of body weight per hour.

2. Administer intravenous or intraosseous isotonic crystalloid to children with severe dehydration, hemodynamic compromise, or when altered mental status precludes safe oral administration of fluid. Give **normal saline** as a 20 mL/kg bolus every 20 min until perfusion improves and urine output is adequate.
3. Treat hypoglycemia with 10% dextrose (5 mL/kg) in infants or 25% dextrose (2 mL/kg) in toddlers and older children.

## **Vomiting**

1. Treat dehydration, hypoglycemia, and electrolyte abnormalities as above.
2. Consider **ondansetron** as an adjunct to oral rehydration therapy in children with persistent vomiting at a dose of 0.15 milligram/kilogram/dose. Oral dosing is preferred as the main objective is to support the success of oral rehydration. Dopamine receptor agonists such as promethazine are not recommended in children and are contraindicated in young children.
3. Most children can be discharged if they are tolerating oral rehydration, have adequate urine output, and ongoing fluid losses have been minimized. Continuation of a normal diet (including lactose-containing milk or formula) is recommended. Patients who cannot tolerate oral fluids, have significant ongoing losses, severe electrolyte abnormalities, or surgical abdominal processes require admission to the hospital.

## **Diarrhea**

1. Treat dehydration and hypoglycemia as above.
2. Children with mild diarrhea who are not dehydrated may continue routine feedings. Do not withhold feedings > 4 hours in a dehydrated

child or for any length of time in a child who is not dehydrated. There is no need to dilute formula because more than 80% of children with acute diarrhea can tolerate full-strength milk safely.

3. Antidiarrheal and antimotility agents such as loperamide, are not recommend in children and contraindicated in young children.
4. Antibiotics are unnecessary for the vast majority of children with acute gastroenteritis and may be associated with increased risk for hemolytic uremic syndrome. See [Table 73-2](#) for specific treatment recommendations by pathogen.
5. All infants and children who appear toxic or have high-risk social situations, significant dehydration, significant ongoing fluid losses, altered mental status, inability to drink, or laboratory evidence of hemolytic anemia, thrombocytopenia, azotemia, or a significant dysnatremia should be admitted.
6. Children who respond to oral or intravenous hydration can be discharged. Instructions should be given to return to the emergency department or seek care with the primary physician if the child becomes unable to tolerate oral hydration, develops bilious vomiting, becomes less alert, or exhibits signs of dehydration, such as no longer wetting diapers. Dietary recommendations include a diet high in complex carbohydrates, lean meats, vegetables, fruits, and yogurt. Fatty foods and foods high in simple sugars should be avoided. The BRAT diet is discouraged because it does not provide adequate energy sources.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 123, “Vomiting, Diarrhea, and Dehydration in Children,” by Stephen B. Freedman and Jennifer D. Thull-Freedman.

# **CHAPTER 74**

## **Pediatric Abdominal Emergencies**

Mark R. Zonfrillo

### **ABDOMINAL PAIN**

Pediatric abdominal pain is a common presenting complaint in the emergency department. The assessment of acute abdominal pain can be challenging given the preverbal state of young children, the varied number of diagnoses that present similarly, and increasing appreciation of risks associated with pediatric diagnostic imaging.

#### **Clinical Features**

Presenting signs and symptoms differ by age. The key gastrointestinal (GI) signs and symptoms include pain, vomiting, diarrhea, constipation, bleeding, jaundice, and masses. Pain in children younger than 2 years typically manifests as fussiness, irritability, lethargy, or grunting. Toddlers and school age children often localize pain poorly and point to their umbilicus. Pain may be peritoneal and exacerbated by motion, or obstructive, spasmodic, and associated with restlessness. Abdominal pain may originate from non-GI sources, and associated symptoms may help localize extra-abdominal causes such as cough with pneumonia, sore throat in streptococcal pharyngitis, and rash in Henoch-Schönlein purpura (HSP).

Vomiting and diarrhea are common in children. These symptoms may be the result of a benign process or indicate the presence of a life-threatening process. Bilious vomiting is almost always indicative of a serious process, especially in the neonate. GI bleeding can result from upper or lower sources. Upper GI bleeding in children presents with hematemesis, which is often frightening to caretakers, but rarely serious in an otherwise healthy

infant or child. Lower GI bleeding presents with melena or hematochesia, and the distinction between painless and painful rectal bleeding can help differentiate likely etiologies (see GI Bleeding below). Jaundice can be an ominous sign, and all icteric infants should be fully evaluated for sepsis, congenital infections, hepatitis, anatomic problems, and enzyme deficiencies. Abdominal masses may be asymptomatic (eg, Wilms tumor) or associated with painless vomiting (eg, pyloric stenosis) or colicky abdominal pain (eg, intussusception).

## **Diagnosis and Differential**

Obtain a thorough history from parent and child (if possible), including the quality and location of pain, chronology of events, feedings, bowel habits, fever, weight changes, and other systemic signs and symptoms. Begin the physical examination with an assessment of the child's overall appearance, vital signs, and hydration status. The patient should be disrobed, and thorough inspection with nontouch maneuvers should precede auscultation and palpation. Extraabdominal areas including the chest, pharynx, testes, scrotum, inguinal area, and neck should also be evaluated. Adolescent females with lower abdominal pain may require a bimanual exam. The likely etiologies of abdominal pain change with age. [Table 74-1](#) classifies emergent and nonemergent conditions by age group.



Age	Emergent	Nonemergent
0 to 3 mo old	Necrotizing enterocolitis Volvulus Testicular torsion Incarcerated hernia Trauma Toxic megacolon Tumor	Colic Acute gastroenteritis Constipation
3 mo to 3 y old	Intussusception Testicular torsion Trauma Volvulus Appendicitis Toxic megacolon Vaso-occlusive crisis	Acute gastroenteritis Constipation Urinary tract infections Henoch-Schonlein purpura (HSP)
3 y old adolescence	Appendicitis Diabetic ketoacidosis Vaso-occlusive crisis Toxic ingestion Testicular torsion Ovarian torsion Ectopic pregnancy Trauma Toxic megacolon Tumor	Constipation Acute gastroenteritis Nonspecific viral syndromes Streptococcal pharyngitis Urinary tract infections Pneumonia Pancreatitis Cholecystitis Renal stones HSP Inflammatory bowel disease Gastric ulcer disease/gastritis Ovarian cyst Pregnancy Pelvic inflammatory disease

Key: HSP = Henoch-Schönlein purpura.

**TABLE 74-1** Classification of Abdominal Pain by Age Group

*Neonates and Young Infants (0 to 3 Months)*

Life-threatening abdominal conditions in young infants include necrotizing enterocolitis, and malrotation with midgut volvulus. Other urgent conditions include pyloric stenosis, incarcerated hernias (see [Chapter 46](#)), testicular torsion, and nonaccidental trauma. Inconsolability, lethargy, and poor feeding may be the only indication of serious underlying disease. Bilious

vomiting in an infant indicates intestinal obstruction and should be considered a surgical emergency until proven otherwise. Common non-life-threatening causes of abdominal pain in young infants include colic (see [Chapter 67](#)) and constipation. Fever requires thorough investigation for a source (see [Chapter 66](#)). Other causes of irritability in infants should be considered, including hair or thread tourniquets of the digits and genitalia, and corneal abrasions (see [Chapter 67](#)).

Helpful studies in this age group include abdominal radiographs to identify obstruction or free air, abdominal ultrasound to diagnose pyloric stenosis, testicular torsion, and hernias, and upper GI contrast studies to identify malrotation. Useful laboratory studies include serum electrolytes to identify abnormalities resulting from vomiting and dehydration and a CBC and coagulation panel to identify DIC in abdominal catastrophe with perforation.

*Malrotation of the intestine* can present with life-threatening volvulus. Symptoms include bilious vomiting, abdominal distention, and obstipation, or, occasionally, streaks of blood in the stool. The vast majority of cases presents within the first month of life. Patients are ill appearing and may present in compensated or decompensated shock. Distended loops of bowel overriding the liver on abdominal radiographs, and a “bird’s beak” appearance on an upper GI series are suggestive of this diagnosis. Immediate surgical consultation and aggressive fluid resuscitation are critical to maximize outcomes.

*Pyloric stenosis* usually presents with progressive nonbilious, projectile vomiting occurring just after feeding. Infants with pyloric stenosis are often described by parents as ravenous. Pyloric stenosis occurs most commonly in the third or fourth week of life, is familial and more common in first-born males. A left upper quadrant pyloric mass, or “olive,” may be present, and peristaltic waves may be noted following a feeding trial in the ED. Ultrasound is the imaging modality of choice. Electrolytes may demonstrate a characteristic hypochloremic metabolic alkalosis, which must be corrected prior to definitive surgical care. While pyloric stenosis is not a surgical emergency, the resultant dehydration from persistent vomiting requires immediate medical treatment.

*Older Infants and Toddlers (3 Months to 3 Years)*

The differential diagnosis of acute abdominal pain in this age group includes intussusception, gastroenteritis (see [Chapter 73](#)), constipation, urinary tract disease (see [Chapter 75](#)), and nonaccidental trauma. Though less common in this age group, acute appendicitis and malrotation with midgut volvulus must be considered.

Imaging studies are guided by the differential diagnosis: radiographs can help rule out free air or obstruction, and confirm suspected constipation; ultrasound can help identify intussusception or appendicitis; and air-contrast enema is both diagnostic and potentially therapeutic for intussusception. A CBC and electrolytes may be helpful in evaluating complications of vomiting and diarrhea, and urinalysis will identify pyelonephritis as a potential cause of abdominal pain in this age group.

*Intussusception* occurs when one portion of the bowel telescopes into another, which can result in a partial or complete obstruction, bowel-wall edema, and eventually ischemia. The greatest incidence occurs between 6 to 18 months of age. The classic presentation of intermittent paroxysms of abdominal pain with pain-free intervals (or lethargy), vomiting (may be bilious) and “currant jelly stool” are not present in all patients. Stool guaiac testing may reveal occult blood. Providers must have a high index of suspicion for intussusception in patients presenting with nonspecific changes in mental status or who are ill appearing without any apparent etiology. In equivocal cases, ultrasound is a sensitive test with a high negative predictive value and may demonstrate the classic “target sign” (see [Fig. 74-1](#)). In more classic cases, air or barium enema can be both diagnostic and therapeutic. Radiologic reduction requires the presence of a pediatric surgeon for irreducible cases or perforation during the procedure.



**FIGURE 74-1.** Ultrasound image of intussusception showing the classic target appearance of bowel-within-bowel. (Reproduced with permission from *Emergency Ultrasound, 2nd ed.* © 2008, McGraw-Hill, New York, Figure 9-15-A.)

*Constipation*, defined as infrequent, hard stools, is a common cause of abdominal pain in children and may be a sign of either a pathologic (eg, Hirschsprung disease, cystic fibrosis, spinal cord abnormality) or functional process. History is key to the diagnosis. For neonates, verify passage of meconium in the first 24 hours of life. A rectal examination is recommended to assess presence of stool, rectal tone, sensation, and size of the anal vault. A careful lower extremity neurologic examination should be completed to assess for neuromuscular causes. A single upright abdominal radiograph may be helpful to visualize fecal retention or impaction, and to help rule out the concern for obstruction. Treatment in the ED with suppositories or enemas may be necessary, and outpatient maintenance therapy is essential to prevent recurrence. Admission is indicated for patients with impaction associated with vomiting, dehydration, and failure of outpatient treatment.

*Children (3 to 15 Years Old)*

Acute abdominal pain in children 3 to 15 years old includes a range of diagnoses, including appendicitis ([Chapter 43](#)), constipation (see above), gastroenteritis ([Chapter 73](#)), urinary tract infection ([Chapter 75](#)), streptococcal pharyngitis ([Chapter 68](#)), pneumonia ([Chapter 71](#)), pancreatitis ([Chapter 42](#)) and functional abdominal pain. An unusual but important cause of abdominal pain in this age group is *Henoch-Schönlein purpura* (HSP), which is discussed further below. The presence of a parent is helpful in examining younger children. Older children may not readily offer important history surrounding embarrassing topics such as constipation and genital pain. Adolescents should be interviewed alone to provide confidentiality and facilitate discussion of potentially important information surrounding sexual activity, substance use, and other sensitive subjects.

*Henoch-Schönlein purpura* is an idiopathic vasculitis of children between 2 and 11 years of age. HSP classically presents with acute abdominal pain, lower extremity purpura, and arthritis. Routine laboratory testing is not needed for classic cases, but can help rule out other conditions. A urinalysis should be obtained to identify hematuria, with subsequent renal function tests when blood is present. Abdominal pain in HSP is caused by bowel wall edema and GI vasculitis. Microscopic and even gross GI bleeding is not uncommon, though rarely life threatening. HSP is a known risk factor for intussusception in older children and this must be considered in patients with significant abdominal pain. Treatment of HSP is primarily supportive: joint pain responds well to non-steroidal antiinflammatory medications; severe abdominal pain may improve with corticosteroids. Consultation and follow-up with a pediatric rheumatologist or nephrologist may be necessary for more severe symptoms or renal involvement with hypertension.

## **GASTROINTESTINAL BLEEDING**

Upper GI (UGI) bleeding is distinguished from lower GI (LGI) bleeding anatomically at the Ligament of Treitz. The presentation of GI bleeding in children varies, and includes bright red blood in small strands or clots in emesis or stool, vomiting of gross blood (hematemesis), black tarry stools

(melena), or profuse bright red blood per rectum. Occult bleeding may present with pallor, fatigue, or anemia.

The first step in management is to evaluate the need for acute resuscitation, and distinguish the bleed as UGI or LGI. Confirmation through gastric or fecal occult blood testing is useful to distinguish substances that grossly mimic blood. A thorough history and physical examination is important to determine the location, amount, and etiology of the bleed. Examination for extragastrointestinal sources is also important (eg, epistaxis as a cause of hematemesis). The differential diagnosis of upper and lower GI bleeding by age group is summarized in [Table 74-2](#). In general, upper GI bleeding in children without underlying portal hypertension is rarely life threatening. While most causes of lower GI bleeding in children are also benign, potential emergent causes include vascular malformations, Meckel diverticula, hemolytic uremic syndrome, and intussusception. In cases of melena, evaluate for causes of upper abdominal bleeding, and HSP. In cases of bright red blood per rectum, evaluate for anal fissure, or hemorrhoids. For painless hematochezia, consider Meckel diverticulum, or arteriovenous malformation. For hematochezia in the setting of abdominal pain, the differential is larger including: HSP, hemolytic uremic syndrome, infections gastroenteritis, inflammatory bowel disease, milk protein allergy, intussusception and necrotizing enterocolitis.

Upper GI Bleeding		
< 2 Months	2 Months to 2 Years	>2 Years
Swallowed maternal blood	Gastroenteritis	Gastroenteritis
Stress ulcer	Toxic ingestion	Mallory-Weiss tear
Vascular malformation	Mallory-Weiss tear	Peptic ulcer disease
Hemorrhagic disease of newborn (vitamin K deficiency)	Vascular malformation	Toxic ingestion
Coagulopathy/bleeding diathesis	Esophagitis	Vascular malformation
	Stress ulcer	Gastritis
	Bleeding diathesis	Varices
	GI duplication	Hemato-bilia
	Foreign body	Foreign body
Lower GI Bleeding		
<2 Months	2 Months to 2 Years	>2 Years
Swallowed maternal blood	Anal fissure	Anal fissure
Milk allergy	Gastroenteritis	Gastroenteritis
Infectious colitis	Milk allergy	Hemorrhoids
Intussusception	Intussusception	Polyps
Volvulus	Volvulus	Colitis (infectious, ischemic)
Meckel diverticulum	Meckel diverticulum	Meckel diverticulum
Necrotizing enterocolitis	Hemolytic uremic syndrome	Intussusception
Vascular malformation	Henoch-Schönlein purpura	Hemolytic uremic syndrome
Hemorrhagic disease of newborn	Polyps: benign, familial	Henoch-Schönlein purpura
Hirschsprung disease	Inflammatory bowel disease	Inflammatory bowel disease
Congenital duplications	GI duplication	Angiodysplasia
	Dieulafoy lesion	Celiac disease
		Dieulafoy lesion
		Rectal ulcer syndrome
		Peptic ulcer disease

**TABLE 74-2** Age-Based Causes of Upper and Lower GI Bleeding

Laboratory studies and imaging should be completed as dictated by other associated signs and symptoms. Mild bleeding can typically be managed by a consultant on an outpatient basis. Moderate and severe bleeding requires acute resuscitation with isotonic crystalloid and, potentially, blood products. While acute shock should be treated immediately, overexpansion of intravascular volume should be avoided, particularly for variceal bleeding or more chronic conditions. Consultation with a pediatric surgeon, gastroenterologist, or critical care physician may be necessary, depending on the etiology.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 124, “Acute Abdominal Pain in Children” by Anupam B. Kharbanda and Rasha D. Sawaya; and Chapter 125, “Gastrointestinal Bleeding in Children” by Robert W. Schafermeyer and Emily MacNeill.



## **CHAPTER 75**

# **Pediatric Urinary Tract Infections**

Lance Brown

Urinary tract infections (UTIs) are relatively common from infancy through adolescence. The incidence and clinical presentation of pediatric UTIs change with age and sex.

## **CLINICAL FEATURES**

There are 3 age-based clinical presentations for pediatric UTIs. Neonates present with a clinical presentation indistinguishable from that of sepsis, and they may have symptoms that include fever, jaundice, poor feeding, irritability, and lethargy. Older infants and young children typically present with gastrointestinal complaints that may include fever, abdominal pain, vomiting, and a change in appetite. School-age children and adolescents typically present with adult-type complaints such as dysuria, urinary frequency, urgency, and hesitancy. Although the majority of infants and young children with fever and UTI have upper-track disease and require long-course antibiotic treatment, older children and adolescents without fever, flank pain, and flank tenderness are likely to have simple cystitis and can be treated with shorter course therapy similar to adults.

## **DIAGNOSIS AND DIFFERENTIAL**

The gold standard for confirming the diagnosis of pediatric UTI is the growth of a single urinary pathogen from a properly obtained urine culture. For infants and children in diapers, catheterization or, rarely, ultrasound guided suprapubic aspiration is required. For children who are toilet trained, urine may be collected as a supervised clean catch specimen. Bagged urine specimens have essentially no role in diagnosis of pediatric UTI.

Because younger children void frequently and do not store urine in the bladder long enough to accumulate leukocytes or nitrites, urinalysis is insensitive in this age group and culture should be sent regardless of dipstick results. Microscopic urinalysis is more specific and is typically considered positive for infection if more than 5 white blood cells per high power field and bacteria are seen. A positive microscopic urinalysis has a sensitivity of 65% for identifying culture-proven UTI. Neither urinary test strips nor microscopic urinalysis can be used to definitively rule out pediatric UTI, though evidence is mounting that delaying treatment until culture results return does not alter the long-term outcome and antibiotics can be safely withheld in the setting of a negative urinalysis and microscopy.

Adolescents may have urinary symptoms as a manifestation of a sexually transmitted disease such as *Chlamydia trachomatis*. An appropriate sexual history and pelvic examination may be indicated and helpful in making this diagnosis (for a discussion of sexually transmitted diseases, see [Chapter 86](#)).

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The treatment and disposition of infants and children with UTI depend on age and are based on the severity of concurrent symptoms. In general, antibiotics should not be given until after urine culture has been obtained.

1. Treat neonates for sepsis and obtain cultures of blood and CSF in addition to urine. Administer parenteral antibiotics and admit to the hospital: **ampicillin** (50 milligrams/kilogram/dose) plus **gentamicin** (3-5 milligrams/ kilogram/dose) or **ampicillin** (50 milligrams/kilogram/dose) plus **cefotaxime** (50 milligrams/kilogram/dose) are appropriate empiric choices.
2. Treat infants from 1 month to 2 years of age who are dehydrated, have persistent vomiting, appear ill or septic, or are medically complicated

typically with intravenous antibiotics such as **ceftriaxone**, **cefotaxime**, or **cefepime** (all are dosed at 50 milligrams/kilogram/dose).

Well appearing infants in this age group may be treated with oral antibiotics, which are as effective as parenteral treatment. A third generation cephalosporin such as cefpodoxime (5 milligrams/kilogram/dose given twice daily) or cefdinir (7 milligrams/kilogram/dose given twice daily), is recommended. Treat for 10 to 14 days, as short-course therapy is less effective in young children. High rates of resistance to **cephalexin**, **amoxicillin**, and **trimethoprim-sulfamethoxazole** are now widespread, though these may be appropriate based on local sensitivities.

3. Children older than 2 years who are otherwise doing well and tolerating oral fluids are treated as outpatients with oral antibiotics for at least 7 days with close follow-up with their primary doctors. Emergency physicians should be familiar with the antibiotic-resistance patterns in their geographic area. Appropriate oral antibiotic choices to treat pediatric UTIs include **cefixime** (8 milligrams/kilogram/d, divided twice daily), cefpodoxime (5 milligrams/kilogram/day divided twice daily) or cefdinir (7 milligrams/kilogram/dose given twice daily). Depending on local sensitivities, **trimethoprim/sulfamethoxazole** (3 to 6 milligrams/kilogram/dose TMP, 15 to 30 milligrams/kilogram/dose SMX, given twice daily), or **cephalexin** (10 to 25 milligrams/kilogram/dose, given 4 times a day), may be used.
4. Adolescent females with cystitis or acute pyelonephritis are treated similarly to adults (see [Chapter 53](#)).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 126, “Urinary Tract Infections in Infants and Children,” by Julie S. Byerley and Michael J. Steiner.

## **CHAPTER 76**

# **Seizures and Status Epilepticus in Children**

Maya Myslenski

The causes and manifestations of seizure activity are numerous, ranging from benign to life threatening. Precipitants of seizures can include head injury, structural brain abnormalities, fever, CNS infection, hypoglycemia, electrolyte abnormalities, hypoxemia, toxin exposure, dysrhythmias, metabolic disorders, congenital infections, or neurocutaneous syndromes.

## **CLINICAL FEATURES**

The clinical features of seizure activity depend on the area of the brain affected and can range from classic tonic-clonic movements to very subtle behavioral changes; they may be generalized (with loss of consciousness) or partial (with focal motor or behavioral features). Rhythmic repetitive movement, incontinence of bowel or bladder, postictal state after a seizure, and tongue biting are strong clues to a seizure.

Motor changes (tonic or clonic) may be focal or generalized, and seizures may present with atony (sudden loss of tone or “drop attack”) in some age groups. More subtle symptoms include staring spells (“absence”) or changes in mental status or behavior, which can be complex, such as automatisms (blinking, bicycling, or lip smacking in infants), vocalizations, or hallucinations.

Signs may include alteration in autonomic dysfunction, such as mydriasis; diaphoresis; tachypnea or apnea; tachycardia; hypertension and salivation; and postictal somnolence. Transient focal deficits may represent Todd’s paralysis following a seizure.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of seizure disorder is based primarily on history and physical examination. Bedside glucose testing should be performed on all children who are seizing or postictal, but the clinical scenario should direct additional laboratory and imaging tests. Screening tests for electrolytes are not indicated in most cases of childhood seizures including simple febrile seizures or first time afebrile seizures, unless otherwise indicated by the specific history. The suggested ED evaluation of differing clinical scenarios presenting with seizure is listed in [Table 76-1](#).

Clinical Scenario	Suggested ED Evaluation	Comments
Simple febrile seizure	Routine testing and imaging not indicated	Consider testing for source of fever (eg, urinalysis)
First afebrile seizure without focal deficits in ED	Routine testing not indicated. Routine LP not recommended	Consider outpatient MRI, EEG
Seizure in known epileptic	Serum drug levels for anti-epileptic medications	Some medication levels not immediately available. Low or high levels may cause seizures
Seizure in setting of ventriculo-peritoneal shunt	Plain radiographs of shunt <i>and</i> neuroimaging. Consider CSF evaluation from shunt-tap if febrile	Quick brain MRI preferred to CT if available. Neurosurgery consult required for shunt tap
Posttraumatic seizure	Consider head CT	Imaging not required for brief impact seizures
Febrile status epilepticus	Head CT and lumbar puncture	Additional evaluation for fever as indicated
Nonfebrile status epilepticus or new-onset focal seizure or focal neurologic deficit in ED	Neuroimaging Complete metabolic panel Consider toxicology screens ECG for possible arrhythmia	Consider nonaccidental trauma and toxic ingestions
Seizure in infants	Bedside glucose, complete metabolic panel, ionized calcium and serum magnesium, ammonia, lactate, urinalysis (look for ketones suggestive of metabolic disease). Consider CSF analysis and culture including HSV PCR	Consider nonaccidental trauma and inborn errors of metabolism. Consider urine organic acid and serum amino acid profile (results not available to ED)

**TABLE 76-1** Suggested ED Evaluation of Pediatric Seizures

*Status epilepticus*, traditionally defined as seizure activity lasting for > 30 min, or multiple seizures without a return to normal mental status between seizures. Five minutes has been suggested as an operational definition because seizures lasting longer than 5 min usually do not resolve without treatment. Status epilepticus is a medical emergency, and is more responsive to medications when treated early and aggressively.

Seizures must be distinguished from other events that masquerade as seizures in children such as breath-holding, syncope, gastroesophageal reflux (Sandifer syndrome), chorea (acute rheumatic fever), myoclonic jerks, rigors, startle reflex, tics, pseudo-seizures and night terrors. Primary seizures should be distinguished from seizures secondary to treatable or life-threatening causes such as trauma, infection, hypoglycemia, metabolic abnormalities, or electrolyte abnormalities.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The management of the seizing child requires immediate supportive care, termination of the seizure with medication, and correction of reversible causes such as hypoglycemia or metabolic abnormalities.

1. Ensure a patent airway, apply oxygen to all seizing patients, and assist ventilation as indicated. If rapid-sequence intubation is required, continuous EEG monitoring should be arranged as neuromuscular blockade obscures the ability to clinically assess for ongoing seizures.
2. Treat **hypoglycemia** with 4 to 5 mL/kg 10% dextrose in infants or 2 mL/kg 25% dextrose in older children IV/IO.
3. Administer **lorazepam**, 0.1 milligram/kilogram or **midazolam** 0.1 to 0.2 milligram/kilogram IV/IO. If vascular access is not available, administer midazolam 0.2 milligram/kilogram IN (maximum dose 10 milligrams). Repeat benzodiazepine dose in 5 min if seizure persists and prepare to support breathing and blood pressure with repeated dosing.

4. Administer *second-line anti-epileptic drugs* if more than 2 doses of benzodiazepine are required. For infants, use **phenobarbital** 20 milligrams/ kilogram IV/IO (maximum 800 milligrams) and for older children use **fosphenytoin** 20 PE/kilogram IV/IO over 20 min.
5. For *refractory status epilepticus*, administer *third-line* agents such as: **valproic acid** 20 milligrams/kilogram IV/IO, or **levetiracetam** 20 to 30 milligrams/kilogram IV/IO.
6. *Fourth-line* treatment strategies include continuous infusions in the ICU setting of propofol, midazolam, or pentobarbital, or induction of general inhaled anesthesia, such as: **propofol** 0.5 to 2.0 milligram/kilogram or 1.5 to 4.0 milligrams/kilogram/h IV/IO; or **midazolam** continuous infusion: 0.05 to 0.4 milligram/kilogram/h IV/IO.
7. Treat electrolyte abnormalities as follows: for hyponatremia <120 mEq/L, 3% NaCl 4 to 6 mL/kg, for hypocalcemia, <7 milligrams/dL of calcium and <0.8 mmol/L of ionized calcium, 0.3 mL/kg of 10% calcium gluconate, over 10 min, and for hypomagnesemia <1.5 mEq/L, 50 milligrams/kilogram of magnesium sulphate over 20 min.

Children who present to the ED following a brief seizure and who regain a normal mental status without focal neurologic deficits are candidates for discharge and outpatient follow-up. Patients presenting to the ED with ongoing seizures meet the definition for status epilepticus and these patients, even when termination is achieved in the ED should be admitted to the hospital for further observation. All infants with true seizures should be admitted to the hospital, as well. Patients with refractory seizures, those with seizures from acute traumatic brain injury, CNS infection, or serious metabolic or electrolyte abnormalities typically require treatment in the ICU setting.

## **Febrile Seizure**

Seizures in the setting of fever are common in children and usually benign. Simple febrile seizures (SFS) occur between 6 months and 6 years of life, are brief (<15 min), and generalized. Patients with SFS require no specific ED evaluation or medication for their seizure, though evaluation for a

treatable source of fever, and antipyretics may be indicated. Children experiencing simple febrile seizure have the same 1% risk of developing epilepsy as the general population. Complex febrile seizures (>15 min in duration, focal, or recurrent) carry a slightly increased risk for epilepsy, but also do not routinely require ED evaluation or treatment. Febrile status epilepticus is treated as above with the addition of neuroimaging, CSF analysis and culture, and potential antibiotics and acyclovir in the right setting.

## **First Seizure**

The overall risk of recurrence after a single afebrile seizure is about 40%. Emergent neuroimaging is not necessary in the ED in the patient with a nonfocal neurologic exam, though outpatient MRI and EEG may be of benefit. Most neurologists do not recommend starting anti-convulsant medication after a first seizure.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 129, “Seizures and Status Epilepticus in Children,” by Maija Holsti.



## **CHAPTER 77**

# **Headache and Altered Mental Status in Children**

Kathleen M. Adalgais

## **HEADACHE**

Up to 1% of emergency department visits are for complaints of headache. The vast majority of headaches in children have a benign etiology. Factors associated with serious or dangerous causes of headache include: preschool age, occipital location, recent onset of headache, and inability of the child to describe the quality of the head pain.

### **Clinical Features**

Headaches can be classified as primary or secondary. Primary headaches are physiologic or functional (migraine, tension, cluster) and tend to be self-limited. They are often recurrent and patients have normal physical examination findings. Secondary headaches often have an anatomic basis (vascular malformation, tumor, or infection) and are associated with higher morbidity and mortality than primary headaches. A careful history and physical examination can usually differentiate between the two. History suggestive of a secondary headache includes acute onset; morning vomiting; behavioral changes; altered mental status; “worst ever” headache; wakes the child from sleep; headache associated with fever, trauma, or toxic exposure; or aggravated by coughing, Valsalva, or lying down. Physical findings suggestive of secondary headaches include blood pressure abnormalities, nuchal rigidity, head tilt, ptosis, retinal hemorrhage or optic nerve distortion, visual field defects, gait disturbances, or focal motor or sensory deficits

## Diagnosis and Differential

There are no evidence-based studies guiding diagnostic workup in children. Obtain a history from all possible sources. The history should include characteristic features of the headache such as age of first occurrence, precipitants, time and mode of onset, location, quality and severity of pain, duration of headache, and associated symptoms. Physical examination should include a thorough general examination in addition to a careful neurologic examination with attention to cranial nerves, gait, strength, and mental status. The selection of studies will depend on findings obtained from the history and physical examination. Head computed tomography and magnetic resonance brain imaging may be indicated in trauma or workup of secondary headaches (eg, patients with ventriculoperitoneal shunts, occipital headaches that are poorly characterized). *Practice guidelines do not recommend routine imaging for children with recurrent headaches and normal findings on neurologic examination.*

## Emergency Department Care and Disposition

1. For secondary headaches: treat underlying cause and pain.
2. For primary headaches, treat based on type of headache diagnosed through historical features. Most primary headaches can be treated with first line oral therapy, typically **ibuprofen** 10 milligrams/kilogram.
3. For migraines: other medications include **prochlorperazine** 0.15 milligram/ kilogram IV (consider administration with **diphenhydramine** 1 milligram/ kilogram IV to prevent dystonic reactions), **dihydroergotamine** 0.1 milligram/kilogram (ages 6 to 9) 0.15 milligram/kilogram (ages 9 to 12) or 0.2 milligram/kilogram (ages 12 to 16) can be given but is contraindicated in patients with complex migraine.
4. Cluster and tension headaches are managed much the same way as migraines. **Sumatriptan** 10 milligrams (20 to 39 kilogram) or 20 milligrams (> 40 kilogram) nasal spray or 0.1 milligram/kilogram subcutaneously and **high-flow oxygen** (7 L/min non-rebreather mask) can

be used for cluster headaches. Tension headaches usually respond to first-line oral therapy such as **ibuprofen** 10 milligrams/kilogram.

5. Address exacerbating factors to avoid reoccurrence of the headache. Consider referral for prophylactic regimens to reduce migraines.
6. In general, most patients may be discharged after relief of symptoms. Patients with life-threatening causes of headache including severe hypertension require admission for definitive care. Patients with intractable pain also may need admission.

## **ALTERED MENTAL STATUS**

Altered mental status (AMS) in a child is defined by failure to respond to the external environment in a manner consistent with the child's developmental level after appropriate stimulation. In treating children with AMS, aggressive resuscitation, stabilization, diagnosis, and treatment must occur simultaneously to prevent morbidity and death.

### **Clinical Features**

The spectrum of AMS ranges from confusion to lethargy, stupor, and coma indicative of depression of the cerebral cortex or localized abnormalities of the reticular activating system. The most simplified and functional coma scale for use in the emergency department setting is the AVPU scale, where A means "alert," V means "responsive to verbal stimuli," P means "responsive to painful stimuli," and U means "unresponsive." The A, V, P, and U values correspond to Glasgow Coma Scale scores of 15, 13, 8, and 3, respectively.

Pathologic conditions affecting mental status can be divided into supratentorial lesions, subtentorial lesions, and metabolic encephalopathy. Supratentorial lesions present with altered level of consciousness and focal motor abnormalities with a rostral-to-caudal progression of dysfunction, and slow nystagmus toward the stimulus during cold caloric testing. Subtentorial lesions produce rapid loss of consciousness, cranial nerve abnormalities, abnormal breathing patterns, and asymmetric or fixed pupils.

Metabolic encephalopathy produces decreased level of consciousness before exhibiting motor signs, which are symmetrical when present. Pupillary reflexes are intact in metabolic encephalopathy except with profound anoxia, opiates, barbiturates, and anticholinergics.

## **Diagnosis and Differential**

A thorough history and physical examination are paramount to determining the diagnosis. Key questions must include prodromal events and associated signs and symptoms, such as fever, headache, weakness, vomiting, diarrhea, gait disturbances, head tilt, rash, palpitations, abdominal pain, hematuria, and weight loss. Inquiries also should be made regarding past medical history, family history, and immunization status. The examination should look for signs of occult infection, trauma, toxicity, or metabolic disease. A useful tool for organizing diagnostic possibilities is in the mnemonic AEIOU TIPS ([Table 77-1](#)).

A	<p><b>Alcohol.</b> Changes in mental status can occur with serum levels &lt;100 milligrams/dL. Concurrent hypoglycemia is common.</p> <p><b>Acid-base and metabolic.</b> Hypotonic and hypertonic dehydration. Hepatic dysfunction, inborn errors of metabolism, diabetic ketoacidosis, primary lung disease, and neurologic dysfunction causing hypercapnia.</p> <p><b>Arrhythmia/cardiogenic.</b> Stokes-Adams, supraventricular tachycardia, aortic stenosis, heart block, ventricular fibrillation, pericardial tamponade</p>
E	<p><b>Encephalopathy.</b> Hypertensive encephalopathy can occur with diastolic pressures of 100 to 110 mm Hg. Reye syndrome. HIV. Postimmunization encephalopathy. Encephalomyelitis.</p> <p><b>Endocrinopathy.</b> Addison disease can present with AMS or psychosis. Thyrotoxicosis can present with ventricular dysrhythmias. Pheochromocytoma can present with hypertensive encephalopathy.</p> <p><b>Electrolytes.</b> Hyponatremia becomes symptomatic around 120 mEq/L. Hypernatremia and disorders of calcium, magnesium, and phosphorus can produce AMS.</p>
I	<p><b>Insulin.</b> AMS from hyperglycemia is rare in children, but diabetic ketoacidosis is the most common cause. Hypoglycemia can be the result of many disorders. Irritability, confusion, seizures, and coma can occur with blood glucose levels &lt;40 milligrams/dL.</p> <p><b>Intussusception.</b> AMS may be the initial presenting symptom.</p>
O	<p><b>Opiates.</b> Common household exposures are to Lomotil, Imodium, diphenoxylate, and dextromethorphan. Clonidine, an <math>\alpha</math>-agonist, can also produce similar symptoms.</p>

<b>U</b>	<b>Uremia.</b> Encephalopathy occurs in over one third of patients with chronic renal failure. Hemolytic uremic syndrome can produce AMS in addition to abdominal pain. Thrombocytopenic purpura and hemolytic anemia also can cause AMS.
<b>T</b>	<b>Trauma.</b> Children with blunt trauma are more likely than adults to develop cerebral edema. Look for signs of child abuse, particularly shaken baby syndrome with retinal hemorrhages. <b>Tumor.</b> Primary, metastatic, or meningeal leukemic infiltration. <b>Thermal.</b> Hypo- or hyperthermia.
<b>I</b>	<b>Infection.</b> One of the most common causes of AMS in children. Meningitis should be considered, particularly in febrile children. <b>Intracerebral vascular disorders.</b> Subarachnoid, intracerebral or intraventricular hemorrhages can be seen with trauma, ruptured aneurysm, or arteriovenous malformations. Venous thrombosis can follow severe dehydration or pyogenic infection of the mastoid, orbit, middle ear, or sinuses.
<b>P</b>	<b>Psychogenic.</b> Rare in the pediatric age group, characterized by decreased responsiveness with normal neurologic examination including oculovestibular reflexes. <b>Poisoning.</b> Drugs or toxins can be ingested by accident, through neglect or abuse, or in a suicide gesture.
<b>S</b>	<b>Seizure.</b> Generalized motor seizures are often associated with prolonged unresponsiveness in children. Seizures in a young febrile patient suggest intracranial infection. Shunt malfunction should be considered among patients with a ventriculoperitoneal shunt for hydrocephalus.

Key: AMS = altered mental status.

**TABLE 77-1 AEIOU TIPS: A Mnemonic for Pediatric Altered Mental Status**

Diagnostic adjuncts may include analysis of blood, gastric fluid, urine, stool, cerebrospinal fluid, electrocardiography, or selected radiographic studies, and should be guided by the clinical situation. Rapid bedside glucose determination is a universally accepted standard. If meningitis or encephalitis is suspected, lumbar puncture and cerebrospinal fluid analysis should be done as rapidly as possible after initial resuscitation and stabilization. A 12-lead electrocardiogram should be obtained in cases in which there are pathologic auscultatory findings or rhythm disturbances.

**Emergency Department Care and Disposition**

Treatment priorities should concentrate on stabilization and reversal of life-threatening conditions.

1. Ensure airway, breathing, and circulation. Immobilize the cervical spine if trauma is suspected and obtain appropriate radiographic studies when the patient is stabilized.
2. Provide continuous pulse oximetry and supplemental **oxygen** as needed to correct hypoxia, including bag-valve-mask and intubation when appropriate. Consider capnometry for intubated patients.
3. Administer fluid resuscitation with 20 mL/kg fluid boluses of **isotonic crystalloid** for hypotension. Fluid boluses may be repeated up to 60 mL/kg, after which the need for pressor agents, such as dopamine, should be considered. Caution should be used when intracranial hypertension is suspected.
4. Treat hypoglycemia with **10% dextrose 4 to 5 mL/kg** in infants or **25% dextrose 2 mL/kg** in older children.
5. Control core body temperature to minimize metabolic demands. Prevent hypothermia with warming lamps and treat hyperthermia when present.
6. Treat seizures with benzodiazepines (see [Chapter 76](#)).
7. For suspected opiate or clonidine overdose, administer **nalaxone** (0.01 to 0.1 milligram/kilogram IV every 2 min). For suspected benzodiazepine overdose, administer **flumezenil** (0.01 milligram/kilogram IV).
8. Administer empiric antibiotics (**ceftriaxone** or **cefotaxime** 50 milligrams/kilogram/dose, consider additional **vancomycin** 10 milligrams/ kilogram/dose) for suspected meningitis.
9. Most patients with AMS will require admission and extended observation. Only those with a transient, rapidly reversible, and benign cause of AMS can be treated and discharged from the emergency department after a period of observation with follow-up scheduled within 24 hours of discharge.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 130, “Headaches in Children,” by Brian R.E. Schultz and Charles G. Macias and Chapter 131 “Altered Mental Status in Children” by Jonathan I. Singer.

## **CHAPTER 78**

# **Syncope and Sudden Death in Children and Adolescents**

Derya Caglar

Syncope is more common in adolescents than younger children. Up to 50% of adolescents experience at least 1 syncopal episode. This condition is usually transient and usually self-limited, but can be a symptom of serious cardiac disease.

Sudden, unexpected death in children comprises 2.3% of all pediatric deaths of which sudden cardiac death makes up about one-third. Except for trauma, sudden cardiac death is the most common cause of sports-related deaths, particularly in basketball, football, and track. Hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in adolescents without known cardiac disease. Other causes of sudden cardiac death in children include myocarditis, congenital heart disease, and conduction disturbances.

## **CLINICAL FEATURES**

Syncope is the sudden onset of falling accompanied by a brief episode of loss of consciousness. Involuntary motor movements may occur with all types of syncopal episodes but are most common with seizures. Two-thirds of children experience light-headedness or dizziness before the episode (“presyncopal” symptoms). [Table 78-1](#) lists the most common causes of syncope by category.



<p><b>Neurally mediated:</b> most common cause of syncope in children  Vasovagal: &lt;1 min duration, prolonged standing, emotional upset, warning signs  Orthostatic: light-headedness with standing may precede; due to hypovolemia  Situational: urination, defecation, coughing, and swallowing may precipitate familial dysautonomia</p>
<p><b>Cardiac dysrhythmias:</b> events that usually start and end abruptly  Prolonged Q-T syndrome  Supraventricular tachycardia / Wolff-Parkinson-White syndrome  Sick sinus syndrome: associated with prior heart surgery  Atrioventricular block: most common in children with congenital heart disease  Pacemaker malfunction</p>
<p><b>Structural cardiac disease:</b>  Hypertrophic cardiomyopathy: commonly exertional syncope; infants present with congestive heart failure and cyanosis  Dilated cardiomyopathy: idiopathic, postmyocarditis, or congenital  Congenital heart disease  Valvular diseases: aortic stenosis often congenital; Ebstein malformation; mitral valve prolapse (associated with syncope but NOT increased risk of sudden death)  Arrhythmogenic right ventricular dysplasia  Pulmonary hypertension: dyspnea on exertion, exercise intolerance, shortness of breath  Coronary artery abnormalities: aberrant left main artery causing external compression during physical exercise</p>
<p><b>Medications and drugs:</b> antihypertensives, tricyclic antidepressants, cocaine, diuretics, antidysrhythmics</p>

**TABLE 78-1** Causes of Syncope in Children and Adolescents

Neurally mediated syncope is the most common cause in children and includes vasovagal, vasodepressor, neurocardiogenic, reflex syncope, and simple fainting. This type of syncope is usually preceded by sensations of nausea, warmth, or light-headedness with a gradual visual grayout. Cardiac syncope occurs when there is an interruption of cardiac output from an intrinsic problem such as tachydysrhythmia, bradydysrhythmia, outflow obstruction, and myocardial dysfunction. Syncope resulting from cardiac causes usually begins and ends abruptly and may be associated with chest pain, palpitations, or shortness of breath. Risk factors associated with serious causes of syncope are presented in [Table 78-2](#). Events easily mistaken for syncope are presented in [Table 78-3](#) in addition to common associated symptoms.

Exertion preceding the event  
 History of cardiac disease in the patient  
 Family history of sudden death, deafness, or cardiac disease  
 Recurrent episodes  
 Recumbent episode  
 Prolonged loss of consciousness  
 Associated chest pain or palpitations  
 Use of medications that can alter cardiac conduction

**TABLE 78-2** Risk Factors for a Serious Cause of Syncope

Condition	Distinguishing Characteristics
Basilar migraine	Headache, rarely loss of consciousness, other neurologic symptoms
Seizure	Loss of consciousness simultaneous with motor event, prolonged postictal phase
Vertigo	Rotation or spinning sensation, no loss of consciousness
Hyperventilation	Inciting event, paresthesias or carpopedal spasm, tachypnea
Hysteria	No loss of consciousness, indifference to event
Hypoglycemia	Confusion progressing to loss of consciousness, requires glucose administration to terminate
Breath-holding spell	Crying before event, child 6 to 18 mo old

**TABLE 78-3** Events Easily Mistaken for Syncope

## DIAGNOSIS AND DIFFERENTIAL

No specific historical or clinical features reliably distinguish between vasovagal syncope and other causes. However, a thorough history and physical examination can help to arouse or allay suspicion of serious causes. The most important step in evaluation of children with syncope is a detailed history, including a thorough description of the event, associated symptoms, circumstances, medications, drugs, intake, and intercurrent illness. Syncope during exercise suggests a more serious cause. Many of the diseases that cause syncope also cause sudden death in children. Approximately 25% of children who suffer sudden death have a history of syncope. If witnesses note that the patient appeared lifeless or

cardiopulmonary resuscitation was performed, a search for serious pathologic conditions must be undertaken.

The physical examination includes complete cardiovascular (heart sounds, murmurs, rhythm, and the character of pulses), neurologic, and pulmonary examinations. Abnormalities found on examination warrant further workup. Tests should be directed by the history: a classic story for vasovagal syncope with a normal physical examination requires no further testing. Palpitations or exertional syncope require ECG evaluation for potential arrhythmias. Sudden collapse, especially during exercise suggests structural abnormalities, particularly when associated with a murmur on physical examination. ECG, chest radiography, and echocardiography should be considered in this setting. Syncope associated with chest pain may require cardiac troponins as part of the evaluation to rule out ischemic heart disease (eg, aberrant left coronary artery). Electrolytes, thyroid studies, and urine pregnancy testing or drug screen should be considered in the appropriate clinical setting.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Obtain an electrocardiogram (ECG) in all patients except those with an unquestionable vasovagal episode.
2. Consider an echocardiogram for patients with known or suspected cardiac disease. If an echocardiogram is not immediately available, the urgency for obtaining the study should be determined in consultation with a cardiologist.
3. If no clear cause is found, the child may be discharged to be further evaluated and followed by the primary care physician unless there are cardiac risk factors or exercise-induced symptoms for which referral to a cardiologist is warranted.
4. Patients with a normal ECG but a history suggesting a dysrhythmia are candidates for outpatient monitoring and cardiac workup.

5. Children with documented dysrhythmias should be admitted. All children admitted for syncope should undergo cardiac monitoring. Children who are survivors of sudden cardiac arrest should be admitted to a pediatric intensive care unit.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 140, “Syncope and Sudden Death,” by William E. Hauda II and Maybelle Kou.

## **CHAPTER 79**

# **Hypoglycemia and Metabolic Emergencies in Infants and Children**

Matthew Hansen

## **HYPOGLYCEMIA**

Hypoglycemia in children may be due to inadequate oral intake, excess insulin, low levels of hyperglycemic hormones (cortisol), inborn errors of metabolism, or systemic infection. Prompt recognition and treatment of hypoglycemia is essential to avoid potentially severe and permanent neurologic injury and bedside glucose testing should be considered absolutely essential in any infant or child with altered mental status.

### **Clinical Features**

Neonates and infants with hypoglycemia typically present with altered mental status and nonspecific symptoms such as poor feeding, an abnormal or high-pitched cry, temperature instability, and irritability or lethargy. Hypoglycemic children may manifest symptoms related to adrenergic hormone release including tachycardia, diaphoresis, tremors, anxiety, irritability, and tachypnea. Severe hypoglycemia may result in apnea or seizures. Hypoglycemia often accompanies critical illness (sepsis) and the features of that illness may dominate the clinical picture, thereby masking the signs of hypoglycemia.

### **Diagnosis and Differential**

Hypoglycemia is defined as a plasma glucose concentration less than 45 milligrams/dL in symptomatic children and less than 35 milligrams/dL in asymptomatic neonates. Bedside glucose testing is the most important diagnostic test in any neonate or infant who is critically ill or has altered mental status. Urine testing for ketones is important as ketonuria is associated with ketotic hypoglycemia, adrenal insufficiency and other inborn errors of metabolism. Absent urine ketones are associated with hyperinsulinemic states such as nesidioblastosis, infants of a diabetic mother, as well as disorders of fatty acid oxidation and mitochondria.

### **Emergency Department Care and Disposition**

1. For neonates administer 5 mL/kg of **10% dextrose** IV/IO/PO/NG. Treat infants with the same dose of 10% dextrose, or 2 mL/kg of **25% dextrose** IV/IO/PO/NG. Give older children 2 mL/kg of **25% dextrose** IV/IO/PO/NG.
2. Administer maintenance dextrose for persistent hypoglycemia using **10% dextrose** at 1.5 times maintenance.
3. When intravenous access is not immediately available, consider **glucagon** 0.03 milligram/kilogram IM.
4. When standard therapy fails, give **hydrocortisone** 25 milligrams IV for neonates and infants, 50 milligrams for toddlers and smaller school aged children and 100 milligrams for everyone else. Steroids should be given early in patients with hypopituitarism and adrenal insufficiency.

## **INBORN ERRORS OF METABOLISM**

Inborn errors of metabolism are challenging childhood disorders representing a broad spectrum of diseases with nonspecific signs and symptoms. Delay in accurate diagnosis and treatment can lead to significant morbidity and mortality. Despite the myriad etiologies, the principles of initial ED diagnosis and management are relatively simple. The sudden acute deterioration of a healthy neonate should always prompt consideration

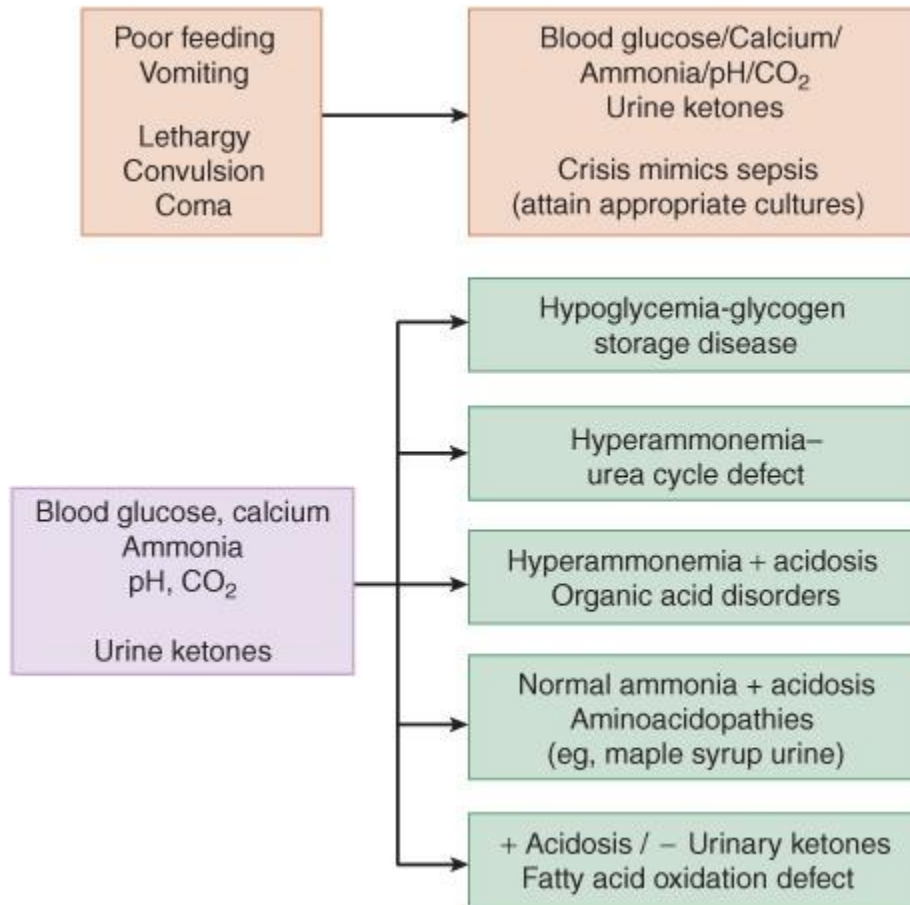
of metabolic disease, and making a definitive diagnosis is less important than having a high index of suspicion and implementing supportive care.

## **Clinical Features**

Vomiting, altered mental status, and poor feeding are the most common features of metabolic emergencies. Seizures may accompany some metabolic crises. Tachypnea due to metabolic acidosis and tachycardia from dehydration as well as hypotension due to hypovolemia or adrenal insufficiency may be noted. Rarely, some metabolic disorders may be associated with characteristic body or urine odors or other phenotypic stigmata (eg, ambiguous genitalia and hyperpigmentation in congenital adrenal hyperplasia, as discussed separately below).

## **Diagnosis and Differential**

Screening laboratories for suspected inborn errors of metabolism include a bedside glucose, urine for ketones, a blood gas analysis for metabolic acidosis, serum ammonia and calcium. [Figure 79-1](#) details the diagnostic evaluation recommended in the emergency department. Additional laboratory tests for definitive diagnosis that should be considered based on initial screening results include liver function tests, CBC, aldolase, creatine kinase, serum amino acids and acylcarnitine profile, urine organic acids, and reducing substances. The differential diagnosis of shock in the neonate includes sepsis, congenital heart defects, and abdominal catastrophes.



**FIGURE 79-1.** Approach to suspected metabolic disorders.

## **Emergency Department Care and Disposition**

Despite the diverse etiology and complexity of inborn errors of metabolism, ED resuscitation and stabilization of patients with these disorders is relatively simple. Neonates, infants, and children presenting in metabolic crisis, regardless of cause, show some combination of dehydration, metabolic acidosis, and encephalopathy, which must be immediately addressed. The goals of treatment are, to improve circulatory status by restoring circulatory volume, provide energy substrate to halt catabolism, remove the inciting metabolic substrate (formula or breast milk), and help eliminate toxic metabolites.



1. Restore circulation with **0.9% saline boluses of 20 mL/kg**.
2. Make patients NPO, and correct hypoglycemia as above with D10 in neonates and infants or D25 in children.
3. Begin **10% dextrose infusion at twice maintenance rate**.
4. Consider broad spectrum antibiotics.
5. Specific metabolic treatments are guided by the underlying defect and should be determined in consultation with a metabolic specialist. Substrates to facilitate shunting of metabolic pathways or hemodialysis may be needed.

Patients with a newly diagnosed or suspected metabolic disorder and those who are dehydrated or otherwise decompensated require admission for dextrose and specific treatment. Transfer to a pediatric hospital may be required.

Perinatal period	Infancy and childhood
Infant of a diabetic mother	Idiopathic ketotic hypoglycemia/starvation
Congenital heart disease	Diabetes mellitus/endocrine disorder
Infection/sepsis	Infection/sepsis
Adrenal hemorrhage	Inborn errors of metabolism
Hypothermia	Hypothermia
Hypoglycemia-inducing drug use by mother	Drug induced (salicylates, etc) Hyperinsulinism
Maternal eclampsia	Idiopathic
Fetal alcohol syndrome	
Hypopituitarism	

**TABLE 79-1** Conditions Associated With Hypoglycemia in Infants and Children

## CONGENITAL ADRENAL HYPERPLASIA

Congenital adrenal hyperplasia results from deficiency in 1 of the enzymes involved in the production of cortisol which leads to decreased cortisol levels sometimes accompanied by mineralocorticoid deficiency (“salt

wasting syndrome”). Steroid precursors may be shunted to androgen production with virilization of females.

## **Clinical Features**

Patients with salt-wasting adrenal hyperplasia typically present in the second to fifth week of life with nonspecific symptoms including shock, vomiting, lethargy, irritability, and poor feeding. On examination, females may have clitoromegaly and males may have a small penis or hypospadias. Hyperpigmentation may be noted on axillae and around the nipples.

## **Diagnosis and Differential**

The most important laboratories include bedside glucose and electrolytes as hyponatremia and hyperkalemia are often present and suggest the diagnosis. Serum potassium levels of 6 to 12 mEq/kg are not unusual but are rarely associated with ECG abnormalities. Definitive diagnosis depends on analysis of blood hormone levels. If possible, blood should be obtained for analysis before treatment with exogenous steroids; however, treatment should not be delayed in the critically ill neonate. The differential diagnosis includes sepsis, congenital heart disease, and other inborn errors of metabolism.

## **Emergency Department Care and Disposition**

1. Administer **0.9% saline boluses of 20 mL/kg IV/IO.**
2. Treat hypoglycemia as above.
3. Administer hydrocortisone IV/IO/IM: **25 milligrams for neonates, 50 milligrams for toddlers and 100 milligrams for older children.**
4. Treat hyperkalemia with **calcium gluconate 100 milligrams/kilogram IV and bicarbonate 1 mEq/kilogram IV. Insulin should be avoided as it may cause profound hypoglycemia.**

Neonates with adrenal crisis from adrenal hyperplasia require hospitalization. Those with a known diagnosis of adrenal hyperplasia who have normal vital signs and are able to tolerate oral intake may be discharged home after administration of hydrocortisone as above with instructions to triple their usual home dose of steroid until fever, vomiting, or diarrhea resolve when next-day follow-up can be assured.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 137, “Hypoglycemia and Metabolic Emergencies in Infants and Children,” by Nadeemuddin Qureshi, Mohammed Al-Mogbil, and Osama Kentab.

# **CHAPTER 80**

## **The Diabetic Child**

Adam Vella

Type 1 diabetes is characterized by an abrupt and frequently complete decline in insulin production. Type 2 diabetes is marked by increasing insulin resistance, and occurs in overweight adolescents with a strong genetic predisposition. Diabetic ketoacidosis (DKA) is the leading cause of mortality in patients with diabetes <24 years of age, and cerebral edema is the leading cause of mortality in DKA.

DKA is much more common in patients with type 1 diabetes than in those with type 2, but patients with type 2 to diabetes may develop hyperglycemic, hyperosmolar nonketotic (HHNK) syndrome with acidosis, which can result in severe total body water, potassium, and phosphorus deficits. About 4% of children with newly diagnosed type 2 diabetes present with HHNK syndrome, which has a case fatality rate of 12%.

### **CLINICAL FEATURES**

Polyuria, polydipsia, and polyphagia are the classic triad leading to the diagnosis of type 1 diabetes. Other common symptoms include weight loss, secondary enuresis, anorexia, vague abdominal discomfort, visual changes, and genital candidiasis in a toilet-trained child.

Premonitory symptoms of cerebral edema occur in as few as 50% and include severe headache, declining mental status, seizures, and papilledema. Cerebral edema typically occurs 6 to 12 hours after initiating therapy and presents as headache, mental status changes, seizure, or coma. Although the etiology of this complication is unknown, it is felt that several factors may contribute, including overly aggressive fluid therapy, rapid correction of blood glucose levels, bicarbonate therapy, and failure of the serum sodium level to increase with therapy.

Occasionally children with DKA present to the ED complaining primarily of abdominal pain, which may mimic acute appendicitis; Kussmaul breathing (hyperpnea from acidosis) may be mistaken for hyperventilation from anxiety or respiratory distress from pulmonary disease.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of diabetes is established by demonstrating hyperglycemia and glucosuria in the absence of other causes such as steroid therapy, Cushing syndrome, pheochromocytoma, hyperthyroidism, or other rare disorders. DKA is generally defined as a metabolic acidosis (pH <7.25 to 7.30 or serum bicarbonate level of <15 mEq/L) with hyperglycemia (serum glucose level of >300 milligrams/dL) in the presence of ketonemia/ketonuria.

Cerebral edema in DKA is a clinical diagnosis based on altered mental status not attributed to hypovolemia and treatment should begin prior to obtaining head CT when suspected. CT imaging can confirm the diagnosis, and intracranial pressure monitoring may be indicated.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

The treatment of DKA consists of judicious fluid resuscitation, insulin therapy, correction of electrolyte abnormalities, and close monitoring. Patients should be placed on a cardiac monitor, noninvasive blood pressure device, and pulse oximetry, and intravenous lines should be established.

1. Administer 10 to 20 mL/kg normal saline boluses until hemodynamic stability is achieved. Give an initial 20 mL/kg normal saline (NS) bolus of normal saline if the child is in shock and repeat if needed. Once vital signs have stabilized, resist the desire to correct the fluid deficit too

rapidly, especially if there is a high calculated osmolarity (ie, > 340 mOsm/L).

2. Follow the initial bolus with NS at 1.5 times maintenance rate in the ED.
3. If [K<sup>+</sup>] is <5.5 mEq/L and patient is urinating, add 30 mEq potassium per L (half as KCl and half as potassium phosphate). If initial [K<sup>+</sup>] is 2.5 to 3.5 mEq/L, add 40 mEq [K<sup>+</sup>]/L; consider adding more if the [K<sup>+</sup>] is <2.5 mEq/L.
4. Begin regular insulin at 0.05 to 0.1 unit/kilogram/h after IV fluid bolus (if given) is complete. Adjust dose to maintain glucose decline at 50 to 100 milligrams/dL/h. Do not decrease insulin infusion below 0.05 unit/kilogram/h until ketonuria has resolved. High-dose insulin therapy and insulin boluses increase the risk of complications and should not be given.
5. Add dextrose to IV fluids when blood glucose is <200 to 250 milligrams/dL. Glucose levels correct faster than ketoacidosis, so supplement with dextrose and continue the insulin drip until ketoacidosis has resolved.
6. Measure serum electrolyte levels every 2 hours; measure serum glucose level every hour.
7. **The use of bicarbonate in the treatment of DKA is contraindicated**, as it does not improve outcome and it has been associated with a fourfold increase in the development of cerebral edema.
8. **Management of cerebral edema.** Treat patients with altered mental status suggestive of cerebral edema with **mannitol** 0.5 to 1 gram/kilogram. Consider 3% hypertonic saline, 10 mL/kg over 30 min. Restrict additional IV fluids to minimum required to maintain IV access. Use caution if endotracheal intubation is required and avoid eucapnea as severe metabolic acidosis requires compensatory respiratory alkalosis and a rise in CO<sub>2</sub> may worsen systemic and intracellular acidosis.
9. Most patients with DKA require admission to the intensive care unit, even when in stable condition, because of intensive monitoring needs. Furthermore, many hospitals restrict the use of insulin infusions to intensive care settings. Patients with cerebral edema require ICU admission and possible intracranial pressure monitoring. Consultation

with the patient's primary care physician and a pediatric endocrinologist should be made early in the course of therapy.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 139, "The Child with Diabetes," by Adam Vella.

# CHAPTER 81

## Fluid and Electrolyte Therapy

Lance Brown

The most common disorder of fluid balance in children requiring emergency care is dehydration. Dehydration is the result of a negative fluid balance that can result from decreased fluid intake (mouth or throat disorders, systemic illness, neurologic illness, and other causes); increased output (vomiting, diarrhea, fever, environmental heat, respiratory illness, renal losses and other causes); or fluid shifts in conditions such as burns and sepsis.

### CLINICAL FEATURES

The clinical appearance of patients with dehydration and fluid and electrolyte disturbances depends on the degree of dehydration, the rate at which the fluid was lost, and the age of the patient. Older children may tolerate a slow total body water loss as great as 40%, while rapid and large volume loss (eg, rotavirus or cholera) can cause rapid deterioration and cardiovascular collapse in young infants.

Though the gold standard for assessing dehydration is comparison of pre-illness weight with weight on presentation to the ED, a reliable and recent pre-illness weight is rarely available in the emergency department. Physical examination can provide an estimation of the degree of dehydration, which is typically classified as mild, moderate, or severe. Clinical signs and symptoms of dehydration are listed in [Table 81-1](#) (also consider [Table 73-1](#) if dehydration is due to GI losses). An important exception to the reliability of signs and symptoms to predict degree of dehydration occurs in hypernatremic dehydration, when fluid loss occurs primarily from the interstitial and intracellular spaces and clinical signs of intravascular volume depletion may be minimal. In this setting, however, the skin may have a characteristic doughy feel.



	None to Mild (<3% body weight loss)	Mild to Moderate (3% to 9% body weight loss)	Severe (>9% body weight loss)
Mental status	Well, alert	Fatigued, restless, irritable	Apathetic, lethargic, unconscious
Thirst	Normal, slight increase, or refusing fluids	Increased, eager to drink	Very thirsty or too lethargic to drink
Heart rate	Normal	Normal to increased	Tachycardia or bradycardia in critically ill children
Pulse quality	Normal	Normal to reduced	Weak, thready
Eyes	Normal	Slightly sunken	Sunken
Tears	Present	Decreased	Absent
Mucous membranes	Moist	Dry	Parched
Anterior fontanelle	Normal	Sunken	Sunken
Capillary Refill	Normal	3 to 4 s	>4 s
Extremities	Warm	Warm to cool	Cold, mottled, cyanotic
Urine output	Normal	Decreased	Minimal

**TABLE 81-1** Clinical Guidelines for Assessing Dehydration in Children

## DIAGNOSIS AND DIFFERENTIAL

If available, the absolute and relative fluid deficit can be calculated from a pre-illness weight: 1 kilogram of weight loss is equivalent to 1 L of fluid deficit. In the absence of a reliable pre-illness comparison weight, the diagnosis of dehydration is based primarily on historical data and physical examination findings ([Table 81-1](#)).

Laboratory tests are not needed in mild to moderate cases of dehydration but may be helpful in some cases where results of a basic metabolic panel may help classify the type of dehydration (eg, isotonic, hypernatremic, and hyponatremic), and identify related problems (eg, renal failure, ketotic hypoglycemia, and diabetic ketoacidosis). The serum bicarbonate level (or total CO<sub>2</sub>) is inversely related to the degree of dehydration (ie, the lower the serum bicarbonate, the greater the degree of dehydration).

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

The management of fluid and electrolyte disturbances in infants and young children revolves around a few basic principles: (a) identification and treatment of shock; (b) administration of appropriate fluids to replace fluid deficits, ongoing losses, and maintenance fluid requirements; (c) identification and treatment of causes that have specific therapies (eg, diabetic ketoacidosis, sepsis, inborn errors of metabolism). The most common approaches to rehydration include oral rehydration therapy and parenteral therapy, though rehydration through nasogastric tube is also effective, simple, and well tolerated.

1. Treat hypovolemic shock with an initial bolus of 20 mL/kg of isotonic crystalloid (normal saline [NS] or lactated Ringer [LR] solution) IV/IO. Repeat boluses every 10 min until mental status, vital signs, and peripheral perfusion improve.
2. Oral rehydration is as effective as intravenous therapy and is recommended by the WHO for even moderate dehydration. Administer frequent small sips of oral rehydration solution containing glucose and electrolytes (eg, Rehydralyte<sup>®</sup>) by mouth or nasogastric tube. Give 50 mL/kg orally over 4 hours for mild dehydration and 100 mL/kg for moderate dehydration. Vomiting is not a contraindication to attempting oral rehydration.
3. Consider ondansetron to facilitate oral rehydration in nauseated or vomiting children: 0.1 milligram/kilogram IV or 0.15 milligram/kilogram PO. The lower age limit, frequency of dosing, and maximum dosing for the safe administration of ondansetron has not been determined.
4. Treat patients who cannot tolerate oral or enteral rehydration with IV or IO administration of NS or LR. After resuscitation from shock using bolus therapy, calculate or estimate the fluid deficit ([Table 81-1](#)) and replace half the total deficit over the first 8 hours and the remaining deficit over the next 16 hours. For example, if the patient weighs 15 kilograms on presentation with an estimated 10% dehydration, then the

fluid deficit is 15 kilograms  $\times$  10% = 1.5 kilograms = 1.5 L. Double the replacement time-frame in cases of severe hypernatremic dehydration to avoid potential cerebral edema. Maintenance fluids and electrolytes (see [Table 81-2](#)) as well as ongoing volume losses should be added to the calculated total fluid deficit.

Body Weight	1 to 10 kg	10 to 20 kg	>20 kg
ED maintenance: 4-2-1 rule*	4 mL/kg/h	4 mL/kg/h for first 10 kg <i>plus</i> 2 mL/kg/h for 10 to 20 kg: [40] + 2 (kg - 10)	4 mL/kg/h for first 10 kg <i>plus</i> 2 mL/kg/h for 10 to 20 kg <i>plus</i> 1 mL/kg/h for >20 kg: [60] + 1(kg - 20)
Total water volume (24 h)	100 mL/kg	1000 mL + 50 mL for each kg >10 to 20 kg	1500 mL + 20 mL/kg for each kg >20 kg
Sodium	3 mEq/kg	3 mEq/kg	3 mEq/kg
Potassium	2 mEq/kg	2 mEq/kg	2 mEq/kg
Chloride	5 mEq/kg	5 mEq/kg	5 mEq/kg

\*The 4-2-1 rule accounts for insensible losses and metabolic water needs, but does not consider fluid deficits or ongoing losses, which should be added to this computed value.

**TABLE 81-2** Maintenance Requirements for Fluid and Electrolytes in Children, Based on Body Weight

5. Treat specific electrolyte disturbances as listed in [Table 81-3](#).

Electrolyte Disorder	Common Causes	Symptoms and Signs	Initial Treatment Recommendations
Hyponatremia	Vomiting, diarrhea, excess free water intake	Mental status changes, seizures, hyporeflexia	IV normal saline starting with a 20 mL/kg bolus For seizures: 4 mL/kg of 3% saline over 30 min
Hypernatremia	Vomiting, diarrhea, insensible losses, diabetes insipidus, renal disease	Diarrhea, mental status changes, ataxia, doughy skin, seizures, hyperreflexia	IV normal saline starting with a 20 mL/kg bolus Further correction to take place slowly over 48 h
Hypokalemia	Vomiting, DKA	Muscle weakness, ileus	Generally tolerated well, replace orally over several days If severe: IV 0.2 to 0.3 mEq/kg/h of KCl
Hyperkalemia*	Cortical adrenal hyperplasia (neonates), renal failure. May be due to hemolysis of blood sample	ECG changes: peaked T waves, prolonged PR interval, widening of QRS	Insulin 0.1 unit/kg <i>plus</i> 25% glucose, 0.5 gram/kg IV Calcium gluconate 10%, 1 mL/kg IV, no faster than 1 mL/min Albuterol, 0.5% solution, 2.5 milligrams via nebulization

			For other treatments, see Chapter 4.
Hypocalcemia	Dietary or, vitamin D deficiency, hypoparathyroid and chronic renal failure	Vomiting, irritability, muscle weakness, tetany, seizures	Calcium gluconate 10%, 1 mL/kg IV, no faster than 1 mL/min
Hypercalcemia	Malignancy, hyper-vitaminosis D or A	Fatigue, irritability, anorexia, vomiting, constipation	IV normal saline starting at 20 mL/kg Bisphosphonates if admission required
Hypomagnesemia	Diarrhea, short gut, diuretics, chemotherapy	Muscle spasms, weakness, ataxia, nystagmus, seizures ECG changes: prolonged PR and QTc, torsades de pointes	For seizures or arrhythmia: IV magnesium sulfate 1 mEq/kg slowly over 4 h Asymptomatic patients can be treated with oral supplements
Hypermagnesemia	Ingestion of antacids or renal dysfunction,	Hypotension, respiratory failure, loss of deep tendon reflexes ECG changes: widening of QRS, PR, QTc	Remove exogenous source of magnesium If severe: calcium gluconate 10% 1 mL/kg IV no faster than 1 mL/min

\*Mild hyperkalemia usually well tolerated in neonates.

**TABLE 81-3** Electrolyte Disorders in Infants and Children and Initial Management

Most children with mild to moderate dehydration can be managed as outpatients without any laboratory evaluation in the emergency department. Admission criteria include young infants with ongoing significant fluid losses; severe dehydration; significant electrolyte or metabolic derangements; persistent vomiting and failed attempts at oral rehydration; or an underlying diagnosis requiring ongoing inpatient treatment (eg, DKA or inborn errors of metabolism).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 142, “Fluid and Electrolyte Therapy in Infants and Children,” by Alan L. Nager.

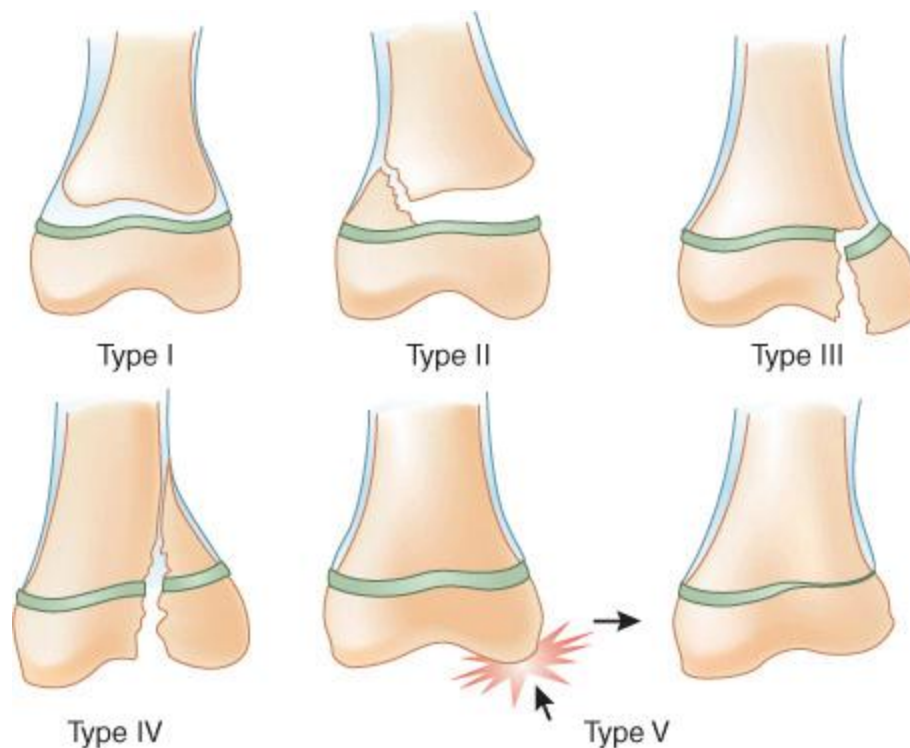
## CHAPTER 82

# Musculoskeletal Disorders in Children

Mark X. Cicero

## CHILDHOOD PATTERNS OF INJURY

The growth plate (physis) is the weakest point in children's long bones and the frequent site of fractures. The ligaments and periosteum are stronger than the physis, therefore they tolerate mechanical forces at the expense of physal injury. The blood supply to the physis arises from the epiphysis, so separation of the physis from the epiphysis may result in growth arrest. The Salter-Harris classification is used to describe fractures involving the growth plate ([Fig. 82-1](#)).



**FIGURE 82-1.** Salter-Harris classification of physeal injuries. The portion of the bone proximal to the thick black line is the metaphysis; the thick green line represents the physis (growth plate), and the portion distal to the physis is the epiphysis.

### **Salter-Harris Type I Fracture**

In type I physeal fracture, the epiphysis separates from the metaphysis. The reproductive cells of the physis stay with the epiphysis. There are no bony fragments. Bone growth is undisturbed. Diagnosis is suspected clinically in children with point tenderness over a growth plate. On radiograph, there may be no abnormality; there may be an associated joint effusion; or there may be epiphyseal displacement from the metaphysis. In the absence of epiphyseal displacement, the diagnosis is clinical. Treatment consists of splint immobilization, ice, elevation, and referral.

### **Salter-Harris Type II Fracture**

Type II physeal fracture is the most common (75%) physeal fracture. The fracture goes through the physis and out through the metaphysis. Growth is preserved because the physis remains with the epiphysis. Treatment is closed reduction (if necessary) with analgesia and sedation followed by cast immobilization.

### **Salter-Harris Type III Fracture**

The hallmark of type III physeal fracture is an intraarticular fracture of the epiphysis with the cleavage plane continuing along the physis. The prognosis for bone growth depends on the circulation to the epiphyseal bone fragment and is usually favorable. Reduction of the unstable fragment with anatomic alignment of the articular surface is critical. Open reduction is sometimes required.

## **Salter-Harris Type IV Fracture**

The fracture line of type IV physeal fractures begins at the articular surface and extends through the epiphysis, physis, and metaphysis. Especially when there is displacement of the bony fragments, open reduction is required to reduce the risk of premature bone growth arrest.

## **Salter-Harris Type V Fracture**

In type V physeal fracture, the physis is essentially crushed by severe compressive forces. There is no epiphyseal displacement. The diagnosis is often difficult. An initial diagnosis of sprain or type I injury may prove incorrect when later growth arrest occurs. Radiographs may look normal or demonstrate focal narrowing of the epiphyseal plate. There is usually an associated joint effusion. Treatment consists of cast immobilization, nonweight bearing, and close orthopedic follow-up in anticipation of focal bone growth arrest.

## **Torus Fractures, Greenstick Fractures, and Plastic Deformities**

Children's long bones are more compliant than those of adults and tend to bow and bend under forces where an adult's might fracture. Torus (*buckle*) fractures involve a bulging or buckling of the bony cortex, usually of the metaphysis. Patients have point tenderness over the fracture site and soft tissue swelling. Radiographs may be subtle but show cortical disruption. Torus fractures are not typically angulated, rotated, or displaced, so reduction is rarely necessary. Splinting in a position of function for 3 to 4 weeks is preferred over casting. Orthopedic follow-up is recommended.

In greenstick fractures, the cortex and periosteum are disrupted on one side of the bone but intact on the other. Treatment is closed reduction and immobilization.

Plastic deformities are seen in the forearm and lower leg in combination with a completed fracture in the companion bone. The diaphyseal cortex is



deformed, but the periosteum is intact.

## **FRACTURES ASSOCIATED WITH CHILD ABUSE**

Certain injury patterns are consistently seen in abused children, particularly multiple fractures in various stages of healing. Please see [Chapter 187](#) for details.

## **SELECTED PEDIATRIC INJURIES**

### **Clavicle Fracture**

Clavicles are commonly fractured in children, and may occur in newborns during difficult deliveries, presenting in neonates with nonuse of the arm. If the fracture was not initially appreciated, parents may notice a bony callus at age 2 to 3 weeks. In older infants and children, the usual mechanism is a fall onto the outstretched arm or shoulder. Care of the patient with a clavicle fracture is directed toward pain control. Even displaced fractures usually heal well, although patients may have a residual bump at the fracture site. A simple sling is effective and less painful than other methods of clavicle immobilization. Newborns require no specific treatment. Orthopedic follow-up can be arranged in the next week. Orthopedic consultation in the ED is required for an open fracture (which also requires antibiotics), anterior or posterior displacement of the medial clavicle, or a skin-tenting fracture fragment that has the potential to convert to an open fracture.

### **Supracondylar and Condylar Fractures**

The most common elbow fracture in childhood is the supracondylar fracture of the distal humerus. The mechanism is commonly a fall onto the outstretched arm. The close proximity of the brachial artery to the fracture

predisposes the artery to injury. Subsequent arterial spasm or compression by casts may further compromise distal circulation. A forearm compartment syndrome (Volkmann ischemic contracture) may occur. Symptoms include pain in the proximal forearm on passive finger extension, stocking-glove anesthesia of the hand, and hard forearm swelling. Pulses may remain palpable at the wrist despite serious vascular impairment. Injuries to the ulnar, median, and radial nerves are also common, occurring in 5% to 10% of all supracondylar fractures. Children complain of pain on passive elbow flexion and maintain the forearm pronated.

Radiographs show the injury, but the findings may be subtle. A posterior fat pad sign is indicative of intraarticular effusion and thus fracture. Normally, the anterior humeral line, a line drawn along the anterior distal humeral shaft, should bisect the posterior two-thirds of the capitellum on the lateral view. In subtle supracondylar fractures, the line often lies more anteriorly.

In cases of neurovascular compromise, immediate fracture reduction is indicated. If an ischemic forearm compartment is suspected after reduction, surgical decompression or arterial exploration may be indicated. Admission is recommended for patients with displaced fractures or significant soft tissue swelling. Open reduction is often required. Outpatient treatment is acceptable for nondisplaced fractures with minimal swelling; however, telephone consultation with an orthopedic surgeon will provide the preferred splinting technique. Such children need orthopedic reassessment within 24 hours.

Lateral and medial condylar fractures and intercondylar and transcondylar fractures carry risks of neurovascular compromise, especially to the ulnar nerve. These patients have soft tissue swelling and tenderness while maintaining the arm in flexion. Most patients require open reduction.

### **Radial Head Subluxation (“Nursemaid’s Elbow”)**

Radial head subluxation is a very common injury seen most often in children 1 to 4 years of age. The typical history is that the child was lifted or pulled by the hand or wrist, though 50% have no such history and parents may report a fall or simply that their child refuses to use the arm. The arm is

held in adduction, flexed at the elbow, with the forearm pronated. Gentle examination demonstrates no tenderness to direct palpation, but attempts to supinate the forearm or move the elbow cause pain. If the history and examination are strongly suggestive, radiographs are not needed. However, if the history is atypical or there is point tenderness or signs of trauma, radiographs should be obtained.

There are two maneuvers for reduction. The first, the supination/flexion technique, is performed by holding the patient's elbow at 90° with one hand and then firmly supinating the wrist and simultaneously flexing the elbow so that the wrist is directed to the ipsilateral shoulder. There may be a "click" with reduction, and the child may transiently cry and resist. The second, the hyperpronation technique, is reported to be more successful, and can be used primarily or when supination/flexion fails. The hyperpronation technique is performed by holding the child's elbow at 90° in one hand and then firmly pronating the wrist while extending the elbow. Usually the child will resume normal activity in 5 to 10 min if reduction is achieved. If the child is not better after a second reduction attempt, alternate diagnoses and radiographs should be considered. No specific therapy is needed after successful reduction. Parents should be reminded to avoid linear traction on the arm because there is a risk of recurrence.

## **Slipped Capital Femoral Epiphysis**

Slipped capital femoral epiphysis (SCFE) is more common in boys and in the obese, with a peak incidence between ages 14 and 16 years (11 and 13 years in girls). Clinically, the child presents with pain at the hip or referred to the thigh or knee. With a chronic SCFE, children complain of dull pain in the groin, anteromedial thigh, and knee, which becomes worse with activity. With walking, the leg is externally rotated and the gait is antalgic. Hip flexion is restricted and accompanied by external rotation of the thigh. Acute SCFE is due to trauma or may occur in a patient with preexisting chronic SCFE. Patients are in great pain, with marked external rotation of the thigh and leg shortening. The hip should not be forced through the full range of motion because this may displace the epiphysis further.

The differential includes septic arthritis, toxic synovitis, Legg-Calvé-Perthes disease, and other hip fractures. Children with SCFE are not febrile or toxic and have normal white blood cell counts (WBCs) and erythrocyte sedimentation rates (ESRs). On radiograph, medial slips of the femoral epiphysis will be seen on anteroposterior views, whereas frog-leg views detect posterior slips. In the anteroposterior view, a line along the superior femoral neck should transect the lateral quarter of the femoral epiphysis, but not if the epiphysis is slipped.

The management of SCFE is operative. Immediate nonweight bearing upon diagnosis is important and admission for surgical pinning is typical. The main long-term complication is avascular necrosis of the femoral head.

## **SELECTED    NONTRAUMATIC MUSCULOSKELETAL                      DISORDERS                      OF CHILDHOOD**

Kawasaki disease is discussed in [Chapter 83](#).

### **Acute Septic Arthritis**

Septic arthritis occurs in all ages, but especially in children younger than 3 years. The hip is most often affected, followed by the knee and elbow. If left untreated, purulent joint infection leads to total joint destruction. Please see [Chapter 180](#) “Acute Disorders of the Joints and Bursa” for additional information.

Radiographs may show joint effusion, but this is nonspecific. The differential includes osteomyelitis, transient tenosynovitis, cellulitis, septic bursitis, acute pauciarticular juvenile rheumatoid arthritis (JRA), acute rheumatic fever, hemarthrosis, and SCFE. Distinguishing septic arthritis from osteomyelitis may be quite difficult. Osteomyelitis is more tender over the metaphysis, whereas septic arthritis is more tender over the joint line. Joint motion is much more limited in septic arthritis. Prompt arthrocentesis

is the key to diagnosis at the bedside or, in the case of the hip, via ultrasound guidance. Synovial fluid shows WBCs and organisms.

Prompt open joint drainage in the operating room is critical in the case of the hip, or arthroscopically or via arthrocentesis in more superficial joints. In infants and children, **oxacillin** 40 milligrams/kilogram IV every 6 hours and **cefotaxime** 50 milligrams/kilogram IV every 8 hours are administered. If resistant organisms are suspected, **vancomycin** 10 to 15 milligrams/kilogram IV every 6 hours is used. The prognosis depends on the duration between symptoms and treatment, which joint is involved (worse for the hip), presence of associated osteomyelitis (worse), and the patient's age (worse for the youngest children).

## **Henoch-Schönlein Purpura**

Henoch-Schönlein purpura (HSP) is a small-vessel vasculitis characterized by purpura, arthritis, abdominal pain, and hematuria. Please see [Chapter 74](#) “Pediatric Abdominal Emergencies” for a discussion of HSP.

# **SELECTED PEDIATRIC RHEUMATOLOGIC DISORDERS**

## **Transient Synovitis of the Hip**

Transient synovitis (also called *toxic synovitis*) is the most common cause of hip pain in children younger than 10 years. The peak age is 3 to 6 years, with boys affected more than girls. The cause is unknown. Symptoms may be acute or gradual. Patients have pain in the hip, thigh, and knee, and an antalgic gait. Pain limits range of motion of the hip, but in contrast to septic arthritis, passive range of motion remains possible. There may be a low-grade fever, but patients do not appear toxic. The WBC and ESR are usually normal or mildly elevated. Radiographs of the hip are normal or show a mild to moderate effusion. The main concern is differentiation from septic arthritis, particularly if the patients are febrile, with elevation of WBC or

ESR and effusion. Diagnostic arthrocentesis is required when the diagnosis is in doubt with fluoroscopic or ultrasound guidance at the discretion of the orthopedic surgeon. The fluid in transient tenosynovitis is a sterile clear transudate.

Once septic arthritis and hip fracture have been ruled out, patients can be treated with crutches to avoid weight bearing for 3 to 7 days, no strenuous activity for 1 to 2 weeks, anti-inflammatory agents such as **ibuprofen** 10 milligrams/kilogram, and close follow-up.

## **Legg-Calvé-Perthes Disease**

Legg-Calvé-Perthes disease is avascular necrosis of the femoral head with subchondral stress fracture. Collapse and flattening of the femoral head ensues, with a potential for subluxation. The result is a painful hip with limited range of motion, muscle spasm, and soft tissue contractures. Children have a limp and chronic dull pain in the groin, thigh, and knee, which becomes worse with activity. Systemic symptoms are absent. Hip motion is restricted; there may be flexion and abduction contracture and thigh muscle atrophy. Initial radiographs (in the first 1 to 3 months) show widening of the cartilage space in the affected hip and diminished ossific nucleus of the femoral head. The second sign is subchondral stress fracture of the femoral head. The third finding is increased femoral head opacification. Deformity of the femoral head then occurs, with subluxation and protrusion of the femoral head from the acetabulum.

Bone scan and magnetic resonance imaging are very helpful in making this diagnosis by showing bone abnormalities well before plain films. The differential diagnosis includes toxic tenosynovitis, tuberculous arthritis, tumors, and bone dyscrasias.

In the ED, the most important thing is to consider this chronic and potentially crippling condition; therefore, orthopedic consultation in the ED is warranted. Nearly all children are hospitalized initially for orthopedic management.

## **Osgood-Schlatter Disease**

Osgood-Schlatter disease is common, and affects preteen boys more than girls. The cause is repetitive stress on the tibial tuberosity by the quadriceps muscle, leading to inflammation. Prolonged basketball play is a frequent culprit. Children have pain and tenderness over the tuberosity. The patellar tendon is thick and tender, with the tibial tuberosity enlarged and indurated.

Radiographs show soft tissue swelling over the tuberosity and patellar tendon thickening without knee effusion. Normally, the ossification site at the tubercle at this age will be irregular, but the prominence of the tubercle is characteristic of Osgood-Schlatter disease.

The disorder is self-limited. Acute symptoms improve after 3 months of restricted knee flexion. Crutches may be necessary, with a knee immobilizer or cylinder cast rarely needed. Exercises to stretch taut and hypertrophied quadriceps muscles are helpful.

## **Acute Rheumatic Fever**

Acute rheumatic fever (ARF) is an acute inflammatory multisystem illness primarily affecting school-age children. It is not common in the United States, but there have been recent epidemics. ARF is preceded by infection with certain strains of group A  $\beta$ -hemolytic *Streptococcus*, which stimulates antibody production to host tissues. Children develop ARF 2 to 6 weeks after symptomatic or asymptomatic streptococcal pharyngitis. Arthritis, which occurs in most initial attacks, is migratory and polyarticular, primarily affecting the large joints. Carditis occurs in 33% of patients and can affect valves, muscle, and pericardium. Sydenham chorea occurs in 10% of patients and may occur months after the initial infection. The rash, erythema marginatum, is fleeting, faint, and serpiginous, usually accompanying carditis. Subcutaneous nodules, found on the extensor surfaces of extremities, are quite rare. Carditis confers greatest mortality and morbidity.

Laboratory tests are used to confirm prior streptococcal infection (throat culture and streptococcal serology) or to assess carditis (electrocardiogram, chest radiograph, and echocardiogram). The differential includes JRA, septic arthritis, Kawasaki disease, leukemia, and other cardiomyopathies

and vasculitides. In the ED, carditis is the main management issue. Most patients are admitted.

Significant carditis is managed initially with **prednisone** 1 to 2 milligrams/ kilogram/day. Arthritis is treated with high-dose aspirin (75 to 100 milligrams/ kilogram/day to start). All children with ARF are treated with **penicillin** (or **erythromycin**, if allergic): **benzathine penicillin** 1.2 million U IM, **procaine penicillin G** 600,000 U IM daily for 10 days, or oral **penicillin VK** 250 milligrams for young children and 500 milligrams for older children given twice daily for 10 days. Long-term prophylaxis is indicated for patients with ARF, and lifelong prophylaxis is recommended for patients with carditis.

## **PostInfectious Reactive Arthritis**

Because of increased group A  $\beta$ -hemolytic streptococcal infections, postinfectious reactive arthritis (PIRA) is also increasing. PIRA is a sterile, inflammatory, nonmigratory mono- or oligoarthritis occurring with infection at a distant site with  $\beta$ -hemolytic *Streptococcus*, or less commonly with *Staphylococcus* and *Salmonella*. Unlike ARF, PIRA is not associated with carditis and in general is a milder illness. However, the arthritis in PIRA is more severe and prolonged as compared with ARF.

To make the diagnosis of PIRA, antecedent infection with group A *Streptococcus* must be determined with throat culture or 4-fold rise in ASO or anti-DNase B titer.

PIRA is responsive to nonsteroidal anti-inflammatory drugs. If group A *Streptococcus* is recovered from the throat, treatment with penicillin or erythromycin should be instituted.

## **Juvenile Rheumatoid Arthritis**

The group of diseases comprised by JRA share the findings of chronic noninfectious synovitis and arthritis, but with systemic manifestations. Pauciarticular disease is the most common form, usually involving a single large joint such as the knee. Permanent joint damage occurs infrequently.



Polyarticular disease occurs in one-third of cases. Large and small joints are affected, and there may be progressive joint damage. Systemic JRA occurs in 20% of patients. This form is associated with high fevers and chills. Extraarticular manifestations are common, including a red macular coalescent rash, hepatosplenomegaly, and serositis. The arthritis in this form may progress to permanent joint damage.

In the ED, laboratory tests focus mostly on excluding other diagnoses. Complete blood count, ESR, and C-reactive protein may be normal. Arthrocentesis may be necessary to exclude septic arthritis, particularly in pauciarticular disease. Radiographs initially show joint effusions but are nonspecific. The diagnosis of JRA is not likely to be made in the ED.

Initial therapy for patients with an established diagnosis includes nonsteroidal anti-inflammatory drugs. Glucocorticoids are occasionally used, for example, for unresponsive uveitis or decompensated pericarditis or myocarditis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 133, “Musculoskeletal Disorders in Children,” and Chapter 143C “Pediatric Procedures: Nursemaids’ Elbow Reduction” by Courtney Hopkins-Mann, Damilola Ogunnaike-Joseph, and Donna Moro-Sutherland.

## CHAPTER 83

# Rashes in Infants and Children

Lance Brown

Though rarely life threatening, rashes are a common reason for ED visits in children. Helpful clues to the specific diagnosis of rash in a child include signs and symptoms that preceded or presented with the exanthem, whether mucous membranes are involved, immunization history, human and animal contacts, and environmental exposures. Identifying outbreaks among multiple children may be useful. Pediatric exanthems can be broadly classified by etiologic agent. With few exceptions, outpatient management is appropriate for most of these conditions.

## VIRAL INFECTIONS

### Enterovirus

Included in this group are **coxsackie viruses**, **echoviruses**, and **polioviruses** with a diverse range of clinical presentations. These infections typically occur in epidemics in the summer and early fall. Many enteroviral infections lack specific clinical syndromes and presentation may include fever, upper and lower respiratory tract symptoms, gastrointestinal symptoms, meningitis, and myocarditis. The rashes of enteroviral infections also have a variety of appearances, including diffuse macular eruptions, morbilliform erythema, vesicular lesions, petechial and purpurial eruptions, rubelliform rash, roseola-like rash, and scarlatiniform eruptions.

One distinctive enteroviral infection is **hand-foot-and-mouth disease**. Initially, patients typically present with fever, anorexia, malaise, and a sore mouth. Oral lesions appear on days 2 or 3 of illness followed by skin lesions. The oral lesions start as very painful 4 to 8 mm vesicles on an erythematous base that then ulcerate. The typical location of the oral lesions is on the buccal mucosa, tongue, soft palate, and gingiva. Skin lesions start

as red papules that change to gray 3 to 7 mm vesicles that ultimately heal in 7 to 10 days. Typical locations of skin lesions include the palms, soles, and buttocks. A similar enanthem without involvement of the hands and feet is caused by a different viral subtype and known as **herpangina**, (most commonly caused by coxsackievirus A).

Management of presumed enteroviral infections typically involves symptomatic therapy ensuring adequate hydration despite the typical mouth discomfort with liberal use of analgesics such as **acetaminophen** (15 milligrams/kilogram per dose, every 4 hours), or **magic mouth-wash** (a compounded suspension of 30 mL of 12.5 milligrams/5 mL diphenhydramine liquid + 60 mL Mylanta + 4 grams Carafate) applied in small quantities to the lesions (or swish and spit) 3 times daily and before feeding. Occasional narcotics may be required to facilitate adequate outpatient hydration.

## **Measles**

Due to immunizations, measles is no longer common, but local epidemics do occur among unimmunized groups. Infection typically occurs in the winter and spring. The incubation period is 10 days, followed by a 3-day prodrome of upper respiratory symptoms and then malaise, fever, coryza, conjunctivitis, photophobia, and cough. Ill appearance is expected. Just before the development of a rash, Koplik spots, tiny white lesions on the buccal mucosa, may be seen with a “grains of sand” appearance that is pathognomonic for measles. The exanthem develops 14 days after exposure. Initially, a red, blanching, maculopapular rash develops. The rash progresses from the head to the feet and rapidly coalesces on the face, and lasts about a week. As the rash resolves, a coppery brown discoloration may be seen and desquamation can occur. Measles is self-limited and treatment is supportive.

## **Rubella**

Now quite rare due to immunizations, rubella (German measles) can be seen in teenagers, typically in the spring. The incubation period is 12 to 25 days and prodromal symptoms are similar to measles. The rash develops as fine, irregular pink macules and papules on the face that spread to the neck, trunk, and arms in a centrifugal distribution. The rash coalesces on the face as the eruption reaches the lower extremities and then clears in the same order as it appeared. Lymphadenopathy typically involves the suboccipital and posterior auricular nodes. Treatment is supportive.

## **Erythema Infectiosum**

Erythema infectiosum (also known as *fifth disease*) is a febrile illness, typically occurring in the spring, and most commonly affecting children ages 5 to 15 years. The rash starts abruptly, as a bright red macular discoloration on the cheeks producing the “slapped-cheek appearance” ([Fig. 83-1](#)). The lesions are closely grouped, tiny papules on an erythematous base with slightly raised edges. The eyelids and chin are characteristically spared. Circumoral pallor is typical. The rash fades after 4 to 5 days. As the illness progresses, and 1 to 2 days after the facial rash appears, a nonpruritic erythematous macular or maculopapular rash appears on the trunk and limbs. This rash may last for 1 week and is not pruritic. As the rash fades, central clearing of the lesions occurs, leaving a lacy reticular appearance. Palms and soles are rarely affected.



**FIGURE 83-1.** Erythema infectiosum (Fifth disease). Toddler with the classic slapped cheek appearance of fifth disease. (Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman. *Atlas of Emergency Medicine, 3rd ed.* 2010 Copyright ©McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Anne W. Lucky, MD.)

The exanthem may recur intermittently in the weeks after the onset of illness. Sun exposure or hot baths may exacerbate the rash. Associated symptoms include fever, malaise, headache, sore throat, cough, coryza, nausea, vomiting, diarrhea, and myalgias. There is no specific therapy beyond symptomatic therapy.

### **Eczema Herpeticum**

In children with existing eczema, this life-threatening, rare, viral infection can arise. The most frequent etiologic agent is herpes simplex virus. Bacterial superinfection with staphylococci or streptococci is presumed. Clinical manifestations of eczema herpeticum include fever and vesicular eruptions in areas of skin contemporaneously affected by eczematous

lesions ([Fig. 83-2](#)). Treatment includes **acyclovir** (20 milligrams/kilogram/dose PO every 8 hours) and either **trimethoprim-sulfamethoxazole** (5 milligrams/ kilogram/dose twice daily) or **clindamycin** (10 milligrams/kilogram/ dose 3 times daily) for 10 days. Inpatient admission is often necessary.



**FIGURE 83-2.** Typical appearance of eczema herpeticum. (Reproduced with permission from Shah BR, Lucchesi M. *Atlas of Pediatric Emergency Medicine*, © 2006, McGraw-Hill, New York.)

## **Varicella (Chicken Pox)**

Due to immunizations, the incidence of varicella has declined dramatically. The etiologic agent is Varicella-Zoster Virus, a herpes virus. It typically occurs in children younger than 10 years but may occur at all ages. Varicella occurs most often in the late winter and early spring. Patients are highly contagious from the prodrome phase of the illness until all lesions are crusted over. The rash starts as faint red macules on the scalp or trunk. Within the first day, lesions begin to vesiculate and develop a red base, producing the characteristic appearance ([Fig. 83-3](#)). Over the next few days, groups of lesions develop, producing the classic appearance of crops of lesions in multiple stages of development. Over the next 1 to 2 weeks, lesions become dry and crusted. The rash typically spreads centrifugally

(outward from the center). The palms and soles are usually spared. Low-grade fever, malaise, and headache are frequently seen but are typically mild. Treatment is symptomatic and includes **diphenhydramine** (1.25 milligrams/ kilogram/dose, every 6 hours as needed for itching) and **acetaminophen** (15 milligrams/kilogram/dose, every 4 hours as needed for fever). Although not needed in previously healthy children, **varicella-zoster immune globulin** and **acyclovir** (20 milligrams/kilogram up to 800 milligrams PO 5 times daily) may be needed for immunocompromised children.

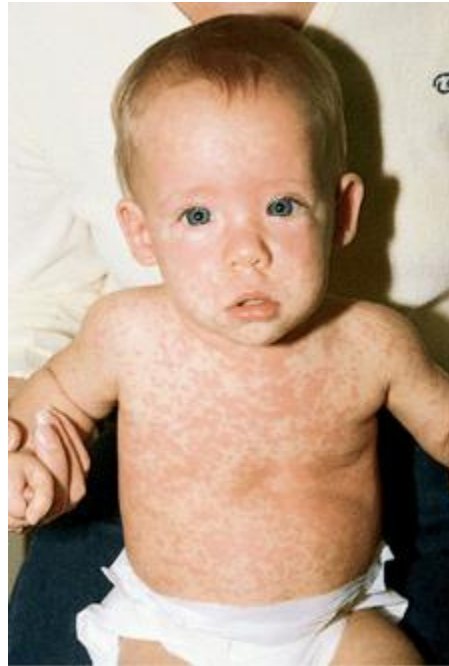


**FIGURE 83-3.** Typical rash of varicella (chicken pox). (Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ. *Atlas of Emergency Medicine, 3rd ed.* 2010 Copyright ©McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Lawrence B. Stack, MD.)

### **Roseola Infantum (Exanthem Subitum)**

Roseola is a common acute febrile illness in children ages 6 months to 3 years and thought to be caused by Human Herpes Virus 6. Roseola presents with an abrupt onset, high fever lasting 3 to 5 days. Associated symptoms are typically mild and may include irritability when the fever is highest, cough, coryza, anorexia, and abdominal discomfort. Febrile seizures may

occur. As the fever begins to resolve, blanching macular or maculopapular, rose or pink discrete lesions develop ([Fig. 83-4](#)). Areas typically affected include the neck, trunk, and buttocks but the face and proximal extremities may also be involved, though mucous membranes are spared. The rash lasts 1 to 2 days and rapidly fades. The treatment is symptomatic.



**FIGURE 83-4.** An infant with roseola infantum. (Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ. *Atlas of Emergency Medicine, 3rd ed.* 2010 Copyright ©McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Raymond C. Baker, MD.)

## FUNGAL INFECTIONS

Tinea infections are common in infants and children and named for the body parts affected: tinea capitis (scalp), corporis (skin), pedis (foot), and cruris (groin). Tinea infections typically manifest as scaly patches with pruritus of varying intensity. Successful treatment for all but tinea capitis is usually accomplished with topical creams including those available over-



the-counter (**clotrimazole**, **miconazole**, **tolnaftate**) or by prescription (**ketoconazole**, **oxiconazole**, **ciclopirox**, **terbinafine**). Treatment is continued for 7 to 10 days after the resolution of lesions. Tinea capitis ranges from mild scalp scaliness with patchy alopecia to a painful, boggy mass known as a kerion. Tinea capitis is treated with oral **griseofulvin** (ultramicrosize 15 milligrams/ kilogram/dose once daily) and **selenium sulfide** shampoo. Treatment of tinea capitis is usually for at least 8 weeks and close follow up is important as treatment response and liver function tests need to be monitored.

## **BACTERIAL INFECTIONS**

### **Impetigo**

Impetigo is a superficial skin infection, typically caused by group A  $\beta$ -hemolytic streptococci or *Staphylococcus aureus*. The lesions usually occur in small children, often in areas of insect bites or minor trauma. The lesions start as red macules and papules that form vesicles and pustules ([Fig. 83-5](#)). The formation of a golden crust results from rupture of the vesicles. The lesions may become confluent. With the exception of regional lymphadenopathy, fever and systemic signs are rare. Most commonly affected areas include the face, neck, and extremities. Diagnosis is based on the appearance of the rash. Appropriate antibiotic choices include oral **cephalexin** (12.5 to 25 milligrams/kilogram/dose 4 times daily), **trimethoprim-sulfamethoxazole** (5 milligrams/kilogram/dose twice daily), **clindamycin** (10 milligrams/kilogram/dose 3 times daily). Further treatment includes local wound cleaning and topical **mupirocin 3 times daily**.



**FIGURE 83-5.** A young girl with crusting impetiginous lesions on her chin. (Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ. *Atlas of Emergency Medicine, 3rd ed.* 2010 Copyright ©McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Michael J. Nowicki, MD.)

## **Bullous Impetigo**

Bullous impetigo typically occurs in infants and young children. Lesions are superficial, thin-walled bullae that characteristically occur on the extremities, rupture easily, leave a denuded base, dry to a shiny coating, and contain fluid that harbors staphylococci. The diagnosis usually is made by the appearance of the characteristic bullae (see [Fig. 83-6](#)). Treatment includes local wound cleaning in addition to oral antistaphylococcal antibiotics such as **clindamycin** (10 milligrams/kilogram/dose 3 times daily), or **trimethoprim-sulfamethoxazole** (5 milligrams/kilogram/dose twice daily) and topical **mupirocin**.



**FIGURE 83-6.** A child with bullous impetigo.

## **Scarlet Fever**

A distinctive rash is seen with scarlet fever. The etiologic agent is group A  $\beta$ -hemolytic streptococci (recently group C streptococci also has been implicated). Scarlet fever usually occurs in school-age children and is diagnosed by the presence of exudative pharyngitis, fever, and the characteristic rash ([Fig. 83-7](#)). Associated symptoms include sore throat, fever, headache, vomiting, and abdominal pain. The rash starts in the neck, groin, and axillae, with accentuation at flexural creases (Pastia lines). The rash is red and punctate, blanches with pressure, and has a rough sandpaper feel. Early in the course of illness, the tongue has a white coating through which hypertrophic, red papillae project (“white strawberry tongue”). Hemorrhagic spots may be seen on the soft palate. The rash typically develops 1 to 2 days after the illness onset. Facial flushing and circumoral

pallor are characteristic. Desquamation occurs with healing approximately 2 weeks after the onset of symptoms.



**FIGURE 83-7.** Scarlatiniform rash of scarlet fever; texture is typically sandpaper-like. (Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ. *Atlas of Emergency Medicine, 3rd ed.* 2010 Copyright ©McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Lawrence B. Stack, MD.)

The diagnosis generally is made on clinical grounds. Throat culture typically shows group A  $\beta$ -hemolytic streptococci or group C streptococci. Treatment is with **penicillin V** (16 milligrams/kilogram/dose 3 times daily) or **erythromycin** (10 to 15 milligrams/kilogram/dose 3 times daily) in the penicillin-allergic patient. Antibiotic treatment shortens the course of the illness and reduces the incidence of rheumatic fever.

## **Erysipelas**

Erysipelas is a cellulitis and lymphangitis of the skin due to group A - hemolytic streptococci. Fever, chills, malaise, headache, and vomiting are

common. The face is the most common site, and the lesion typically forms in the area of a skin wound or pimple. The rash starts as a red plaque that rapidly enlarges. Increased warmth, swelling, and a raised, sharply demarcated, indurated border are typical. Diagnosis is by history and the appearance of the rash. Initial treatment may be inpatient with intravenous **penicillin G** (50,000 U/kilogram/dose, every 6 hours) or **erythromycin** (10 milligrams/ kilogram/dose, every 6 hours) in the penicillin-allergic patient. Outpatient treatment includes **cephalexin** (12.5 to 25 milligrams/kilogram/dose 4 times daily), **erythromycin** (10 to 15 milligrams/kilogram/dose 3 times daily), or **clindamycin** (10 milligrams/kilogram/dose 3 times daily). Rapid clinical improvement is expected after treatment has begun.

## CELLULITIS

Cellulitis manifests a local inflammatory response at the site of infection with erythema, warmth, and tenderness. Fever is uncommon and likely indicates a more serious systemic infection, sepsis, or an unrelated concurrent viral infection. Community-associated methicillin resistant *Staphylococcus aureus* (CA-MRSA) is becoming an increasingly common cause of cellulitis in children. Traditionally, oral **cephalexin** (12.5 to 25 milligrams/ kilogram/dose 4 times daily) has been the antibiotic of choice. With the rise of CA-MRSA, **clindamycin** (10 milligrams/kilogram/dose 3 times daily) or **trimethoprim-sulfamethoxazole** (5-10 milligrams/kilogram/dose twice daily) are more common choices. Identifying underlying abscesses may require needle aspiration or bedside ultrasonography.

## UNCLEAR ETIOLOGY

### Kawasaki Disease

Kawasaki disease (mucocutaneous lymph node syndrome) is a generalized vasculitis of unknown cause that typically occurs in children younger than

9. Diagnosis depends on the following clinical findings. The patient should have at least 5 days of fever and the illness must not be explained by another known disease process. Then, 4 of the following 5 criteria must be met: (a) conjunctivitis; (b) rash; (c) lymphadenopathy; (d) oropharyngeal changes (injection of the pharynx, cracked lips, and prominent papillae of the tongue); or (e) extremity erythema and edema.

Typical rash appearances have been described as erythematous, morbilliform, urticarial, scarlatiniform, or erythema multiforme-like. Perineal rash is not uncommon. Associated findings may include leukocytosis, elevation of acute-phase reactants (eg, erythrocyte sedimentation rate and C-reactive protein), elevated liver function tests, hypoalbuminemia, anemia, arthritis, arthralgia, and irritability. Later in the illness, findings may include a rise in the platelet count (usually > 1 million), desquamation of the fingers and toes, and coronary artery aneurysms. One to 2% of patients with coronary artery aneurysms develop sudden cardiac death.

Treatment consists of **intravenous  $\gamma$ -globulin** and **aspirin** (25 milligrams/ kilogram/dose 4 times). The use of steroids is controversial.

## **Henoch-Schönlein Purpura**

Henoch-Schönlein purpura (HSP) is the most common vasculitis in childhood (see [Chapter 74](#) “Pediatric Abdominal Emergencies”). There are 4 main features to HSP: palpable purpura ranging in size from 2 to 10 mm primarily involving the buttocks, thighs, legs, and arms; gastrointestinal symptoms including vomiting, diarrhea, and abdominal pain; polyarthralgias; and hematuria and proteinuria. Children with HSP are generally well appearing and afebrile. HSP is typically self-limited and requires no treatment and no laboratory evaluation other than urinalysis (and renal function tests in the presence of hematuria). Consideration can be given to **prednisone** (1 milligram/kilogram/dose) for 2 weeks followed by a 2-week taper for severe joint and gastrointestinal symptoms and **ibuprofen** (10 milligrams/ kilogram/dose as needed every 6 hours) for severe arthralgias and extremity edema.

## **Pityriasis Rosea**

Pityriasis rosea is seen characteristically in older school-age children and young adults in the spring and fall. Pityriasis rosea does not appear to occur in epidemics and is not contagious. The rash evolves over weeks. The rash begins with a herald patch: a single red lesion with a raised border on the trunk. One to 2 weeks later, a widespread eruption of pink maculopapular oval patches erupts on the trunk in a pattern following the ribs (“Christmas tree distribution”). There may be mucous membrane involvement. Pityriasis rosea typically lasts 3 to 8 weeks. Testing for secondary syphilis is commonly done because of similarities in appearance of the rash of secondary syphilis. Treatment is symptomatic and includes **diphenhydramine** (1.25 milligrams/kilogram/dose, every 6 hours as needed for itching).

For further reading in *Emergency Medicine, A Comprehensive Study Guide*, 7th ed., see Chapter 134, “Rashes in Infants and Children,” by Gary Bonfante and Alexander M. Rosenau.

## **CHAPTER 84**

# **Sickle Cell Anemia in Children**

Ilene Claudius

Sickle cell emergencies in children include vasoocclusive crises, hematologic crises, and infections. All children with sickle cell anemia (SCA) presenting with fever, pain, respiratory distress, or a change in neurologic function require a rapid and thorough ED evaluation.

## **VASOOCCLUSIVE CRISES**

Vasoocclusive sickle episodes are due to intravascular sickling, which leads to tissue ischemia and infarction. Bones, soft tissue, viscera, and the central nervous system (CNS) may be affected. Pain may be the only symptom.

## **PAIN CRISES**

### **Clinical Features**

Pain crises are the most common SCA related presentation to the ED, and typically affect the long bones and back. They can be triggered by stress, extremes of cold, dehydration, hypoxia, or infection, but most often occur without a specific cause. In an individual patient, recurrent pain crises tend to be similar in location and quality to previous episodes. Although, typically, there are no physical findings, pain, local tenderness, swelling, and warmth may occur. Low-grade temperature elevations can occur, but true fever is rare. Infants and toddlers can present initially with dactylitis, a swelling of hands or feet, and low-grade temperature caused by ischemia and infarction of the bone marrow.

### **Diagnosis and Differential**



Differentiating between infection and vasoocclusive crisis can be difficult, particularly since infection can precipitate a pain crisis. Fever, limited range of motion of a joint, and pain that differs in location or quality from previous crises should raise concern for an infection. Pain crises can be associated with leukocytosis; however, a left shift is suspicious for infection. Sedimentation rates are unreliable markers for infections in SCA patients due to anemia. All pain crises represent ischemia, but bony infarcts present with severe, difficult to control pain, significant tenderness, and leukocytosis. Patients with bony infarcts are at risk of fat embolism.

Pain localized to the hip or inguinal area worsening with weight-bearing may be due to avascular necrosis of the femoral head, which may demonstrate flattening and collapse of the femoral head on plain radiograph.

Abdominal pain resulting from vasoocclusive crises is common and is typically abrupt in onset, and poorly localized. Tenderness and guarding may be present on examination, but not rebound or rigidity. If not typical of a pain crisis, non-SCD related causes need to be considered (eg, appendicitis), as well as SCA-related cholelithiasis/cholecystitis (gallstones can occur as early as 2 years of age), intrahepatic cholestasis (sudden right upper quadrant pain and tenderness, jaundice, anorexia, hepatomegaly, and sometimes fever), splenic sequestration, or hepatic sequestration (anemia and hepatomegaly). Laboratories, ultrasound, and CT scan may be necessary to differentiate among these etiologies.

## **Emergency Department Care and Disposition**

Pain management must be individualized, using previously effective regimens as a guide.

### **1. Mild pain**

- a. Oral hydration
- b. Oral NSAIDS (10 milligrams/kilogram **ibuprofen**)
- c. Oral narcotics (0.2 milligram/kilogram **hydrocodone**)

### **2. Moderate to severe pain**

- a. IV hydration (D5½NS at 1.5 X the age-appropriate maintenance rate),
  - b. Oral or IV NSAIDS (0.5 milligram/kilogram **ketorolac**)
  - c. IV narcotics (0.1 to 0.15 milligram/kilogram **morphine** or 0.015 milligram/ kilogram **hydromorphone**) every 15 to 30 min until pain control is achieved
3. Packed red blood cell (PRBC) transfusion for a significant drop in hemoglobin or Hgb <5 grams/dL.
  4. Admission is warranted for poor pain control or inadequate oral fluid intake. Children who have presented repeatedly for the same pain crisis also should be considered for admission.
  5. Children discharged from the ED should continue analgesics at home on a scheduled basis with PCP or hematology follow-up the next day.

## **ACUTE CHEST SYNDROME**

Acute chest syndrome is believed to represent a combination of pneumonia, pulmonary infarction, and pulmonary emboli from necrotic bone marrow. It is a leading cause of death in all patients with SCA, but especially those older than 10 years.

### **Clinical Features**

Acute chest syndrome should be considered in all patients with SCA who present with complaints of chest pain, especially when associated with tachypnea, dyspnea, cough, or other symptoms of respiratory distress. Significant hypoxia and rapid deterioration to respiratory failure can occur.

### **Diagnosis and Differential**

Chest radiographs should be obtained but may be normal during the first hours to days. The diagnosis is made in the setting of a new infiltrate on chest radiograph in the setting of chest pain and respiratory symptoms. Both

pneumonia and acute chest syndrome typically cause leukocytosis. Thrombocytopenia may accompany a severe crisis. Sputum and blood cultures are rarely positive. Pneumonia is most commonly due to atypical bacteria (chlamydia, mycoplasma), but can represent community acquired pathogens (*Streptococcus pneumoniae*) as well.

## **Emergency Department Care and Disposition**

Because deterioration can be rapid, close monitoring is required, typically in the ICU.

1. Initial stabilization: oxygen, hydration (see Pain Crisis), analgesia (see Pain Crisis)
2. Antibiotics: macrolide (eg, azithromycin 10 milligrams/kilogram PO or IV) and third generation cephalosporin (eg, 50 milligrams/kilogram/d ceftriaxone or cefotaxime IV)
3. Simple PRBC transfusion (5 mL/kg for pretransfusion Hgb of 4 to 6 grams/ dL, 10 mL/kg for pretransfusion Hgb of > 6 grams/dL) should be considered in children with severe anemia or significant hypoxia ( $\text{PaO}_2 < 70$  mm Hg or oxygen saturation > 10% decrease from baseline). If the patient has a pretransfusion Hgb  $\geq 10$  grams/dL, exchange transfusion must be used in lieu of simple transfusion to prevent further sludging.
4. All children with suspected acute chest syndrome should be admitted to the hospital.

## **ACUTE CENTRAL NERVOUS SYSTEM EVENTS**

### **Clinical Features**

Acute stroke should be considered in any patient with SCA who presents with sudden onset headache or neurologic changes, including hemiparesis, seizures, speech defects, visual disturbances, transient ischemic attacks,

vertigo, cranial nerve palsies, paresthesias, altered mental status or coma. Children with SCA are at significantly higher risk of acute ischemic stroke than unaffected children.

## **Diagnosis and Differential**

Because of the challenges in diagnosing acute ischemic stroke (eg, limited availability of MRI, CT negative in acute setting), providers may need to initiate treatment based exclusively on clinical suspicion. If clinical history is appropriate for a subarachnoid hemorrhage, workup should proceed as for a patient without SCA. Acute chest crisis, sudden severe anemia, and meningitis can cause neurologic symptoms, and appropriate tests should be sent to investigate these possibilities in the appropriate clinical setting.

## **Emergency Department Care and Disposition**

Suspected CNS vasooclusion necessitates immediate stabilization and careful monitoring. Urgent exchange transfusion should be initiated with the goal of <30% HbS (usually 1 to 2 blood volumes). As in any ischemic stroke, temperature, glucose, and oxygenation should be monitored and controlled. Alteplase has no role in the management of SCA related ischemic stroke in children. Intracranial hemorrhage should be managed in conjunction with a neurosurgeon. All children with suspected stroke should be admitted to the pediatric intensive care unit.

# **PRIAPISM**

## **Clinical Features and Diagnosis**

Priapism, a painful sustained erection in the absence of sexual stimulation, occurs when sickled cells accumulate in the corpora cavernosa. It can affect any male with SCA regardless of age, and severe prolonged attacks can

cause impotence. Typically, the patient has an edematous and tender penis, with difficulty urinating.

## **Emergency Department Care and Disposition**

Patients with priapism should receive IV hydration with D5½NS at 1.5 to 2 times maintenance and appropriate analgesia. As in non-SCA priapism, needle aspiration of the corpora cavernosa and administration of a vasoconstrictor (eg, 1:1,000,000 epinephrine solution) may be required. Patients failing this may require transfusion or exchange transfusion (see Acute Chest Crisis for transfusion guidelines). Management and admission decisions should be made promptly in consultation with urology and pediatric hematology.

## **HEMATOLOGICAL CRISES**

### **ACUTE SEQUESTRATION CRISES**

#### **Clinical Features and Diagnosis**

Intrasplenic trapping of red cells primarily affects children under 5 years of age, but can occur in older patients with SCA. Often preceded by a viral syndrome, splenic sequestration presents with sudden onset left upper quadrant pain; pallor and lethargy; tender splenomegaly; and progresses to hypotension, shock, and death. A CBC shows profound anemia (hemoglobin <6 grams/dL, or > 3 grams/dL lower than the patient's baseline level). Minor episodes can occur with insidious onset of abdominal pain, slowly progressive splenomegaly, and a more minor fall in hemoglobin level (generally the hemoglobin level remains > 6 grams/dL).

Patients may have accompanying mild neutropenia or thrombocytopenia. Less commonly, sequestration can occur in the liver. Clinical features

include an enlarged and tender liver with associated hyperbilirubinemia, severe anemia, and elevated reticulocyte count. Cardiovascular collapse is rare in this condition.

## **Emergency Department Care and Disposition**

PRBC transfusion and admission is required for major episodes. Occasionally, children with minor episodes can be observed, and discharged with close follow-up with a hematologist.

## **APLASTIC EPISODES**

Potentially life-threatening aplastic episodes are precipitated primarily by viral infections (typically parvovirus B19), and present with gradual onset of pallor, dyspnea, and fatigue. The CBC shows an unusually low hemoglobin and reticulocyte count, with normal white blood cell and platelet counts. Recovery is spontaneous within 1 to 2 weeks, and patients can typically be temporized with PRBC transfusion in the ED (with close outpatient follow-up) or inpatient setting. Rapid transfusion in severe anemia can precipitate heart failure, so small volumes (3 to 5 mL/kg) given over 3 to 4 hours should be used in severely anemic patients (Hgb  $\leq$  6 grams/dL).

## **HEMOLYTIC CRISES**

Bacterial and viral infections in children with SCA can precipitate rapid hemolysis, with sudden-onset of jaundice and pallor. A CBC shows hemoglobin level decreased from baseline, with markedly increased reticulocytosis. Specific therapy is rarely required, though transfusion may be helpful in symptomatic patients. Care should be directed toward treating the underlying infection. Close follow-up to monitor hemoglobin level and reticulocyte count should be arranged if discharged from the ED.

# INFECTIONS

## Clinical Features

Poor splenic function renders children with SCA particularly susceptible to bacterial infections, particularly in early childhood.

## Diagnosis and Differential

The NIH recommends a CBC, urinalysis, blood, urine, and throat cultures, pulse-oximetry, and a chest radiograph in all febrile children with SCA. A lumbar puncture is indicated only for clinical suspicion of meningitis. Risk factors for sepsis in children with SCA include temperature  $> 40^{\circ}\text{C}$ , WBC  $>30,000$  or  $<5000$  cells/ $\text{mm}^3$ , platelet count  $<100,000/\text{mm}^3$ , Hgb  $<5$  grams/dL, ill appearance, and history of pneumococcal sepsis.

## Emergency Department Care and Disposition

Children meeting any high-risk feature (above in Diagnosis) should receive a parenteral antibiotic with activity against *Streptococcus pneumoniae* and *H influenzae* (eg, ceftriaxone 50 milligrams/kilogram IV or IM) as soon as possible. Vancomycin should be added if the patient is at high risk for penicillin-resistant pneumococcal infection. Septic shock must be managed aggressively with early goal-directed therapy. Patients over 6 to 12 months of age without high-risk criteria who have reliable next day follow-up can be discharged following a parenteral dose of ceftriaxone pending culture results.

# VARIANTS OF SICKLE CELL DISEASE

*Sickle cell trait* is the carrier state of SCA (heterozygous). These patients are typically asymptomatic, though baseline microscopic hematuria may exist, and experience sickling only in the presence of extreme hypoxia or high altitude. *Sickle cell-hemoglobin C disease* is a heterozygous condition characterized by mild to moderate anemia and fewer complications. However, splenomegaly can persist to adulthood, and these patients remain at risk for sequestration crises. *Sickle cell  $\beta$ -thalassemia* disease is a heterozygous condition with variable severity and symptoms.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 135, "Sickle Cell Disease," by Ilene Claudius.



## **CHAPTER 85**

# **Hematologic-Oncologic Emergencies in Children**

Ilene Claudius

Childhood cancer is a leading cause of death in children, but with improvements in management and outcomes, many patients with new, active, or treated malignancies present to the ED. This chapter will cover the most common pediatric malignancies and hematologic issues. More information on malignancy-related complications is provided in [Chapter 139](#) and hemophilia and Von Willebrand Disease are discussed in detail in [Chapter 135](#).

## **CHILDHOOD LEUKEMIA**

Acute Lymphoblastic Leukemia (ALL) is the most common pediatric malignancy, with a peak incidence between 3 to 5 years of age and a 75% to 80% 5-year survival.

### **Clinical Features**

Patients can present with any of the following signs or symptoms of bone marrow infiltration: pallor, fatigue, easy bruising, fever, or bone pain. Many have hepatomegaly or splenomegaly. Rarely, acute myelogenous leukemia (AML) can present with gingival hyperplasia or subcutaneous masses (chloromas).

### **Diagnosis and Differential**

The complete blood count (CBC) with manual differential is the most useful test, though leukocytosis and blasts may be absent early in the

disease process, requiring close follow-up of patients with insidious complaints such as bone pain. WBC counts below  $4000/\text{mL}^3$ , mild anemia, and mild thrombocytopenia should raise suspicion in these cases. Abnormalities of 2 or more cell lines make leukemia more likely. If the CBC is concerning for acute leukemia, obtain a chest radiograph (for mediastinal mass); electrolytes with creatinine, uric acid, and phosphate (for evidence of tumor lysis); liver function tests and lactate dehydrogenase, PT/PTT (looking for disseminated intravascular coagulation); type and screen if anemic; and blood and urine cultures if febrile.

The differential diagnosis is extensive depending on the patient's presenting symptom. Aplastic anemia and viral infections can cause bone marrow suppression; rheumatologic diseases can overlap with symptoms and findings of leukemia; and idiopathic immune thrombocytopenia can be difficult to differentiate, though classically involves isolated destruction of the platelets without affecting other cell lines.

## **Emergency Department Care and Disposition**

Chemotherapy need not be initiated immediately in most cases. ED care is directed at potential complications and symptoms.

### 1. Anemia:

- a. Irradiated, leukodepleted packed red blood cells (PRBCs) (10 mL/kg) for life-threatening hemorrhage or hemolysis.
- b. If no hemorrhage or hemolysis, nonemergent transfusions can be given to keep hemoglobin (Hb)  $>8$  grams/dL. This should be done in coordination with oncologist.

### 2. Thrombocytopenia:

- a. Platelets (10 mL/kg) for life-threatening hemorrhage, consumption, or urgent need for invasive procedure (eg, lumbar puncture).
- b. If no urgent indication exists, nonemergent transfusions can be given to keep platelets  $>10\,000/\text{mL}^3$ . This does not need to be done in the ED.

### 3. Infection: Fever and neutropenia typically become an issue after the initiation of chemotherapy. However, granulocyte function is impaired in newly presenting leukemics, and fever or suspicion of infection in a new

leukemic should be treated emergently with broad spectrum antibiotics. In children with known neutropenia from treatment, consider unusual infections such as perirectal abscess/cellulitis and typhlitis (an appendicitis-like syndrome, treated nonsurgically) as well as bacteremia. Common choices include:

- a. Cefepime (50 milligrams/kilogram) or ceftazidime (50 milligrams/kilogram)
  - b. If ill-appearing, add gentamycin (2.5 milligrams/kilogram)
  - c. If suspicion of gram positive infection, add vancomycin (15 milligrams/ kilogram)
  - d. If anaerobic source (eg, typhlitis), add clindamycin (10 milligrams/kilogram) or metronidazole (7.5 milligrams/kilogram)
4. Tumor lysis describes the release of intracellular potassium, phosphate, and uric acid, and subsequent decline in serum calcium that occurs from turnover of tumor cells. It typically occurs with chemotherapy, but can occur prior to treatment, particularly in patients with a high tumor burden. Recognition and treatment of this life and kidney threatening condition must begin in the ED, and prevention should be considered in patients with a particularly high white blood cell (WBC) count. Specifics of management are discussed in [Chapter 139](#).
5. Hyperleukocytosis typically requires treatment if symptoms of stasis (eg, stroke, dyspnea) occur or presenting  $WBC > 200,000/mL$  for AML and  $> 300,000/mL$  for ALL in the absence of symptoms. Treatment includes:
- a. Aggressive hydration with normal saline bolus of 20 mL/kg repeated as tolerated.
  - b. If patient is symptomatic after hydration, arrange for leukapheresis.
  - c. Avoid PRBC transfusions and diuretics if possible.
  - d. Anticipate and initiate treatment for tumor lysis syndrome.
  - e. If asymptomatic with high levels, consider hydroxyurea which will half WBCs in 24 to 48 hours.

## LYMPHOMA

Hodgkin lymphoma is a lymphoid neoplasm preferentially affecting adolescents. Most cases present in the cervical or supraclavicular lymph nodes causing nontender, nonerythematous, rubbery lymphadenopathy. Systemic symptoms (eg, fever, night sweats, weight loss) occur in less than one-third of teens. A chest radiograph may demonstrate an anterior mediastinal mass. Non-Hodgkin's lymphoma can originate in or outside of the lymphatic system, and occurs in older children, particularly those with a history of immunosuppression. Because the tumor can occur in any organ, presenting signs and symptoms differ by location. A CBC, electrolytes and creatinine (looking for tumor lysis), and chest radiograph (looking for mediastinal mass) should be performed in the ED. ED care involves management of acute complications such as superior vena cava syndrome (see [Chapter 139](#) for details), avoidance of steroid therapy except in life threatening situations, and consultation with an oncologist.

## CENTRAL NERVOUS SYSTEM TUMORS

Brain tumors are common pediatric malignancies, and typically present with headaches related to increased intracranial pressure. In infants, overt signs of increased pressure (eg, bulging fontanel) can sometimes be appreciated. Vomiting, ataxia, cranial nerve palsies, or vague neurologic signs and symptoms can occur as well. CT scan or MRI are acceptable imaging studies in the ED. Seizures should be treated if present, and dexamethasone (1 milligrams/year of age up to 10 milligrams) can be given to reduce vasogenic edema. Further management should be determined by oncology and neurosurgery.

## EXTRACRANIAL SOLID TUMORS

**Neuroblastoma** is a primitive ganglion tumor that can arise in the adrenal, other abdominal location, chest, or neck. Patients can present with a painless mass, hepatomegaly, or symptoms of mass effect from compression of the bowel, bladder, lymphatics, spinal cord, trachea, or

superior vena cava. Occasionally, retrobulbar metastasis can cause raccoon eyes and proptosis. Paraneoplastic manifestations can include hypertension, watery diarrhea, and opsoclonus-myoclonus syndrome (rapid, multidirectional eye movements and jerking of the extremities). CBC for evidence of bone marrow infiltration and chest radiograph for mediastinal mass should be obtained in the ED.

**Wilms tumor**, or nephroblastoma, primarily affects young children (<10 years of age), and typically presents with an abdominal mass with few symptoms other than those explained by local compression. Many patients present with lung metastasis.

**Germ cell tumors** present as masses in the ovary or testicle, and are particularly common in boys with a history of an undescended testicle. Diagnosis is by ultrasound.

**Retinoblastoma** is a white-grey intraocular malignancy, that typically presents in children less than 2 years of age with loss of the normal red reflex ([Fig. 85-1](#)). Strabismus, decreased visual acuity, a fixed pupil, or an injected, painful eye are less common presentations. One-quarter are bilateral, and diagnosis is made through ophthalmology consultation and CT scan.



**FIGURE 85-1.** Typical appearance of leukocoria in a patient with retinoblastoma. (Reproduced with permission from Shah BR, Lucchesi M. *Atlas of Pediatric Emergency Medicine*, © 2006, McGraw-Hill Companies, Inc. All rights reserved.)

Bone and tissue sarcomas include **rhabdomyosarcoma** (a painless tissue mass), **osteosarcoma** (a bony tumor around the metaphysis of the knee, proximal humerus, pelvis, or mandible), and **Ewing sarcoma** (a bony tumor of the long bones and axial skeleton). Osteosarcoma and Ewing sarcoma can present as a dull, aching pain, particularly at night, a tender mass that seems to appear following a minor trauma, or with systemic symptoms. Diagnosis of rhabdomyosarcoma is by CT scan, while osteosarcoma and Ewing often are seen on plain radiograph, with osteosarcoma demonstrating a mixed sclerotic/lytic picture and Ewing having a destructive, “moth-eaten” appearance.

## **ANEMIA**

## **Clinical Features**

Anemia may be asymptomatic or accompanied by pallor and fatigue or heart failure.

## **Diagnosis and Differential**

Iron deficiency anemia due primarily to excessive cow's milk intake typically affects children aged 6 months to 3 years of age. Children outside of this age range should be assessed for occult GI bleeding with stool guaiac testing. Laboratory studies demonstrate anemia with a low reticulocyte count and low mean corpuscular volume (MCV). Hemolytic anemia can be primary or secondary to an underlying infection or intrinsic disorder. Patients have isolated anemia with high MCV, spherocytes and schistocytes on peripheral smear, elevated indirect bilirubin, and a positive direct antibody test.

## **Emergency Department Care and Disposition**

Most asymptomatic or minimally symptomatic patients with iron deficiency anemia can be safely discharged with careful follow-up, reduction of milk intake below 24 oz/d, and initiation of iron therapy (2 to 3 milligrams/kilogram/dose of elemental iron 3 times daily). Hemolytic anemia may require more careful observation or transfusion, based on the patient's clinical status and hemoglobin level. Steroids are indicated in the treatment of autoimmune hemolytic anemia. If transfusion is required, patients with very low hemoglobin levels (<6 grams/dL) should receive 3 to 5 mL/kg of PRBCs over 3 hours, while patients with a pretransfusion Hb >6 grams/dL can receive 10 mL/kg.

**IDIOPATHIC  
PURPURA (ITP)**

**THROMBOCYTOPENIC**

ITP is an autoimmune disorder of platelet destruction.

## **Clinical Features**

While often an isolated condition in preschool-aged children, ITP can be a feature of rheumatologic disorders (eg, lupus) or infections (eg, HIV, hepatitis C), particularly in teens. Patients can present with petechiae or bleeding (frequently epistaxis or gingiva) and, in the younger child without a coexisting condition, ITP often follows a viral infection by days to weeks.

## **Diagnosis and Differential**

Other autoimmune and infectious disorders should be considered, particularly in the teen. On CBC, other cell lines are not affected and platelets are often below  $20,000/\text{mL}^3$  with a large platelet volume. CBC and blood type should be sent.

## **Emergency Department Care and Disposition**

ITP in children carries an excellent prognosis, with 70% resolving spontaneously within 6 months, regardless of treatment. Children with minimal symptoms and platelets  $>10,000/\text{mL}^3$  are often followed without intervention. For children with significant bleeding of platelet counts  $<10,000/\text{mL}^3$ , the following options exist:

1. **Prednisone** 1 to 2 milligrams/kilogram/d for 2 to 4 weeks. Steroids should only be started in conjunction with a hematologist once leukemia has been excluded.
2. **IVIG** 1 gram/kilogram. This infusion typically runs over 4 to 6 hours and requires admission.
3. **Anti-Rh (D) immunoglobulin** (WinRho<sup>®</sup>) 75 micrograms/kilogram over 1 hour can be used in patients whose blood type is Rh+. Because these



antibodies bind the RBCs, hemolysis ensues, dropping the Hb 1 to 2 grams/dL over the week following treatment. In April 2010, a boxed warning was issued by the FDA in response to hemolysis-related deaths of primarily older adults following use of WinRho<sup>®</sup> for ITP, recommending 8 hours of monitoring for signs of hemolysis (back pain, chills, fever, discolored urine). Premedication with acetaminophen and diphenhydramine is recommended.

4. In the case of a life-threatening bleed, single-donor **platelet transfusion** (20 to 30 mL/kg), IVIG (1 gram/kilogram over 30 min), and high-dose methylprednisolone (30 milligrams/kilogram) should be considered. This platelet dose is much higher than the normal dose of 5 to 10 mL/kg of a single donor pack or 1 U/10 kilogram of the pooled or “random” donor packs. In a patient without antibodies, this lower dose will increase the platelet level by 30,000 to 50,000.

## **NEUTROPENIA**

Neutropenia implies a neutrophil count below  $1000/\text{mL}^3$ , but risk of infections increases significantly when the count falls below  $500/\text{mL}^3$ .

### **Clinical Features**

Neutropenia can be asymptomatic or associated with serious bacterial illness.

### **Diagnosis and Differential**

Benign forms of neutropenia include: benign transient neutropenia (from viral infections or medications), autoimmune neutropenia, and cyclic neutropenia. More serious forms of neutropenia are chronic and persistent, such as congenital agranulocytosis, or chemotherapy related.

## **Emergency Department Care and Disposition**

Patients with benign forms of neutropenia and evidence of infection can typically be discharged home, though consultation with the patient's hematologist and a single dose of **ceftriaxone** (50 milligrams/kilogram) may be considered depending on the clinical situation. Patients with fever and a more serious neutropenia variant should have blood cultures sent and broad spectrum antibiotics initiated.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 136, "Oncology and Hematology Emergencies in Children," by Rick Place, Anne Marie T. Lagoc, Thomas A. Mayer, and Christopher J. Lawlor

# CHAPTER 86

## Renal Emergencies in Infants and Children

Deborah R. Liu

Renal emergencies in children represent a large and varied group of disease processes. This chapter will focus on common renal emergencies, including acute renal failure (ARF) in children, acute glomerulonephritis, and nephrotic syndrome. For discussion of other renal emergencies, see the following topics and chapters: end-stage renal disease ([Chapter 52](#) “Emergencies in Renal Failure and Dialysis Patients”), urolithiasis ([Chapter 56](#) “Urologic Stone Disease”), hypertension ([Chapter 26](#) “Hypertension”), Henoch-Schönlein Purpura ([Chapter 74](#) “Pediatric Abdominal Emergencies”), hemolytic uremic syndrome ([Chapter 133](#) “Evaluation of Anemia and the Bleeding Patient”), and hematuria ([Chapter 53](#) “Urinary Tract Infections and Hematuria”).

### ACUTE RENAL FAILURE

Acute renal failure (ARF) is the abrupt loss of renal function such that body fluid homeostasis can no longer be maintained. As a whole, ARF in children is relatively sporadic, with specific incidences related to individual causes. Some of the more common causes of ARF in children include severe dehydration, sepsis, pyelonephritis, hemolytic uremic syndrome, acute glomerulonephritis, postoperative complications, and posterior urethral valves in boys.

#### Clinical Features

The clinical signs of ARF are varied and are determined by the underlying cause. Patients present with symptoms of the underlying cause (eg, bloody diarrhea and abdominal pain in hemolytic uremic syndrome; or fever,

hypotension, and petechiae in sepsis). Ultimately, the patient will manifest stigmata of renal failure: nausea and anorexia due to uremia, headache from hypertension, edema (periorbital, scrotal or labial, dependent, or generalized), weight gain, and decreased urine output.

## **Diagnosis and Differential**

ARF may be anatomically categorized as prerenal, renal, or postrenal in etiology. [Table 86-1](#) lists common causes of renal failure in infants and children. Urinalysis helps distinguish among the three forms of ARF and should be obtained along with microscopic evaluation. Children who are not toilet-trained or those with significantly decreased urine output require catheterization to obtain urine. Prerenal causes of ARF are associated with little blood or protein on urinalysis, but typically manifest high urine specific gravity ( $> 1.025$ ). Children with acute tubular necrosis typically have granular casts on urinalysis but usually have normal specific gravity. Glomerulonephritis and other glomerular diseases are characterized by hematuria and proteinuria. A positive urine dipstick test for blood without red blood cells on microscopy suggests hemoglobinuria or myoglobinuria. Basic blood tests such as serum electrolytes, BUN and creatinine, as well as a complete blood count (CBC) should be obtained in all cases of ARF to help identify the cause of the ARF and guide management. Additional blood tests may be indicated depending on the clinical scenario.

Prerenal or Inadequate Renal Perfusion	Renal or Intrinsic Renal Injury or Disease	Postrenal or Obstruction
Dehydration from GI losses	Tubular diseases (tubular necrosis; effects of contrast agents, chemotherapy)	Solitary kidney
Blood loss from trauma	Myoglobinuria, hemoglobinuria, tumor lysis syndrome	Urethral obstruction (posterior urethral valves, ureteropelvic junction obstruction)
Capillary leakage in burns, sepsis, third spacing	Glomerular diseases (Henoch-Schönlein purpura, glomerulonephritis, nephrotic syndrome)	Pelvic mass (lymphoma, rhabdomyosarcoma)
Limited cardiac output in congenital heart disease, myocarditis, cardiogenic shock	Interstitial lesions from medications (antibiotics, antifungals, NSAIDs)	Nephrolithiasis

**TABLE 86-1** Causes of Renal Failure in Infants and Children

## Emergency Department Care and Disposition

The goals of treatment for ARF are to identify the underlying cause of renal failure and to correct fluid and electrolyte imbalances. Address life-threatening complications, such as severe hyperkalemia (see [Chapters 4](#) and [81](#)) or hypertensive emergency (see [Chapter 26](#)) immediately. Consult a pediatric nephrologist for most cases of ARF as many cases require inpatient admission.

1. For *prerenal ARF*, resuscitate with crystalloid fluids, starting with **20 mL/kg of normal saline without added potassium**. For prerenal ARF due to hemorrhagic shock, give crystalloid fluids until blood products are available, then transfuse packed red blood cells in aliquots of 10 mL/kg.
2. For ARF due to intrinsic renal injury or disease, treatment depends on the clinical state of the patient and the etiology of the ARF. For example, it may be necessary to **fluid restrict** despite oliguria in order to achieve overall fluid balance in the hypervolemic patient. It is imperative to monitor the patient's weight and fluid input as well as output. Treat hypertension with an antihypertensive agents as described in [Chapter 26](#).

3. For postrenal (obstructive) ARF, insert a **Foley catheter** to relieve the obstruction. Fluid management is again directed towards achieving homeostasis, with careful record of total input and output. Antihypertensive agents and/or diuretics may also be necessary to control significant hypertension.

## **ACUTE GLOMERULONEPHRITIS**

Acute glomerulonephritis is characterized by hematuria and proteinuria. There are many causes of acute glomerulonephritis, including postinfectious etiologies, the most common of which follows infection with group A  $\beta$ -hemolytic *Streptococcus*. Other less frequent causes include Henoch-Schönlein purpura, hemolytic uremic syndrome, systemic lupus erythematosus, IgA nephropathy, and Goodpasture syndrome.

### **Clinical Features**

Patients typically present with sudden onset of brown, tea-colored, or grossly bloody urine. Patients may also note foamy urine due to proteinuria. Other symptoms include decreased urine output, headaches due to hypertension, or peripheral edema. In post-*streptococcal* glomerulonephritis, there may be a history of sore throat 1 to 2 weeks preceding urinary symptoms. The physical examination can be completely normal, or may demonstrate hypertension, edema, or even congestive heart failure.

### **Diagnosis and Differential**

The diagnosis of glomerulonephritis is made by examination of the urine. The urinalysis shows hematuria and proteinuria (usually at least 2+ protein [100 milligrams/dL] on the urine dipstick). Red blood cell casts are seen on microscopy. Other laboratory testing should include a CBC, electrolytes, BUN and creatinine, and urine culture (infection may present as hematuria

with proteinuria). In addition, tests for poststreptococcal glomerulonephritis (antistreptolysin O titers, C3 and C4 complement levels) should be sent. To aid in differentiating glomerulonephritis from nephrotic syndrome, serum albumin and serum triglycerides and cholesterol levels should be obtained.

## **Emergency Department Care and Disposition**

The treatment of glomerulonephritis is determined by the underlying cause. Address hypertensive emergencies first. Patients with new-onset glomerulonephritis and oliguria or hypertension usually require admission. Patients with mild disease may be discharged home after consultation with a pediatric nephrologist, on a low-sodium diet with monitoring of fluid intake and with close follow-up.

## **NEPHROTIC SYNDROME**

The hallmarks of nephrotic syndrome include significant proteinuria, hypoproteinemia, edema, and hyperlipidemia. Nephrotic syndrome can be divided into primary (only affecting the kidney) or secondary nephrotic syndrome (multisystem disease with kidney involvement). The most common form of primary nephrotic syndrome is minimal change disease. Other causes include focal glomerulosclerosis, mesangial proliferative glomerulonephritis, and membranoproliferative glomerulonephritis. Secondary forms of nephrotic syndrome include lupus, Henoch-Schönlein purpura, sickle cell disease, and drug or toxin exposure (eg, heavy metals).

### **Clinical Features**

Patients typically present with edema, which may involve the face, abdomen, scrotum or labia, or extremities. Because facial swelling or puffy eyes are not specific symptoms, patients are often misdiagnosed as having an allergic reaction by a prior medical provider. The patient or parent may note foamy urine (proteinuria) or dark urine (hematuria). Extreme

hypoproteinemia may cause pleural effusions with associated shortness of breath or orthopnea as well as abdominal ascites causing pain, nausea, vomiting, or anorexia.

## **Diagnosis and Differential**

The initial diagnostic criteria include edema, heavy proteinuria (usually 3+ [300 milligrams/dL] or 4+ [2000 milligrams/dL] on urine dipstick testing), and hypoproteinemia (serum albumin <3.0 grams/dL). Hypercholesterolemia (> 200 milligrams/dL) is classically seen with nephrotic syndrome, though an inconsistent finding. Further testing is useful to distinguish primary from secondary nephrotic syndrome, and may include serum antinuclear antibody, serum immunoglobulins, screening for sickle cell disease, or serum complement levels.

## **Emergency Department Care and Disposition**

Managing the fluid status of the nephrotic syndrome patient can be challenging. Some patients may be intravascularly depleted but show signs of fluid overload with significant edema. Mild cases of nephrotic syndrome do not require any fluid resuscitation. Treatment of most cases of nephrotic syndrome should be performed in consultation with a pediatric nephrologist.

1. Treat hypovolemic shock with 20 mL/kg normal saline, even in the setting of severe edema.
2. Treat mild to moderate dehydration with small, but frequent amounts of a low-sodium oral solution.
3. Treat volume overload and edema with **furosemide** 0.5 to 1 milligrams/kilogram/dose. If the serum albumin is extremely low, administer **25% albumin** (0.5 to 1 gram/kilogram) over 4 hours followed by furosemide 1 to 2 milligrams/kilogram IV (higher dose of furosemide may be needed in this setting).



4. Definitive treatment of nephrotic syndrome often includes oral corticosteroids, however, this should be initiated in conjunction with a pediatric nephrologist.

Many patients with nephrotic syndrome can safely be discharged home on a low-salt diet with close follow-up. Indications for admission include severe edema (eg, pulmonary effusion or ascites causing respiratory symptoms), symptomatic hypertension, suspected bacterial infection (eg, spontaneous bacterial peritonitis with ascites), significant intravascular dehydration, and renal insufficiency.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 128, “Renal Emergencies in Infants and Children,” by Christine E. Koerner.

**SECTION 10 Infectious and Immunologic  
Diseases**

# CHAPTER 87

## Sexually Transmitted Diseases

David M. Cline

This chapter covers the major sexually transmitted diseases (STDs) in the United States, with the exception of human immunodeficiency virus, which is discussed in [Chapter 92](#). Vaginitis and pelvic inflammatory disease (PID) are covered separately in [Chapters 63](#) and [64](#), respectively.

### GENERAL RECOMMENDATIONS

Multiple STDs infections frequently occur concurrently, compliance and follow-up are often limited or unreliable, infertility and other long-term morbidities may result from lack of treatment. When an STD is suspected, treat with single-dose regimens whenever possible. Ascertain pregnancy status and consider an obstetrics consultation if the patient is pregnant. Screen for other STDs (HIV infection, syphilis, and hepatitis) in the ED or through follow-up. Provide counseling for STD prevention in the ED and assure HIV testing in the ED or through follow-up as indicated. Advise that the partner(s) seek treatment and counsel on the appropriate time to reengage in sexual relations. Arrange follow-up as local resources allow.

### CHLAMYDIAL INFECTIONS

#### Clinical Features

*Chlamydia trachomatis* causes urethritis, epididymitis, orchitis, proctitis, or Reiter syndrome (nongonococcal urethritis, conjunctivitis, and rash) in men and urethritis, cervicitis, PID, and infertility in women. In both sexes, asymptomatic infection is common. There is a high incidence of coinfection with *Neisseria gonorrhoeae*. The incubation period is 1 to 3 weeks, with

symptoms varying from mild dysuria with purulent or mucoid urethral discharge to sterile pyuria and frequency (urethritis). Women may present with mild cervicitis or with abdominal pain, findings of PID, or peritonitis. Men may present with a tender swollen epididymis or testicle.

## **Diagnosis and Differential**

Diagnosis is best made with indirect detection methods such as enzyme-linked immunosorbent assay or DNA probes, which have a sensitivity of 75% to 90%. The Centers for Disease Control and Prevention (CDC) recommends a nucleic acid amplification test to be used as screening tests for *Chlamydia*. Culture is possible but difficult and produces a low yield.

## **Emergency Department Care and Disposition**

1. **Azithromycin** 1 gram PO as a single dose or **doxycycline** 100 milligrams PO twice daily for 7 days is the treatment of choice for uncomplicated urethritis or cervicitis.
2. Alternatives include 7-day treatment with **erythromycin** 500 milligrams PO 4 times a day, **ofloxacin** 300 milligrams twice daily, or **levofloxacin** 500 milligrams PO daily.

# **GONOCOCCAL INFECTIONS**

## **Clinical Features**

*Neisseria gonorrhoeae* (GC) is a gram-negative diplococcus that causes urethritis, epididymitis, orchitis, and prostatitis in men and urethritis, cervicitis, PID, and infertility in women. Rectal infection and proctitis with mucopurulent anal discharge and pain can occur in both sexes. The incubation period ranges from 3 to 14 days. Women tend to present with nonspecific lower abdominal pain and mucopurulent vaginal discharge with

findings of cervicitis and possibly PID. Eighty percent to 90% of men develop symptoms of urethritis: dysuria and purulent penile discharge within 2 weeks. Men also may present with acute epididymitis and orchitis or prostatitis. Occasionally, GC can be isolated from the throat, but it rarely causes symptomatic pharyngitis. Disseminated GC is a systemic infection that occurs in 2% of untreated patients with GC, most often women, and is the most common cause of infectious arthritis in young adults. An initial febrile bacteremic stage includes skin lesions (tender pustules on a red base, usually on the extremities, and may include palms and soles), tenosynovitis, and myalgias. Over the next week, these symptoms subside, followed by mono- or oligoarticular arthritis with purulent joint fluid.

## **Diagnosis and Differential**

For uncomplicated GC, urethral or cervical cultures are the standard diagnostic tests. A Gram stain of urethral discharge showing intracellular gram-negative diplococci is very useful in men; cervical smears are unreliable in women. Diagnosis of disseminated GC is primarily clinical because results of culture of blood, skin lesions, and joint fluid are positive in only 20% to 50% of patients. Culturing the cervix, rectum, and pharynx may improve the yield. A positive GC culture result from a partner supports the diagnosis.

## **Emergency Department Care and Disposition**

1. Effective therapy for uncomplicated gonorrhea (not PID) includes single-dose regimens of **cefixime** 400 milligrams PO, or **ceftriaxone** 250 milligrams IM.
2. Alternatives include single-dose regimens of **spectinomycin** 2 grams IM, or **ceftizoxime**, 500 milligrams IM single dose, or **cefoxitin**, 2 grams IM single dose, plus **probenecid**, 1 gram PO single dose, or **cefotaxime**, 500 milligrams IM single dose.
3. Disseminated gonorrhea is treated initially with parenteral **ceftriaxone** 1 gram daily IM/IV, 24 to 48 hours after there is clinical improvement, then

the patient can be switched to oral **cefixime** 400 milligrams daily for 7 to 10 total antibiotic therapy days.

4. Treatment for possible coinfection with *Chlamydia* also should be given (see above).

## TRICHOMONAS INFECTIONS

### Clinical Features

*Trichomonas vaginalis* is a flagellated protozoan that causes vaginitis with malodorous yellow-green discharge and urethritis. Abdominal pain also may be present. Trichomoniasis in pregnancy has been associated with premature rupture of membranes, preterm delivery, and low birth weight. In men, infection is often asymptomatic (90% to 95%), but urethritis may be present. The incubation period varies from 3 to 28 days.

### Diagnosis and Differential

Diagnosis is based on finding the motile, flagellated organism on a saline wet preparation of vaginal discharge or in a spun urine specimen.

### Emergency Department Care and Disposition

1. **Metronidazole** 2 grams PO in a single dose is the treatment of choice (alternatively, 500 milligrams PO twice daily for 7 days). Alternatively **tinidazole**, 2 grams PO single dose.
2. Metronidazole is a pregnancy category B drug, and it is the drug of choice for treating symptomatic pregnant patients. The CDC guidelines state that pregnant women may be treated with a single 2-gram dose of **metronidazole**.

## SYPHILIS

## Clinical Features

*Treponema pallidum*, a spirochete, causes syphilis. It enters the body through mucous membranes and nonintact skin. Syphilis occurs in 3 stages. The primary stage is characterized by the chancre (see [Fig. 87-1](#)), a single painless ulcer with indurated borders that develops after an incubation period of 21 days on the penis, vulva, or other areas of sexual contact (including the vagina or cervix). The primary chancre heals and disappears after 3 to 6 weeks. The secondary stage occurs several weeks after the chancre disappears. Rash and lymphadenopathy are the most common symptoms. The rash starts on the trunk, spreads to the palms and soles, and is polymorphous, most often dull red and papular (similar to that of *Pityriasis rosacea*), but it may also take on other forms such as psoriatic or pustular lesions. The rash is not pruritic. Constitutional symptoms are common, including fever, malaise, headache, and sore throat. Mucous membrane involvement (“mucous patches”) includes oral or vaginal lesions, and condyloma lata, which are flat, moist, wartlike growths, may occur at the perineum, anogenital region, or adjacent areas (thighs). This stage also resolves spontaneously. *Latency* refers to the period between stages during which a patient is asymptomatic. Any patient with secondary or latent syphilis who presents with neurologic symptoms or findings should have a lumbar puncture and cerebrospinal fluid testing for neurosyphilis. Late stage or tertiary syphilis, which is less common (classically found in 33% of untreated patients), occurs years after the initial infection and affects primarily the cardiovascular and neurologic systems. Specific manifestations include neuropathy (tabes dorsalis), meningitis, dementia, and aortitis with aortic insufficiency and thoracic aneurysm formation.



**FIGURE 87-1.** Syphilis chancre in a male. A painless ulcer caused by syphilis is seen on the distal penile shaft with a smaller erosion on the glans. The ulcer is quite firm on palpation. (Reproduced with permission from Wolff K, Johnson RA. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology*, 6th ed. © 2009 by McGraw-Hill Companies, Inc. All rights reserved.)

## **Diagnosis and Differential**

Syphilis may be diagnosed in the early stages with dark-field microscopic identification of the treponemes from the primary chancre or secondary condyloma or oral lesions. Serologic tests include nontreponemal (VDRL and rapid plasma reagin) and treponemal (fluorescent treponemal antibody absorption test). Nontreponemal test results are positive about 14 days after the appearance of the chancre. There is a false positive rate of approximately 1% to 2% of the population. Treponemal tests are more sensitive and specific but harder to perform.

## **Emergency Department Care and Disposition**



1. Syphilis in all stages remains sensitive to penicillin, which is the drug of choice: **benzathine penicillin G** 2.4 million units IM as a single dose. Latent or tertiary syphilis is treated as above with 3 weekly IM injections.
2. Doxycycline, 100 milligrams po twice daily for 14 days or tetracycline, 500 milligrams 4 times daily for 14 days.



**FIGURE 87-2.** Genital herpes in a male. Classic vesicles are shown proximally on the penis; several formerly vesicular lesions have crusted over. (Reproduced with permission from Wolff K, Goldsmith LA, Katz SI, et al: *Fitzpatrick's Dermatology in General Medicine, 7th ed.* © 2008 by McGraw-Hill Companies, Inc. All rights reserved.)

3. Intravenous high-dose penicillin is the only treatment with proven benefit for neurosyphilis (tertiary).

## **HERPES SIMPLEX INFECTIONS**

### **Clinical Features**

Herpes simplex virus type 2 and, less often, type 1 cause genital herpes by invading mucosal surfaces or nonintact skin. In primary infections, clusters of painful pustules or vesicles on an erythematous base occur 7 to 10 days after contact with an infected person. These lesions ulcerate and may coalesce over the next 3 to 5 days, and in women a profuse watery vaginal discharge may develop. Tender inguinal adenopathy is usually present. Dysuria is common and may lead to frank urinary retention due to severe pain. Systemic symptoms are common in first infections and include fever, chills, headache, and myalgias. The untreated illness lasts 2 to 3 weeks and then heals without scarring. The virus remains latent in the body, however, and continues to be shed in urogenital secretions of asymptomatic patients, making transmission to partners possible. Recurrences occur in most patients (60% to 90%) but are usually briefer and milder without systemic symptoms.

## **Diagnosis and Differential**

The diagnosis is usually clinical, based on the characteristic appearance. Viral cultures for herpes simplex virus taken from vesicles or early ulcers are more reliable than the Tzanck smear for intranuclear inclusions.

## **Emergency Department Care and Disposition**

1. Treatment of choice for primary genital herpes is a 7- to 10-day course of **acyclovir** 400 milligrams PO 3 times daily, **valacyclovir** 1 gram PO twice daily, or **famciclovir** 1 gram PO twice daily.
2. In those cases severe enough to require hospitalization, treatment with intravenous **acyclovir** 5 to 10 milligrams/kilogram body weight every 8 hours IV may be given.
3. Treatment for episodes of recurrent genital herpes consist of a 5-day course of **acyclovir** 400 milligrams PO twice daily, **valacyclovir** 500 milligrams to 1 gram PO twice daily, or **famciclovir** 250 milligrams PO twice daily. If started at the onset of symptoms, antiviral therapy may reduce the severity and duration of the episode.

# CHANCROID

## Clinical Features

Caused by *Haemophilus ducreyi*, a pleomorphic gram-negative bacillus, chancroid is more common in the tropics, but in recent years there has been a rise in cases in the United States, with epidemic outbreaks. Incubation is 4 to 10 days. A tender papule on an erythematous base appears on the external genitalia and then over 1 to 2 days erodes to become a painful purulent or pustular ulcer with irregular edges (see [Fig. 87-3](#)). Multiple ulcers may be present. The ulcers are usually 1 to 2 cm in diameter with sharp, undermined margins and are very painful. “Kissing lesions” may occur due to autoinoculation of adjacent skin. Tender inguinal adenopathy, usually unilateral, follows in 50% of untreated patients within 1 to 2 weeks, and these nodes may meet together to form a mass (bubo) that becomes necrotic, suppurates, and drains. Constitutional symptoms are rare.



**FIGURE 87-3.** Chancroid ulcer in a male. The lesion is very painful. The friable base of the ulcer is covered with yellow-gray necrotic exudates. (Reproduced with permission from Wolff K, Goldsmith LA, Katz SI, et al: *Fitzpatrick’s Dermatology in General Medicine, 7th ed.* © 2008 by McGraw-Hill Companies, Inc. All rights reserved.)

## Diagnosis and Differential

Diagnosis is usually clinical, with care to exclude syphilis. Sometimes the organism may be cultured from a swab of the ulcer or pus from a bubo, but special media are required.

## Emergency Department Care and Disposition

1. Treatment regimens include **azithromycin** 1 gram PO as a single dose, **ceftriaxone** 250 milligrams IM as a single dose, **erythromycin** 500 milligrams PO 3 times a day for 7 days, or **ciprofloxacin** 500 milligrams PO twice daily for 3 days. Symptoms usually improve within 3 days, but large ulcers may require 2 to 3 weeks to heal.
2. Bubo may be aspirated to relieve pain from swelling but should not be excised.

# LYMPHOGRANULOMA VENEREUM

## Clinical Features

Three serotypes of *C trachomatis* are associated with lymphogranuloma venereum (LGV), which is endemic in other parts of the world but uncommon in the United States. The primary lesion, usually occurring 5 to 21 days after exposure, is a painless, small papule or vesicle (see [Fig. 87-4](#)) that may go unnoticed and heals spontaneously in a 2 to 3 days. After anal intercourse, primary LGV may present as painful mucopurulent or bloody proctitis. Several weeks to months after the primary lesion, painful inguinal adenopathy (unilateral in 60%) occurs. The nodes mat together to form a bubo (often with a purplish hue to the overlying skin) and often suppurate and form fistulae. “Groove sign,” an indentation across the bubo that parallels the inguinal ligament, may be seen. Systemic symptoms may include fever, chills, arthralgias, erythema nodosum, and, rarely,

meningoencephalitis. Late sequelae include scarring; urethral, vaginal, and anal strictures; and occasionally lymphatic obstruction.



**FIGURE 87-4.** Lymphogranuloma venereum chancre. This ulceration was painless to the patient. (Reproduced with permission from Wolff K, Goldsmith LA, Katz SI, et al: *Fitzpatrick's Dermatology in General Medicine, 7th ed.* © 2008 by McGraw-Hill Companies, Inc. All rights reserved.)

## **Diagnosis and Differential**

Diagnosis is through serologic testing and culture of LGV from a lesion. A complement fixation titer for LGV greater than 1:64 is consistent with infection.

## **Emergency Department Care and Disposition**

1. Doxycycline 100 milligrams po twice daily for 21 days is the treatment of choice.

2. An alternative is erythromycin 500 milligrams po 4 times daily for 21 days.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 144, "Sexually Transmitted Diseases," by Flavia Nobay and Susan B. Promes.

# **CHAPTER 88**

## **Toxic Shock**

Vicky Weidner

### **TOXIC SHOCK SYNDROME**

Toxic shock syndrome (TSS) is a severe, life-threatening syndrome that can progress rapidly to multisystem dysfunction, severe electrolyte disturbances, renal failure and shock. Colonization or infection by *Staphylococcus aureus* has been implicated in the majority of cases. Although TSS was initially related to menstruating females, now the spectrum of patients at risk includes both genders and all ages. Risk factors for TSS includes recent menstruation, postpartum or postabortion status, cutaneous lesions such as burns, tattoos, piercings, varicella lesions or recent surgical sites, and intracavitary foreign objects such as nasal packing, intrauterine devices, and vaginal sponges.

#### **Clinical Features**

TSS is characterized by high fever, profound hypotension, diffuse erythrodermatous rash, mucus membrane hyperemia, diffuse myalgias, nonfocal neurologic abnormalities to include lethargy, agitation, confusion, headache or seizures, vomiting, diarrhea or abdominal pain that can rapidly progresses to multisystem dysfunction, organ failure, and death. The rash associated with TSS is described as a “painless sunburn” that typically fades within 3 days and is followed by full-thickness desquamation.

#### **Diagnosis and Differential**

TSS must be considered in any acute febrile illness associated with erythroderma, hypotension, and multiorgan involvement. Diagnostic criteria are listed in [Table 88-1](#). When considering TSS, the evaluation should include arterial blood gas analysis; a complete blood count with a differential count; electrolyte determinations, including magnesium, calcium, creatinine phosphokinase; coagulation panel; urinalysis; electrocardiogram; and a chest x-ray. If neurologic abnormalities are present, a head CT and lumbar puncture should be considered. Other syndromes to consider in the differential diagnosis of TSS include streptococcal TSS (STSS), Kawasaki disease, staphylococcal scalded skin syndrome, Rocky Mountain spotted fever, and septic shock.

1. Fever: temperature $\geq 38.9^{\circ}\text{C}$ ( $\geq 102.0^{\circ}\text{F}$ )
2. Rash: diffuse macular erythroderma
3. Desquamation: 1 to 2 wk after onset of illness
4. Hypotension
5. Multisystem involvement ( $\geq 3$ of the following)
a. Gastrointestinal: vomiting or diarrhea at onset of illness
b. Muscular: severe myalgias or creatine kinase elevation twice the normal level
c. Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperemia
d. Renal: serum urea nitrogen or creatinine at least twice the upper limit of normal for laboratory or urinary sediment with pyuria ( $\geq 5$ leukocytes/high power field) in the absence of urinary tract infection
e. Hepatic: total bilirubin, alanine aminotransferase enzyme, or aspartate aminotransferase enzyme levels at least twice the upper limit of normal for the laboratory
f. Hematologic: platelets $< 100\,000/\text{mL}$
g. Central nervous system: disorientation or alterations in consciousness
6. Laboratory criteria: negative CSF, blood, or throat cultures (if collected) for any organism except <i>Staphylococcus Aureus</i>

Case classification: *probable*: a case in which 5 of the 6 specified criteria described above are present; *confirmed*: a case in which all 6 of the specified criteria described above are present, including desquamation, unless the patient dies before desquamation occurs.

Source: Adapted from Toxic-Shock Syndrome (TSS): 1997 Case Definition. Centers for Disease Control and Prevention. Available at: [http://www.cdc.gov/ncphi/diss/nndss/casedef/toxicss\\_1990.htm](http://www.cdc.gov/ncphi/diss/nndss/casedef/toxicss_1990.htm).

**TABLE 88-1** Diagnostic Criteria for Toxic Shock Syndrome

## Emergency Department Care and Disposition



The most important treatment of TSS consists of early, aggressive fluid resuscitation with continuous monitoring of blood pressure, heart rate, respiratory rate, oxygenation, urinary output, and central venous pressure. Early surgical consultation should be considered for removal of infected tissue. The early use of appropriate antibiotics will not affect the acute disease presentation but will reduce bacterial load, further toxin production, and recurrence.

1. An initial fluid bolus of 1 to 2 L of crystalloid intravenous (IV) fluids (normal saline) should be used initially for hypotension and fluid resuscitation. A central venous pressure catheter should guide further fluid resuscitation. Large volumes of IV fluids may be required (up to 20 L) over the first 24 hours.
2. If needed to maintain blood pressure, start **dopamine** 5 to 20 micrograms/ kilogram/min. If there is no response to an infusion of 20 micrograms/ kilogram/min, start norepinephrine to keep the mean arterial pressure at least at 65 mm Hg. Usual doses of **norepinephrine** range from 2.5 to 20 micrograms/kilogram/min.
3. Fresh-frozen plasma, packed red blood cells, or platelets may be given to correct any coagulation abnormalities (see [Chapter 138](#)).
4. All potentially infected sites, including blood, should be cultured before initiating antibiotic therapy.
5. Any foreign bodies such as tampons, piercings, surgical or nasal packing should be removed immediately.
6. Antistaphylococcal antimicrobial therapy with  $\beta$ -lactamase resistant agent, such as **nafcillin or oxacillin** 2 grams IV every 4 hours, *plus* **clindamycin** 600 to 900 milligrams IV every 8 hours. **Vancomycin** 1 gram every 12 hours or **linezolid** 600 milligrams every 12 hours may be added if methicillin resistant strains are suspected. Parenteral antibiotics are needed for at least 3 days and then oral for another 10 to 14 days.
7. Other considerations include the use of methylprednisolone and IV immunoglobulin. If no improvement with 6 hours of aggressive therapy, 1 to 2 grams/kilogram of IV immunoglobulin has shown some improvement. (Contraindicated in Immunoglobulin A deficiency.)

# **STREPTOCOCCAL TOXIC SHOCK SYNDROME**

STSS is defined as any group A streptococcal infection associated with invasive soft tissue infection, early onset of shock, and organ failure. STSS is very similar to TSS but is associated with soft tissue infection that is culture positive for *Streptococcus pyogenes*. Labeled the “flesh-eating bacteria” by the news media, group A streptococcal infections cause streptococcal necrotizing fasciitis, with mortality rates of 30% to 80%.

## **Clinical Features**

Abrupt onset of soft tissue pain localized to one area that is out of proportion to physical findings combined with fever is the most common presentation of STSS followed closely by shock and multisystem organ involvement. The area of involvement is most commonly the extremities but can be anywhere on the body. The global erythematous rash is much less common in STSS. Vesicles and bullae at the site of soft tissue infection that progress to a violaceous or blue discoloration are ominous signs of necrotizing fasciitis or myositis. Necrotizing fasciitis may develop rapidly and carries a poor prognosis. Adult respiratory distress syndrome develops in 55% of patients.

## **Diagnosis and Differential**

Diagnostic criteria are listed in [Table 88-2](#). Laboratory evaluation includes a complete blood count with differential; arterial blood gas analysis; liver function tests; serum electrolyte, magnesium, and calcium determinations; Creatinine Kinase; a coagulation profile; blood cultures; electrocardiogram; chest x-ray; and urinalysis and cultures of the affected area. Immediate surgical consultation with a CT or MRI of the affected area may help

confirm the diagnosis but should not delay consultation. The differential diagnosis is the same as for TSS.

Hypotension
Multiorgan involvement (characterized by $\geq 2$ of the following)
1. Renal impairment: creatinine level twice normal
2. Coagulopathy
3. Liver involvement: enzyme or bilirubin level twice normal
4. Acute respiratory distress syndrome
5. Generalized erythematous macular rash that may desquamate
6. Soft tissue necrosis, including necrotizing fasciitis or myositis or gangrene
Laboratory Criteria: isolation of group A streptococcus

Case classification: *Probable*: a case that meets the clinical case definition in the absence of another identified etiology for the illness and with isolation of group A streptococcus from a nonsterile site (eg, throat, vagina, sputum) *Confirmed*: a case that meets the clinical case definition and with isolation of group A streptococcus from a normally sterile site (eg, blood or cerebrospinal fluid or, less commonly, joint, pleural, or pericardial fluid).

**TABLE 88-2** Diagnostic Criteria for Streptococcal Toxic Shock Syndrome  
Hypotension

### Emergency Department Care and Disposition

1. Treatment is similar to that for TSS, with aggressive fluid resuscitation, central venous monitoring, intensive care unit admission and vasopressors, as needed. Intubation is almost always warranted because of the high incidence of ARDS.
2. Antistreptococcal antimicrobial therapy is started with IV **penicillin G** 24 million units per day in divided doses *plus* IV **clindamycin** 900 milligrams every 8 hours or **linezolid** 600 milligrams IV every 12 hours. In penicillin-allergic patients **ceftriaxone** 2 grams IV every 24 hours plus **clindamycin** 900 milligrams IV every 8 hours can be used. Administration of IV immunoglobulin can be helpful. See details under TSS care.
3. **Immediate surgical consultation is mandatory because most patients require debridement, fasciotomy or amputation.**

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 145, “Toxic Shock Syndrome and Streptococcal Toxic Shock Syndrome,” by Shawna J. Perry and Reneé D. Reid.

# **CHAPTER 89**

## **Septic Shock**

John E. Gough

The majority of sepsis cases are caused by gram-negative and gram-positive bacteria; however, sepsis is a heterogeneous clinical syndrome that can be caused by any class of microorganism including fungi, mycobacteria, viruses, rickettsiae, and protozoa. Predisposing factors for gram negative bacterial sepsis include diabetes mellitus, lymphoproliferative diseases, cirrhosis, burns, invasive procedures, and chemotherapy. Risk factors for gram positive sepsis include vascular catheters, burns, indwelling mechanical devices, and injection drug use. Nonbacterial sepsis is more commonly seen in immunocompromised individuals

### **CLINICAL FEATURES**

Vital signs often reveal hyperthermia or hypothermia, tachycardia, and tachypnea. Early clinical features of sepsis include obtundation; hyperventilation; hot, flushed skin; and a widened pulse pressure. In elderly, very young, or immunocompromised patients, the clinical presentation may be atypical, with no fever or localized source of infection.

In the early stages of septic shock, vasodilation is common. Myocardial depression occurs; however, cardiac output and stroke volume are usually maintained. Sepsis is the most common condition associated with acute lung injury and acute respiratory distress syndrome (ARDS). Clinically, severe refractory hypoxemia, noncompliant “heavy” lungs, and a CXR showing bilateral pulmonary alveolar infiltrates are present.

Renal manifestations of septic shock include acute renal failure with azotemia, oliguria, and active urinary sediment. The most frequent hepatic abnormality is cholestatic jaundice. Concentrations of transaminase, alkaline phosphatase (1 to 3 times the normal level), and bilirubin (usually not >10 milligrams/dL) increase.

The most frequent hematologic changes of septic shock are neutropenia or neutrophilia, thrombocytopenia, and disseminated intravascular coagulation (DIC). Neutrophilic leukocytosis with a “left shift” results from demargination and release of less mature granulocytes from the marrow. Gram-negative infections precipitate DIC more readily than do gram-positive infections. Hyperglycemia can develop, even without a history of diabetes. Uncontrolled hyperglycemia is a significant risk for adverse outcome. Adrenal insufficiency may also be seen.

Blood gas analysis performed early in the course of septic shock usually demonstrates respiratory alkalosis. As perfusion worsens and continues, tissue hypoxia generates more lactic acid and metabolic acidosis worsens.

Cutaneous lesions that occur as a result of sepsis can be divided into 5 categories: direct bacterial involvement of the skin and underlying soft tissues (cellulitis, erysipelas, and fasciitis); lesions from hematogenous seeding of the skin or the underlying tissue (petechiae, pustules, cellulitis, ecthyma gangrenosum); lesions resulting from hypotension and/or DIC (acrocyanosis and necrosis of peripheral tissues); lesions secondary to intravascular infections (microemboli and/or immune complex vasculitis); and lesions caused by toxins (toxic shock syndrome).

## **DIAGNOSIS AND DIFFERENTIAL**

Septic shock should be suspected in any patient with a temperature of  $> 38.3^{\circ}\text{C}$  ( $> 100.9^{\circ}\text{F}$ ) [ $>38.5^{\circ}\text{C}$  ( $101.3^{\circ}\text{F}$ ) in children] or  $<36^{\circ}\text{C}$  ( $<96.8^{\circ}$ ) and a systolic blood pressure of  $<90$  mm Hg (or 2 SD below normal for age in children) with evidence of inadequate organ perfusion. The hypotension of septic shock does not typically reverse with rapid volume replacement of at least 1 L of isotonic crystalloid (or 20 mL/kg in children). History and physical examination findings combined with some basic laboratory or radiologic investigations usually identify a presumptive source for sepsis. Focus particular attention on the central nervous system, pulmonary system, intraabdominal structures, urinary tract, skin, and soft tissues. See [Table 89-1](#) for diagnostic criteria, use of the qualifier “some” in the table

acknowledges the fact that clinicians use judgement when making the diagnosis of “sepsis” in an individual patient.

**Infection, documented or suspected, and some\* of the following:**

**General variables**

Fever [ $>38.3^{\circ}\text{C}$  ( $>100.9^{\circ}\text{F}$ ), or  $>38.5^{\circ}\text{C}$  ( $>101.3^{\circ}\text{F}$ ) in children]  
Hypothermia [core temperature  $<36^{\circ}\text{C}$  ( $<96.8^{\circ}\text{F}$ )]  
Core to peripheral temperature gap  $>3^{\circ}\text{C}$  ( $>5.4^{\circ}\text{F}$ )  
Heart rate  $>90$  beats/min or in children  $>2$  SD above the normal value for age  
Tachypnea:  $>30$  breaths/min or in children  $>2$  SD above normal for age  
Altered mental status or in children, a decrease in Glasgow Coma Score of  $>3$  points from abnormal baseline or a Glasgow Coma Score  $<11$   
Significant edema or positive fluid balance ( $>20$  mL/kg over 24 h)  
Hyperglycemia (plasma glucose level  $>140$  milligrams/dL or  $7.7$  mmol/L) in the absence of diabetes

**Inflammatory variables**

Leukocytosis (WBC count  $>12,000$  /mm<sup>3</sup>), or in children above normal for age  
Leukopenia (WBC count  $<4000$ /mm<sup>3</sup>) or in children below normal for age  
Normal WBC count with  $>10\%$  immature forms  
Plasma C-reactive protein level  $>2$  SD above the normal value  
Plasma procalcitonin level  $>2$  SD above the normal value

**Hemodynamic variables**

Arterial hypotension (SBP  $<90$  mm Hg; mean arterial pressure  $<70$  mm Hg; or SBP decrease of  $>40$  mm Hg in adults or in children  $<2$  SD below normal for age)  
Mixed venous oxygen saturation  $<70\%$   
Cardiac index  $<3.5$  L/min/m<sup>2</sup>  
Need for vasoactive drugs to maintain blood pressure in normal range

**Organ dysfunction variables**

Arterial hypoxemia ( $\text{PaO}_2/\text{FiO}_2 < 300$ ) *in absence of cyanotic heart disease*  
 $\text{PaCO}_2 > 65$  torr or 20 mm Hg over baseline  $\text{PaCO}_2$   
Acute oliguria (urine output  $< 0.5$  mL/kg/h or 45 mmol/L for at least 2 h, despite adequate fluid resuscitation)  
Creatinine level increase  $\geq 0.5$  milligram/dL, or *in children*  $\geq 2$  times upper limit of normal for age or twofold increase in baseline creatinine level  
Coagulation abnormalities (INR  $> 1.5$  or activated partial thromboplastin time  $> 60$  s, or *in children* INR  $> 2$ )  
Ileus (absent bowel sounds)  
Thrombocytopenia (platelet count  $< 100,000/\text{mm}^3$ ) or *in children* platelet count  $< 80,000/\text{mm}^3$  or a decline of 50% in platelet count from highest value recorded over the past 3 d (for patients with chronic hematologic/oncologic diseases)  
Hyperbilirubinemia (plasma total bilirubin  $> 4$  milligrams/dL or 70 mmol/L) or *in children* alanine transaminase level 2 times upper limit of normal for age

**Tissue perfusion variables**

Hyperlactatemia (lactate level  $> 3$  mmol/L) or *in children*  $> 2$  times upper limit of normal  
Decreased capillary refill or mottling  
*Unexplained metabolic acidosis: base deficit  $> 5.0$  mEq/L*

Key:  $\text{FiO}_2$  = fraction of inspired oxygen, INR = international normalized ratio, SBP = systolic blood pressure, SD = standard deviation, WBC = white blood cell.

Criteria exclusive to children are listed in *italics*.

\*See the Diagnosis section for discussion of the use of the quantifier "some."

**TABLE 89-1** Diagnostic Criteria for Sepsis in Adults and Children

## ANCILLARY STUDIES

Although there is no specific laboratory test for the diagnosis of septic shock, tests are useful because they (1) assess the general hematologic and metabolic state of the patient, (2) provide results that suggest the potential for occult bacterial infection, and (3) can detect a specific microbial cause of infection. Basic laboratory studies should include a complete blood count and platelet count; DIC panel; serum electrolytes; liver function panel; renal function panel; arterial blood gas analysis, lactic acid level; and urinalysis. Blood should be typed and cross-matched if low hematocrit is suspected. A CXR should be part of the basic evaluation. Perform lumbar puncture in any patient with a clinical presentation compatible with meningitis. In adults, obtain at least 2 separate sets of specimens for blood culture from different venipuncture sites. Other laboratory tests for markers of sepsis



may be considered: C-reactive protein (CRP), serum lactic acid, procalcitonin, and semiquantitative interleukin-6 levels.

The differential diagnosis of septic shock includes the other nonseptic causes of shock such as cardiogenic, hypovolemic, anaphylactic, neurogenic, obstructive (pulmonary embolism, tamponade), and endocrine (adrenal insufficiency, thyroid storm) causes.

## **EMERGENCY DEPARTMENT DISPOSITION**

**Early goal directed therapy.** Three components comprise the core of early goal-directed therapy: (1) optimization of oxygenation, ventilation, and circulation; (2) initiation of drug therapy, including antibiotics; and (3) control of the source of sepsis.

1. Aggressive airway management with high-flow oxygen (keeping oxygen saturation greater than 90%) through endotracheal intubation may be necessary.
2. Rapid infusion of crystalloid IV fluid (lactate Ringer solution or normal saline) at 500 mL (20 mL/kg in children) every 5 to 10 min should be accomplished. Often, 4 to 6 L (60 mL/kg in children) is necessary. In the early goal-directed therapy guidelines, early invasive monitoring (central venous pressure and, in appropriate cases, monitoring via arterial catheter) is recommended. Maintain central venous pressures between 8 and 12 mm Hg, mean arterial pressure > 65 mm Hg, and venous oxygenation saturation level > 70%. Keep the patient's hematocrit at > 30% if the venous oxygen saturation target (70%) is not achieved. Urine output (> 30 mL/h in adult, > 1 mL/kg/h in children) should be monitored. With ongoing blood loss current international guidelines recommend transfusion at a hemoglobin level of 7 to 9 grams/L.
3. **Vasopressors.** Adults: If there is no hemodynamic response after administration of 3 to 4 L of fluid or if there are signs of fluid overload (continued elevated central venous pressure or pulmonary edema), administer dopamine or norepinephrine (central line required). The **dopamine** dose ranges from 5 to 20 micrograms/kilogram/min. If there is no response to an infusion of 20 micrograms/kilogram/min, start

norepinephrine to keep the mean arterial pressure at least at 65 mm Hg. Usual **doses of norepinephrine** range from 2.5 to 20 micrograms/kilogram/min. Children: Infants <6 months of age are insensitive to dopamine and dobutamine, due to incomplete development of sympathetic innervation and insufficient stores of norepinephrine. Pediatric dopamine-resistant shock commonly responds to norepinephrine or epinephrine.

4. The source of infection must be removed (eg, removal of indwelling catheters and incision and drainage of abscesses).
5. Empiric antibiotic therapy is ideally begun after obtaining cultures, but administration should not be delayed. Dosages should be the maximum allowed and given intravenously.
6. ADULTS (nonneutropenic-source unknown): therapy should be effective against gram-positive and gram-negative organisms. **Imipenem** 500 mg IV every 6 hours can be used. Alternatives include **ertapenem**, 1 gram IV every 24 hours **plus vancomycin**, 15 milligrams/kilogram every 6 hours or 1 gram IV every 12 hours.
7. Pneumonia is suspected source: **ceftriaxone**, 1 to 2 grams IV every 12 hours **plus azithromycin**, 500 milligrams IV, then 250 milligrams IV every 24 hours, or **levofloxacin**, 750 milligrams IV every 24 hours **or moxifloxacin**, 400 milligrams IV every 24 hours **plus vancomycin**, 15 milligrams/kilogram IV every 6 hours, or 1 gram IV every 12 hours.
8. Biliary source suspected: **ampicillin/sulbactam**, 3 grams IV every 6 hours or **piperacillin/tazobactam**, 4.5 grams IV every 6 hours or **ticarcillin/clavulanate**, 3.1 grams IV every 4 hours.
9. Intraabdominal source is suspected: **imipenem**, 500 milligrams IV every 6 hours to 1 gram IV every 8 hours **or meropenem**, 1 gram IV every 8 hours or doripenem 500 milligrams IV every 8 hours or **ertapenem**, 1 gram IV every 24 hours or **ampicillin/sulbactam**, 3 grams IV every 6 hours or **piperacillin/tazobactam**, 4.5 grams IV every 6 hours.
10. Urinary source: **piperacillin/tazobactam**, 4.5 grams IV every 6 hours or **ampicillin**, 1 to 2 grams IV every 4 to 6 hours **plus gentamicin**, 1.0 to 1.5 milligrams/kilogram every 8 hours.
11. IV drug use or indwelling device source suspected: there is a high probability of gram-positive etiology, **vancomycin** 15

milligrams/kilogram IV every 6 hours, or 1 gram IV every 12 hours is recommended.

12. CHILDREN (nonneutropenic) Neonates <1 week of age: **ampicillin**, 25 milligrams/kilogram IV every 8 hours **plus cefotaxime**, 50 milligrams/kilogram IV every 12 hours. Neonates 1 to 4 weeks: **ampicillin**, 25 milligrams/kilogram IV every 6 hours **plus cefotaxime**, 50 milligrams/kilogram IV every 8 hours. Infants 1 to 3 months: **ceftriaxone**, 75 milligrams/kilogram IV every 24 hours **or cefotaxime**, 50 milligrams/kilogram IV every 8 hours. Children 1 to 3 months: **ceftriaxone**, 75 to 100 milligrams/kilogram every 24 hours **or cefotaxime**, 50 milligrams/kilogram IV every 8 hours.
13. NEUTROPENIC CHILDREN AND ADULTS. For adults: **ceftazidime**, 2 gram IV every 8 hours; for children, 50 milligrams/kilogram IV every 8 hours up to adult dosage **or imipenem**, 500 milligrams IV every 6 hours to 1 gram IV every 8 hours in adults; for children: age > 3 months, 15 to 25 milligrams/kilogram IV every 6 hours; age 1 to 3 months, 25 milligrams/kilogram IV every 6 hours; age 1 to 4 weeks, 25 milligrams/kilogram IV every 8 hours; age <1 week, 25 milligrams/kilogram IV every 12 hours **PLUS vancomycin**, 15 milligrams/kilogram IV 6 hours.
14. DIC should be treated with fresh frozen plasma, 15 to 20 mL/kg initially, to keep PT at 1.5 to 2 times normal, and treated with a platelet infusion of 6 units, to maintain a serum concentration of at least 50,000/ $\mu$ L.
15. Corticosteroids are not recommended for septic patients who are not in shock. Dosages of hydrocortisone should be  $\leq$  300 milligrams/d. An adrenocorticotropic hormone stimulation test is not recommended, and hydrocortisone is preferred over dexamethasone.
16. Current international guidelines recommend “judicious glycemic control” to keep glucose levels <150 milligrams/dL in patients with septic shock.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 146, “Septic Shock,” by Jonathan Jui.

# **CHAPTER 90**

## **Soft Tissue Infections**

Chris Melton

Patients with soft tissue infections present frequently to the emergency department (ED). The management of these infections involves an understanding of appropriate antibiotic treatment, outpatient or inpatient treatment options, and an understanding of when surgical intervention is necessary.

### **METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS***

Community-acquired MRSA is epidemic across all populations. A significant majority of soft tissue infections in adults and children are caused by community-acquired MRSA. Understanding the treatment of community-acquired MRSA is vital for those managing soft tissue infections in the ED.

#### **Clinical Features**

Community-acquired MRSA is a frequent cause of skin and soft tissue infections. Lesions are typically warm, red, tender, and may be draining a purulent fluid. MRSA lesions are frequently mistaken as spider bites by patients as well and clinicians.

#### **Diagnosis and Differential**

The diagnosis of MRSA is largely clinical. Community-acquired MRSA should be considered in any infection where *S aureus* or *Streptococcus* is

typically considered the etiologic agent. This includes skin and soft tissue infections as well as sepsis and pneumonia. Bedside ultrasound is helpful to identify abscess collections in equivocal cases.

## **Emergency Department Care and Disposition**

1. For many community-acquired MRSA cutaneous infections, adequate incision and drainage (I&D) is adequate to manage these infections. Suggested criteria for withholding antibiotics include: abscesses that are small <5 cm, abscesses in immunocompetent patients, and abscesses without accompanying cellulitis.
2. If local epidemiology supports MRSA as the likely etiology of cellulitis, antibiotics effective against MRSA should be given. These include **clindamycin** 300 milligrams PO 4 times daily or **trimethoprim/sulfamethoxazole** double strength 1 to 2 tablets twice a day for 7 to 10 days. Consider adding **Cephalexin** 500 milligrams 4 times daily to a regimen with trimethoprim/sulfamethoxazole to cover streptococcus. If the infection is severe, **vancomycin** 1 gram every 12 hours should be used, and inpatient therapy is indicated.
3. Patients who are at the extremes of age, have fever, significant comorbidities, or have a large number of lesions may require admission for parenteral antibiotics.

## **NECROTIZING SOFT TISSUE INFECTIONS**

Necrotizing soft tissue infections are a spectrum of conditions that may be polymicrobial or monomicrobial. Group A *Streptococcus* and *S aureus* are often the etiologic agent in monomicrobial infections. Clostridial infections are now uncommon secondary to improved hygiene and sanitation.

### **Clinical Features**

Patients present with pain out of proportion to physical findings and a sense of heaviness in the affected part. Physical findings typically include a combination of edema, brownish skin discoloration, bullae, malodorous serosanguineous discharge, and crepitation. The patient frequently has a low-grade fever and tachycardia out of proportion to the fever. Mental status changes, including delirium and irritability, may accompany necrotizing soft tissue infections.

## **Diagnosis and Differential**

Familiarity with the disease and an appreciation of the subtle physical findings are the most important factors in making the diagnosis of necrotizing soft tissue infections. Additional findings that may confirm the clinical suspicion include gas within soft tissue on plain radiographs, metabolic acidosis, coagulopathy, hyponatremia, leukocytosis, anemia, thrombocytopenia, myoglobinuria, and renal or hepatic dysfunction.

## **Emergency Department Care and Disposition**

1. The patient with necrotizing soft tissue infections should be adequately resuscitated with crystalloid intravenous (IV) fluids and packed red blood cells if there is significant hemolysis with anemia.
2. Urine output and central venous pressure readings should be used to assess volume status. Vasoconstrictors should be avoided in these patients because of compromised perfusion in the affected extremity.
3. IV antibiotics should be administered including **vancomycin** 1 gram IV every 12 hours *plus* **meropenem** 500 to 1000 milligrams IV every 8 hours. Alternatively **piperacillin/tazobactam** 4.5 grams IV every 6 hours may be used. The use of clindamycin should also be considered as it inhibits toxin synthesis.
4. Tetanus prophylaxis should be administered as indicated.
5. Surgical consultation for debridement should be obtained immediately and may include fasciotomy or amputation. Hyperbaric oxygen therapy and IV immunoglobulin therapy are controversial and typically the decision of the treating surgeon.

# CELLULITIS

Cellulitis is a local soft tissue inflammatory response secondary to bacterial invasion of the skin. It is more common in the elderly, immunocompromised patients, and patients with peripheral vascular disease.

## Clinical Features

Cellulitis presents as localized tenderness, erythema, and induration. Lymphangitis and lymphadenitis may accompany cellulitis and indicate a more severe infection. Patients may have fever and chills but are infrequently bacteremic.

## Diagnosis and Differential

The clinical presentation is usually sufficient for diagnosis. Obtaining a white cell count or blood cultures rarely changes management of otherwise healthy patients with simple cellulitis. The differential diagnosis includes any erythematous skin condition. Cellulitis of the lower extremity is sometimes complicated by deep venous thrombosis and may require venogram or Doppler studies for a complete evaluation. In patients with systemic toxicity (fever and leukocytosis) cultures of pus, bullae or blood should be obtained.

## Emergency Department Care and Disposition

1. Simple cellulitis in which MRSA is not suspected can be treated in an outpatient setting using **cephalexin** 500 milligrams PO 4 times daily, **dicloxacillin** 500 milligrams PO 4 times daily, or **clindamycin** 300 milligrams 4 times daily. If local epidemiology supports a high likelihood of MRSA in patients with soft tissue infections, antibiotics effective

against MRSA should be given. In these cases clindamycin, trimethoprim/ sulfamethoxazole, or doxycycline +/- cephalixin should be given.

2. All patients discharged should have close follow-up within 2 to 3 days to evaluate the cellulitis and response to therapy. Skin markers may be helpful to mark the extent of cellulitis in patients discharged from the ED.
3. All patients with systemic toxicity or evidence of bacteremia should be admitted to the hospital. Patients with diabetes mellitus, alcoholism, or other immunosuppressive disorders should be considered for admission for IV antibiotics.
4. IV antibiotics, such as clindamycin, vancomycin or linezolid, should be used in patients requiring hospital admission.

## **ERYSIPELAS**

Erysipelas is a superficial cellulitis with lymphatic involvement caused primarily by group A *Streptococcus*. Infection is usually through a portal of entry in the skin.

### **Clinical Features**

Onset is acute, with sudden high fever, chills, malaise, and nausea. Over the next 1 to 2 days, a small area of erythema with a burning sensation develops. The erythema is sharply demarcated from the surrounding skin and is tense and painful. Lymphangitis and lymphadenitis are common. Purpura, bullae, and necrosis may accompany the erythema. It is primarily an infection of the lower extremities.

### **Diagnosis and Differential**

The diagnosis is based primarily on physical findings. Leukocytosis is common. Cultures, ASO titers, and anti-DNAase B titers are of little use in the ED. Differential diagnosis includes other forms of local cellulitis. Some



believe necrotizing fasciitis is a complication of erysipelas and should be considered in all cases.

## **Emergency Department Care and Disposition**

1. Treatment is with parenteral antibiotics active against streptococci including **ceftriaxone** 1 gram every 24 hours or **cefazolin** 1 to 2 grams every 8 hours. If it is difficult to distinguish between cellulitis and erysipelas, cover for *S aureus* as well as streptococci (see earlier). If the disease is severe, treat for MRSA with vancomycin, clindamycin, or linezolid and admit to the hospital.
2. Patients with mild disease may be treated with an initial dose of parenteral antibiotics and discharged on **penicillin** 500 milligrams PO every 6 hours. If the patient is allergic to penicillin a macrolide or cephalosporin may be used. Duration of treatment is 5 to 10 days and these patients should be reevaluated in 2 days for follow up.

## **CUTANEOUS ABSCESSSES**

Cutaneous abscesses are the result of a breakdown in the cutaneous barrier, with subsequent contamination with resident bacterial flora. Incision and drainage (I and D) is usually the only necessary treatment.

### **Clinical Features and Diagnosis**

Patients present with an area of swelling, tenderness, and overlying erythema. The area of swelling is frequently fluctuant. Cutaneous abscesses are usually localized, although they may cause systemic toxicity in the immunosuppressed. Cutaneous abscesses should be inspected closely for predisposing injury and foreign bodies. Radiography may be indicated if foreign body is suspected. Needle aspiration or ultrasound may aid in the diagnosis when it is unclear whether the patient has an abscess or cellulitis.

## Emergency Department Care and Disposition

1. See [Chapter 7](#) for information on procedural sedation.
2. **Bartholin gland abscess** presents as unilateral painful swelling of the labia with a fluctuant 1 to 2 cm mass. These infections are typically polymicrobial, however, may contain *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Routine antimicrobial treatment is not necessary unless there is a suspicion of sexually transmitted disease. Treatment involves I&D along the vaginal mucosal surface of the abscess, generally followed by the insertion of a Word catheter. The Word catheter can be left in place for up to 4 weeks. Sitz baths are recommended after 2 days. Follow up with gynecology is recommended within 2 days in patients with severe symptoms and within one week in patients with mild symptoms.
3. **Hidradenitis suppurativa** is a recurrent chronic infection involving the apocrine sweat glands. These abscesses tend to occur in the axilla and in the groin. The causative organism is usually *Staphylococcus*, although *Streptococcus* also may be present. The abscesses are typically multiple and in different stages of progression. ED treatment involves I and D of any acute abscess, treating with antibiotics for any cellulitis that may be present, and referral to a surgeon for definitive treatment.
4. **Infected sebaceous cysts** may develop in the sebaceous glands, which occur diffusely throughout the skin. Cysts present with an erythematous, tender, cutaneous mass that is often fluctuant. I&D is the appropriate ED treatment, with wound rechecks in 2 to 3 days in the ED or physician's office. The cyst contains a capsule that must be removed to prevent recurrence. This capsule can sometimes be grasped at the time of the initial I&D however, this is typically done at a later follow-up visit.
5. **Pilonidal abscess** presents as a tender, swollen, and fluctuant mass along the superior gluteal fold. Treatment includes I&D followed by iodoform gauze packing. The patient should be rechecked in 2 to 3 days, and the wound should be repacked. Surgical referral is usually necessary for definitive treatment. Antibiotics are not necessary unless there is an accompanying cellulitis.

6. *Staphylococcal soft tissue infection* may cause *folliculitis*, the inflammation of a hair follicle caused by bacterial invasion, and is usually treated with warm compresses. When deeper invasion occurs, the soft tissue surrounding the hair follicle becomes infected, and a furuncle (boil) is formed. Warm compresses are usually adequate to promote spontaneous drainage. If several furuncles coalesce, they may form a large area of interconnected sinus tracts and abscesses called a *carbuncle*. Carbuncles usually require surgical referral for wide excision.
7. In the healthy, immunocompetent patient, routine use of antibiotics following abscess I&D is not indicated unless there is a secondary infection.
8. In the potentially immunocompromised patient, the threshold for antibiotic use should be lowered. Patients presenting with secondary cellulitis or systemic symptoms should be considered for antibiotic therapy. Abscesses involving the hands and face also should be treated more aggressively with antibiotics.
9. Prophylaxis for endocarditis in patients with structural cardiac abnormalities should be considered (see [Chapter 93](#) for information on those at risk).

## SPOROTRICHOSIS

Sporotrichosis is caused by traumatic inoculation of the fungus *Sporothrix schenckii*, which is found on plants and in the soil.

### Clinical Features

After a 3-week incubation period, 3 types of infection may occur. The fixed cutaneous type occurs at the site of inoculation and looks like a crusted ulcer or verrucous plaque. The local cutaneous type also remains at the site of inoculation but presents as a subcutaneous nodule or pustule. The surrounding skin may become erythematous. The lymphocutaneous type is the most common of the three. It presents as a painless nodule at the site of

inoculation that develops subcutaneous nodules that migrate along lymphatic channels.

## **Diagnosis and Differential**

The diagnosis is based on the history and physical examination. Tissue biopsy cultures are often diagnostic but of limited use in the ED. The differential diagnosis includes tuberculosis, tularemia, cat-scratch disease, leishmaniasis, nocardiosis, and staphylococcal lymphangitis.

## **Emergency Department Care and Disposition**

1. **Itraconazole** 100 to 200 milligrams/d PO for 3 to 6 months is highly effective when treating sporotrichosis.
2. If disseminated, sporotrichosis may be treated with IV **amphotericin B** 0.5 milligram/kilogram/d.
3. Most cases of cutaneous sporotrichosis can be treated on an outpatient basis. Those patients who have systemic symptoms or who are acutely ill should be admitted for possible treatment with amphotericin B.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 147, "Soft Tissue Infections," by Elizabeth W. Kelly and David Magilner.

# CHAPTER 91

## Disseminated Viral Infections

Matthew J. Scholer

Viral illnesses are among the most common reasons that people come to an emergency department (ED). This chapter reviews some of the more serious viral infections that cause disseminated illness or have a predilection for the central nervous system (CNS). Treatment of primary herpes zoster is discussed in [Chapter 83](#), and mononucleosis is discussed in [Chapters 68](#) and [153](#). Genital herpes is discussed in [Chapter 87](#). Treatment of the human immunodeficiency virus is covered in [Chapter 92](#), and treatment of cytomegalovirus is discussed in [Chapter 99](#).

## HERPES SIMPLEX VIRUS 1

Transmission of herpes simplex virus (HSV) occurs by contact of mucous membranes or open skin with infected fluids (saliva, vesicle fluid, semen and cervical fluid) from persons with ulcerative lesions or from those who are shedding the virus without overt disease. **Herpes simplex infections are treatable with antiviral drugs, so early recognition of serious infection is important.**

### Clinical Features

Symptomatic HSV-1 infection most commonly results in orolabial lesions, whereas HSV-2 is most often implicated in genital herpes (see [Chapter 87](#)). Primary infections typically produce more extensive lesions involving mucosal and extramucosal sites, and may be accompanied by systemic signs and symptoms. Gingivostomatitis and pharyngitis are typical manifestations of primary HSV-1 infection. The lesions are distributed throughout the mouth and consist of small, thin-walled vesicles on an

erythematous base, although they do not always become vesicular. The primary lesions generally last for 1 to 2 weeks. In children younger than 5 years, it may present as a pharyngitis or gingivostomatitis associated with fever and cervical lymphadenopathy. Recurrent HSV-1 infections present as herpes labialis. These lesions occur in 60% to 90% of infected individuals, are usually milder, and generally occur on the lower lip at the outer vermilion border. Less common skin manifestations include herpetic whitlow (finger) and herpes gladiatorum (skin), the latter is commonly seen in wrestlers and other athletes involved in close contact sports.

HSV-1 is one of the most common viral causes of encephalitis in the United States. It occurs most commonly in patients <20 years and > 50 years of age. The mortality rate for untreated disease is > 70%. The hallmark of HSV encephalitis is acute onset of fever and neurologic symptoms including focal motor or cranial nerve deficits, ataxia, seizures, and altered mental status or behavioral abnormalities. Herpes infections, including herpes zoster, in immunocompromised hosts can lead to widespread dissemination with multiorgan involvement.

## **Diagnosis and Differential**

The diagnosis of HSV infection is largely clinical because confirmatory testing takes days to weeks to be performed and thus is of little use in the ED setting. If testing is desired for mucocutaneous lesions, fluid from an unroofed vesicle can be sent for viral culture. Polymerase chain reaction or direct fluorescent antibody testing may also be performed on swabbed tissue. A Tzanck test is generally not useful.

Temporal lobe lesions on CT scan or MRI are strongly suggestive of HSV encephalitis. CSF analysis shows a lymphocytic pleocytosis in most cases. CSF erythrocytosis is not a common finding. PCR testing of the CSF is the testing modality of choice for HSV meningoencephalitis, with 94% sensitivity and 98% specificity.

## **Emergency Department Care and Disposition**

1. Severe disease, including suspected or confirmed CNS infection or disseminated disease should be treated with IV **acyclovir** 10 to 15 milligrams/kilogram every 8 hours (based on ideal body weight). Immunocompromised patients with severe mucocutaneous involvement may be treated with IV acyclovir 5 milligrams/kilogram every 8 hours.
2. Immunocompetent adult patients with primary HSV infection can be treated orally with **acyclovir** 400 milligrams tid, **valacyclovir** 1000 milligrams PO every 12 hours or **famciclovir** 250 milligrams tid. Treatment is most effective if initiated within 48 to 72 hours of symptom onset and should be continued for 7 to 10 days.
3. Recurrent HSV may be treated orally with **acyclovir** 400 milligrams PO tid for 5 days, **valacyclovir** 2000 milligrams PO every 12 hours for 1 day (labialis) or 500 milligrams PO every 12 hours for 3 days (genital) or **famciclovir** 1500 milligrams PO 1 dose (orolabial) or 1000 milligrams PO 1 dose (genital). Less severe outbreaks may be treated with **topical acyclovir** 5% ointment applied 6 times a day for 7 days. Treatment should be started as soon as is possible after symptom onset.

## **VARICELLA AND HERPES ZOSTER**

Varicella-zoster virus (VZV) is the causative organism of both varicella (chickenpox) and herpes zoster (shingles). Herpes zoster is the reactivation of latent herpes which can occur once immune response against the virus wanes, either with advancing age or immunosuppression. The presence of herpes zoster in an otherwise young, healthy person may be a sign of HIV infection.

### **Clinical Features**

Varicella (chicken pox) is a febrile illness with a vesicular rash. The rash is superficial, concentrated more on the torso and face, and typically has lesions at varying stages, including papules, vesicles, and crusted lesions. Nonspecific symptoms of headache, malaise and loss of appetite are often present. CNS complications such as cerebellar ataxia, meningitis and

meningoencephalitis are well described. Infection of cerebral arteries resulting in vasculopathy and stroke may also occur. Pneumonitis can be severe and is more common in pregnant women.

Herpes zoster (shingles) causes lesions identical to those of chickenpox that occur in a unilateral, dermatomal distribution. A prodrome of malaise, headache, and photophobia may occur with localized pain, itching, and parasthesias preceding the outbreak of the rash. Although any dermatomal level may be affected, involvement of the trigeminal (face) or thoracic dermatomes are most common. Ocular involvement (herpes zoster ophthalmicus) may occur due to involvement of the ocular branch of the trigeminal nerve (see [Chapter 149](#) “Ocular Emergencies”). The course of the disease is approximately 2 weeks but may persist for a full month. Pain that continues beyond 30 days is termed postherpetic neuralgia (PHN), occurs more often with advancing age, and may last for months or years.

Rash involving more than three dermatomes or crossing the midline should raise the suspicion of disseminated disease, which can occur in immunocompromised patients. Although the skin may be the only involved structure, the virus may spread to the visceral organs and cause pneumonitis, hepatitis, and encephalitis.

## **Diagnosis and Differential**

Clinical diagnosis is sufficient in most cases. Laboratory diagnosis through viral culture, antigen testing, or PCR testing of vesicle fluid may be appropriate in patients with atypical illness or severe disease. Although smallpox has been eradicated, it remains a potential threat as a biologic weapon, and the lesions could be confused with those of varicella. The lesions of smallpox are larger, distributed more on the extremities, and all lesions are at the same stage of development. A chest radiograph should be obtained if pneumonitis is suspected. An MRI of the brain, lumbar puncture, and PCR testing for VZV are appropriate for suspected central neurologic involvement.

## **Emergency Department Care and Disposition**



1. Healthy children need only supportive care for chickenpox. **Acyclovir** 20 milligrams/kilogram (max 800 milligrams) PO qid for 5 days is appropriate for high-risk patients including children > 12 years of age, adults, those with chronic skin or pulmonary disorders, those receiving long-term salicylate therapy and immunocompromised patients. **Famciclovir** and **valacyclovir** are not licensed for the treatment of varicella in the United States.
2. To reduce the severity of PHN, start antiviral medication within 72 hours of the onset of rash, and consider treatment at > 72 hours if new vesicles are still present or developing. A 7 to 10 day course of **acyclovir** 800 milligrams PO 5 times a day, **valacyclovir** 1 gram PO tid or **famciclovir** 500 milligrams PO tid may be used. Herpes zoster may require narcotic analgesia.
3. Corticosteroids in combination with antivirals may provide a modest decrease in acute pain and should be considered in patients without contraindications to their use; however, they do not decrease the development of PHN.
4. Immunocompromised patients with chicken pox, shingles (regardless of the time since rash onset), visceral involvement or disseminated disease should be treated with **acyclovir** 10 milligrams/kilogram IV every 8 hours and admitted to the hospital for continued management.

## ARBOVIRAL INFECTIONS

Arboviral infections are spread by biting mosquitoes, ticks, and flies. *West Nile virus* and the viruses that cause *La Cross encephalitis*, *St. Louis encephalitis*, *eastern equine encephalitis* and *western equine encephalitis* are found in North America. Discussion of viral hemorrhagic fever due to dengue, yellow fever, and chikungunya can be found in [Chapter 98](#) “World Travelers.”

### Clinical Features

Most symptomatic arbovirus infections cause a nonspecific mild illness. Severe human arboviral diseases commonly manifest as 4 syndromes: fever and myalgia, arthritis and rash, encephalitis, and hemorrhagic fever. These syndromes can overlap. Headache is a common symptom of most arboviral infections and may be quite severe. Hemorrhagic fever presents with bleeding from the gums, petechiae, and GI tract. The classic presentation of viral encephalitis is fever, headache, and altered level of consciousness. Patients can be lethargic and confused, and occasionally present with seizures. In general, individuals at extremes of age are more likely to have severe disease.

## **Diagnosis and Differential**

Arboviral infection should be considered based on clinical presentation and knowledge of local epidemiologic patterns combined with suspicious travel or exposure history. Cerebrospinal fluid typically shows a lymphocytic pleocytosis and a slightly elevated protein level, although these findings are nonspecific. MRI may show foci of increased signal intensity in the brain parenchyma. Serologic testing (antibody detection) is the main method for confirmation of arboviral infections.

## **Emergency Department Care and Disposition**

Supportive and symptomatic therapy is the mainstay of management (see also [Chapter 98](#)). Specific antiviral drugs, interferon, and steroids are not useful. When CNS infection is suspected, empiric treatment with antibiotics and acyclovir is appropriate until bacterial meningitis and HSV encephalitis are ruled out. The decision whether or not to admit to the hospital should be based on severity of symptoms, clinical assessment and level of suspicion for other serious etiologies.

## **INFLUENZAE A AND B**

Influenza (flu) occurs worldwide with peak activity found in temperate climates between late December and early March. Influenza virus is transmitted by aerosolized or droplet transmission from the respiratory tract of infected persons, or by direct contact. After exposure the incubation period is usually about 2 days, with viral shedding (contagiousness) beginning about 24 hours before the onset of symptoms.

## **Clinical Features**

Classic flu symptoms are abrupt in onset and include fever of 38.6°C to 39.8°C (101°F to 103°F) with chills or rigor, headache, myalgia, and generalized malaise. Respiratory symptoms include dry cough, rhinorrhea, and sore throat, frequently with bilateral tender, enlarged cervical lymph nodes. The elderly usually do not have classic symptoms and may present with only fever, malaise, confusion, and nasal congestion. Almost 50% of affected children have gastrointestinal symptoms, but these are unusual in adults. The fever generally lasts 2 to 4 days, followed by recovery from most of the systemic symptoms within 3 to 7 days. Cough and malaise may persist for several weeks.

Common respiratory complications of acute influenza infection include primary influenza pneumonitis, secondary bacterial pneumonia, croup, and exacerbation of chronic obstructive pulmonary disease. The presence of dyspnea and hypoxia should raise the suspicion for pulmonary involvement. Aspirin therapy should not be used in patients with suspected or confirmed influenza due to the association with Reye syndrome. Other rare complications include Guillain-Barré syndrome, myocarditis, and pericarditis.

## **Diagnosis and Differential**

A clinical diagnosis of influenza during a known outbreak is highly accurate. Although rapid diagnostic tests are available for influenza, the performance characteristics of these tests vary and results should be

interpreted in the context of the clinical and epidemiologic information available. During influenza season, testing should be obtained in the following patients with acute febrile respiratory illness: immunocompetent outpatients at high risk for influenza complications and are within five days of symptom onset; and immunocompromised outpatients, regardless of time since illness onset. Hospitals may implement protocols to test patients admitted with respiratory disease. Once influenza has been documented in the community, influenza testing is no longer indicated for otherwise healthy outpatients with signs and symptoms consistent with influenza, especially during the peak of activity.

## **Emergency Department Care and Disposition**

1. Treatment is with **oseltamivir** 75 milligrams PO twice daily for 5 days (in children, 30 milligrams if 1 year or older and weight 15 kilograms or less, 45 milligrams for weight 16 to 23 kilograms, 60 milligrams for weight 24 to 40 kilograms; above 40 kilograms adult dose, give po twice daily for 5 days), or **zanamivir** 2 puffs twice daily for 5 days for 7 years old children to adult, take 2 doses on day 1 at least 2 hours apart. Zanamivir may cause bronchospasm and should be avoided in patients with underlying pulmonary disease.
2. **A mantadine** or **rimantadine** are alternatives for influenza A only.
3. When started within 48 hours of symptom onset, these medications can reduce the duration of uncomplicated influenza illness by approximately 1 day.
4. In general, treatment is recommended for any patient with confirmed or suspected influenza who is hospitalized; has severe, complicated or progressive illness; or is at higher risk for influenza complications.
5. Specific recommendations for use of individual drugs vary by susceptibility and resistance patterns. Up to date CDC guidelines can be accessed at their influenza website, [www.cdc.gov/flu](http://www.cdc.gov/flu).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 148, “Disseminated Viral Infections,” by Sukhjit S. Takhar and Gregory J. Moran.

# CHAPTER 92

## HIV Infections and AIDS

Scott Felten

Risk factors for HIV infection include unprotected sexual activity, injection drug use, blood transfusion before 1985, and maternal-neonatal transmission.

### CLINICAL FEATURES

Acute HIV infection, essentially indistinguishable from a “flu-like” illness, usually goes unrecognized but is reported to occur in 50% to 90% of patients. The time from exposure to onset of symptoms is usually 2 to 4 weeks, and the most common symptoms include fever (> 90%), fatigue (70% to 90%), sore throat (> 70%), rash (40% to 80%), headache (30% to 80%), and lymphadenopathy (40% to 70%). Other reported symptoms include myalgias, diarrhea, and weight loss. Seroconversion, reflecting detectable antibody response to HIV, usually occurs 3 to 8 weeks after infection. This is followed by a long period of asymptomatic infection except for possible persistent generalized lymphadenopathy. Early symptomatic infection is characterized by conditions that are more common and more severe in the presence of HIV infection but, by definition, are not AIDS indicator conditions. Examples include thrush, persistent vulvovaginal candidiasis, peripheral neuropathy, cervical dysplasia, recurrent herpes zoster infection, and idiopathic thrombocytopenic purpura. At this time CD4 counts are 200 to 500 cells/mm<sup>3</sup>. As the CD4 count drops below 200 cells/mm<sup>3</sup>, the frequency of opportunistic infections dramatically increases. AIDS is defined by the appearance of any indicator condition ([Table 92-1](#)) including a CD4 count lower than 200 cells/mm<sup>3</sup>. Late symptomatic or advanced HIV infection exists in patients with a CD4 count lower than 50 cells/mm<sup>3</sup> or clinical evidence of end-stage disease, including disseminated *Mycobacterium avium* complex or disseminated

cytomegalovirus (CMV). In today's era of highly active antiretroviral therapy (HAART), longevity is more dependent on age and other comorbidities than HIV status provided the patient adheres to HAART and the therapy is effective in suppressing viral load and maintaining normal CD4 counts.

CD4 count <200 cell/ $\mu$ L
Cervical cancer (invasive)
Cryptococcosis
Cryptosporidiosis
Cytomegalovirus retinitis
Esophageal candidiasis
Herpes simplex virus
Histoplasmosis, disseminated
HIV encephalopathy
HIV wasting syndrome
Isosporiasis
Kaposi sarcoma
Lymphoma (brain)
<i>Mycobacterium avium</i> complex
<i>Mycobacterium tuberculosis</i> disease
<i>Pneumocystis carinii</i> pneumonia
Progressive multifocal leukoencephalopathy
Recurrent bacterial pneumonia
<i>Salmonella</i> septicemia (recurrent)
Toxoplasmosis (brain)
Tuberculosis (pulmonary)

Key: HIV = human immunodeficiency virus.

**TABLE 92-1** Indicator Conditions for Acquired Immunodeficiency Syndrome

## Constitutional Symptoms and Febrile Illnesses

Systemic symptoms, such as fever, weight loss, and malaise, are common in HIV-infected patients and account for most HIV-related ED presentations. Appropriate laboratory investigation includes electrolytes, complete blood count, blood cultures, urinalysis and culture, liver function tests, chest radiographs, and in selected patients, serologic testing for syphilis, cryptococcosis, toxoplasmosis, CMV, and coccidioidomycosis. Lumbar puncture should be considered if there are neurologic signs or symptoms or unexplained fever.

In HIV patients without obvious focalizing signs or symptoms, sources of fever vary by stage of disease. Patients with CD4 counts higher than 500 cells/mm<sup>3</sup> generally have sources of fever similar to those in nonimmunocompromised patients. Those with CD4 counts between 200 and 500 cells/mm<sup>3</sup> are most likely to have early bacterial respiratory infections. For patients with CD4 counts lower than 200 cells/mm<sup>3</sup>, likely infections include *Pneumocystis pneumonia* (PCP actually *P jiroveci*), central line infection, *M. avium* complex (MAC), *Mycobacterium tuberculosis*, CMV, drug fever, and sinusitis. Disseminated MAC occurs predominately in patients with CD4 counts below 100 cells/mm<sup>3</sup>. Persistent fever and night sweats are typical. Associated symptoms include weight loss, diarrhea, malaise, and anorexia. Diagnosis is made with acid-fast stain of stool or other body fluids or culture. A more focal and invasive form of MAC has emerged, called *immune reconstitution illness to MAC*, which presents with lymphadenitis and follows weeks to months after starting HAART.

CMV is the most common cause of serious opportunistic viral disease in HIV-infected patients. Disseminated disease commonly involves the gastrointestinal or pulmonary system. The most important manifestation is retinitis.

Infective endocarditis is a concern especially in intravenous (IV) drug users (see [Chapter 93](#)).

Non-Hodgkin lymphoma is the most commonly occurring neoplasm in HIV patients and typically presents as a high-grade, rapidly growing mass lesion.

## **Pulmonary Complications**

Pulmonary presentations are among the most common reasons for ED visits by HIV-infected patients. The most common causes of pulmonary abnormalities in HIV-infected patients include community-acquired bacterial pneumonia, PCP, *Mycobacterium tuberculosis*, CMV, *Cryptococcus neoformans*, *Histoplasma capsulatum*, and neoplasms. Nonopportunistic bacterial pneumonias outnumber atypical infections

including *Streptococcus pneumoniae* (most common), *Haemophilus influenzae*, and *Staphylococcus aureus*. Productive cough, leukocytosis, and the presence of a focal infiltrate suggest bacterial pneumonia, especially in those with earlier-stage disease. Evaluation should include pulse oximetry, arterial blood gas analysis, sputum culture and Gram stain, acid-fast stain, blood cultures, and chest radiograph.

The classic presenting symptoms of PCP are fever, cough (typically nonproductive), and shortness of breath (progressing from being present only with exertion to being present at rest). Negative radiographs are reported in 15% to 20% of patients. Hypoxia or increased alveolar-arterial oxygen gradient identify patients at risk.

Classic pulmonary manifestations of tuberculosis (TB) include cough with hemoptysis, night sweats, prolonged fevers, weight loss, and anorexia. TB is common in patients with CD4 counts between 200 and 500 cells/mm<sup>3</sup>. Classic upper lobe involvement and cavitary lesions are less common, particularly among late-stage AIDS patients. False negative purified protein derivative TB test results are frequent among AIDS patients due to immuno-suppression. There is a high index of suspicion for TB in HIV patients with pulmonary symptoms due to high rates of person to person transmission.

Consider disseminated fungal infection in the severely immunosuppressed.

## **Neurologic Complications**

Central nervous system (CNS) disease occurs in 90% of patients with AIDS, and 10% to 20% of HIV-infected patients initially present with CNS symptoms. ED evaluation includes neuro exam, computed tomography, and lumbar puncture. Cerebrospinal fluid studies should include pressures; complete cell count; glucose; protein; Gram stain; India ink stain; bacterial, viral, and fungal cultures; toxoplasmosis; and *Cryptococcus* antigen and coccidioidomycosis titer.

Common causes of neurologic symptoms include AIDS dementia, *Toxoplasma gondii*, and *Cryptococcus neoformans*. Symptoms may include headache, focal neurologic deficits, altered mental status, or seizures. AIDS



dementia complex (also referred to as HIV encephalopathy or sub-acute encephalitis) is a progressive process commonly heralded by subtle impairment of recent memory and other cognitive deficits caused by direct HIV infection. Other, less common, CNS infections that should be considered in the presence of neurologic symptoms include bacterial meningitis, histoplasmosis (usually disseminated), CMV, progressive multifocal leukoencephalopathy, herpes simplex virus, neurosyphilis, and TB. HIV patients may experience HIV neuropathy characterized by painful sensory symptoms of the feet.

## **Gastrointestinal Complications**

The most frequent presenting symptoms include odynophagia, abdominal pain, bleeding, and diarrhea. ED evaluation includes stool for leukocytes, ova, parasites, acid-fast staining, and culture.

Diarrhea is the most frequent gastrointestinal complaint and is estimated to occur in 50% to 90% of AIDS patients. Common causes include bacterial organisms, such as *Shigella*, *Salmonella*, enteroadherent *Escherichia coli*, and *Campylobacter*; parasitic organisms, viruses, fungi, and antiviral therapy. Oral candidiasis, or thrush, affects more than 80% of AIDS patients. The tongue and buccal mucosa are commonly involved, and the plaques characteristically can be easily scraped from an erythematous base. Esophageal involvement may occur with *Candida*, herpes simplex, and CMV. Complaints of odynophagia or dysphagia are usually indicative of esophagitis and may be extremely debilitating.

Hepatomegaly occurs in approximately 50% of AIDS patients. Elevation of alkaline phosphatase levels is frequently seen. Jaundice is rare. Coinfection with hepatitis B and hepatitis C is common, especially among IV drug users.

Anorectal disease is common in AIDS patients. Proctitis is characterized by painful defecation, rectal discharge, and tenesmus. Common causative organisms include *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, syphilis, and herpes simplex.

## **Cutaneous Manifestations**

Generalized conditions such as xerosis, seborrheic eczema, and pruritus are common. Kaposi sarcoma appears more often in homosexual men than in other risk groups. Clinically, it consists of painless, raised brown-black or purple papules, and nodules that do not blanch. Common sites are the face, chest, genitals, and oral cavity. Reactivation of varicella-zoster virus is more common in patients with HIV infection and AIDS than in the general population. Herpes simplex virus infections are common. HIV patients may develop bullous impetigo and *Pseudomonas* associated chronic ulcerations. MRSA infection, scabies, human papillomavirus, hypersensitivity reactions to medications are common.

## **Ophthalmologic Manifestations**

Seventy-five percent of patients with AIDS develop ocular complications. CMV retinitis is the most frequent and serious ocular opportunistic infection and the leading cause of blindness in AIDS patients. The presentation of CMV retinitis is variable. It may be asymptomatic early on but later causes changes in visual acuity, visual field cuts, photophobia, scotoma, or eye redness or pain. Herpes zoster ophthalmicus is another diagnosis to consider and is recognized by the typical zoster rash in the distribution of cranial nerve VI.

## **DIAGNOSIS AND DIFFERENTIAL**

Benefits of early HIV diagnosis include early and aggressive antiretroviral therapy, which can lead to immune reconstitution; prevention of viral mutation and drug resistance; slowing of disease progression and improved long-term prognosis.

The most common assay used to detect viral antibody is a screening enzyme-linked immunoassay (ELISA) followed by a confirming western blot test on ELISA-positive specimens. ELISA is approximately 99% specific and 98.5% sensitive; the western blot test is nearly 100% sensitive and specific if performed under ideal laboratory circumstances. Diagnosis

of acute-stage HIV infection is not possible with standard serologic tests because seroconversion has not yet occurred. Methods for earlier detection of HIV-1 include techniques to detect DNA, RNA, or HIV antigens. The single-use diagnostic system is used to screen rapidly for antibodies to HIV-1 in serum or plasma. Rapid tests are available in many ED's nationwide but must be confirmed with Western Blot testing.

Knowledge of current or recent CD4 counts and a HIV viremia load will help in the management of HIV patients. CD4 counts below 200 cells/mm<sup>3</sup> and viral load greater than 50,000 copies/mL is associated with increased risk of progression to AIDS-defining illness. When these levels are unavailable, a total lymphocyte count of <1200 cells/mm<sup>3</sup> combined with clinical symptoms is strongly predictive of a positive CD4 count of <200 cells/mm<sup>3</sup>.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. The initial evaluation of HIV-infected and AIDS patients begins with a heightened awareness of the need for universal precautions. Respiratory isolation should be instituted for patients with suspected TB.
2. All unstable patients should have airway management as indicated, oxygen, pulse oximetry, cardiac monitoring, and IV access.
3. Seizures, altered mental status, gastrointestinal bleeding, and coma should be managed with standard protocols.
4. Suspected bacterial sepsis and focal bacterial infections should be treated with standard antibiotics (see [Chapter 89](#)).
5. Systemic *M avium* should be treated with **clarithromycin** 500 milligrams PO twice daily plus **ethambutol** 15 milligrams/kilogram PO plus **rifabutin** 300 milligrams PO daily. Treatment of immune reconstitution illness to MAC should include continuation of highly active antiretroviral therapy, antimicrobials as above, and possibly steroids.
6. Systemic CMV should be treated with **ganciclovir** 5 milligrams/kilogram IV every 12 hours or **foscarnet** 90 milligrams/kilogram IV every 12 hours.

7. Ophthalmologic CMV is treated with a ganciclovir implant plus **ganciclovir** 1.0 to 1.5 grams PO 3 times daily or 5 milligrams/kilogram IV twice daily for 14 to 21 days.
8. Pulmonary PCP should be treated with **trimethoprim-sulfamethoxazole** (TMP-SMX), with TMP 15 to 20 milligrams/kilogram/d IV or PO divided 3 times daily, for 3 weeks. The typical oral dose is 2 tablets of TMP-SMX double strength 3 times daily. An alternative is **pentamidine** 4 milligrams/kilogram/d IV or IM for 3 weeks. Oral steroids should be given if hypoxic: **prednisone** 40 milligrams twice daily for 5 days, then 40 milligrams daily for 5 days, and then 20 milligrams daily for 11 more days.
9. Pulmonary TB may be treated with **INH** 5 milligrams/kilogram/d PO plus **rifabutin** 10 milligrams/kilogram/d PO or rifampin 10 milligrams/kilogram/d PO plus **pyrazinamide** 15 to 30 milligrams/kilogram/d PO plus **ethambutol** 15 to 20 milligrams/kilogram/dose PO daily.
10. CNS toxoplasmosis can be treated with **pyrimethamine** 200 milligrams initially then 50 to 75 milligrams/d PO plus **sulfadiazine** 4 to 6 grams/d PO plus **folinic acid** 10 milligrams/d PO **for 6 to 8 weeks**. Plus/minus Leucovorin 10 to 25 milligrams daily.
11. CNS cryptococcosis can be treated with **amphotericin B** 0.7 milligrams/ kilogram/d IV and **flucytosine** 25 milligrams/kilogram IV 4 times daily for 2 weeks. When improved, **fluconazole** 400 milligrams PO daily for 8 to 10 weeks can be used.
12. Candidiasis (thrush) can be treated with **clotrimazole** 10 milligram troches 5 times per day or **nystatin** 500,000 units/mL gargle with 5 mL 5 times per day.
13. Esophagitis can be treated with **fluconazole** 100 to 400 milligrams daily PO.
14. Salmonellosis can be treated with **ciprofloxacin** 500 milligrams PO twice daily for 2 to 4 weeks.
15. Cutaneous herpes simplex can be treated with **acyclovir** 200 milligrams PO 5 times daily for 7 days or **famciclovir** 125 milligrams PO twice daily for 7 days or **valacyclovir** 1 gram PO BID for 7 days or **acyclovir** 5 to 10 milligrams/kilogram IV every 8 hours for 7 days for severe illness.
16. Cutaneous herpes zoster can be treated with **acyclovir** 800 milligrams PO 5 times daily, or **valacyclovir** 1 gram PO 3 times daily, or

- famciclovir** 500 milligrams PO 3 times daily.
17. Herpes zoster ophthalmicus should be treated with **acyclovir** 800 milligrams PO 5 times daily for 7 to 10 days.
  18. *Candida* or *Trichophyton* should be treated with topical **clotrimazole** or **miconazole** or **ketoconazole** 3 times daily for 3 weeks.
  19. Although rarely started in the ED, antiretroviral therapy is started for CD4 counts below 350 cells/mm<sup>3</sup> or history of AIDS Defining Illness (see [Table 92-1](#)) or pregnancy, HIV associated neuropathy, and Hepatitis B confection regardless of the CD4 count. New protocols recommending treatment upon initial HIV positive testing may be forthcoming, but are not currently available. Initial treatment includes 2 nucleoside reverse transcriptase inhibitors plus 1 or 2 protease inhibitors or 1 non-nucleoside reverse transcriptase inhibitor drug. See the Centers for Disease Control and Prevention website: <http://www.cdc.gov/hiv/>.
  20. The decision to admit an AIDS patient should be based on severity of illness, with attention to new presentation of fever of unknown origin, hypoxia worse than baseline or Pao<sub>2</sub> below 60 mm Hg, suspected PCP, suspected TB, new CNS symptoms, intractable diarrhea, suspected CMV retinitis, herpes zoster ophthalmicus, or a patient unable to perform self-care.
  21. Post exposure prophylaxis should be initiated as quickly as possible, preferably within 1 to 2 hours. Risks for seroconversion include (1) deep injury, (2) visible blood on the injuring device, (3) needle placement in a vein or an artery of the source patient, and (4) a source patient with late-stage HIV infection. Treatment regimes vary by type of exposure. CDC guidelines recommend 2 general alternatives: a basic regimen, which consists of 2-drug therapy, often consisting of azidothymidine and lamivudine, and an expanded regimen, which adds a third drug, such as indinavir or nelfinavir.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 149, "Human Immunodeficiency Virus Infection and Acquired Immunodeficiency Syndrome," by Richard E. Rothman, Catherine A. Marco, and Samuel Yang.

# CHAPTER 93

## Infective Endocarditis

John C. Nalagan

Infective endocarditis (IE) is the result of infection and damage to the endocardium of the heart due to either a cardiac structural abnormality or risk factors such as injection drug use, indwelling catheters, poor dental hygiene, or infection with HIV. IE most commonly involves the mitral valve and has 3 main classifications. Native valve endocarditis is the most common form (59% to 70%) and most often affects patients with mitral valve prolapse, bicuspid aortic valve, calcific aortic stenosis, or rheumatic heart disease. *Streptococcus viridians*, *Staphylococcus aureus*, and enterococcus are most commonly involved with mortality rates from 16% to 27%. Endocarditis involving injection drugs is the second classification and has an estimated incidence of 2% to 5%. There is a predilection for the tricuspid valve and is associated with high recurrence and mortality rates, particularly in HIV patients. *S aureus* is the main pathogen. Prosthetic valve endocarditis affects 1% to 4% of valve recipients within a year of surgery. It is divided into early (<60 days post op) and late (>60 days). Early disease is usually nosocomial involving *Staphylococcus epidermis* and almost twice the mortality, whereas late is typically community acquired.

### CLINICAL FEATURES

IE presents along a continuum from acute to insidious and indolent. Symptoms are consistent with bacteremia (blood cultures are negative in approximately 5%). Fever is the most common manifestation followed by chills, weakness, and dyspnea. Cardiac manifestation such as heart murmurs are present in up to 85% of cases, although this can be difficult to hear in the ED. Dyspnea is common and often due to acute or progressive CHF, which occurs in approximately 70% of patients. Embolic phenomenon occurs about 50% of the time and is due to the embolization of

friable vegetation fragments. Embolic stroke in the distribution of the MCA is the most common CNS complication followed by subarachnoid hemorrhage due to mycotic aneurysm. Other symptoms due to embolic phenomenon are chest and abdominal pain, flank pain with hematuria, and acute limb ischemia. Cutaneous findings occur in 18% to 50% of patients and include petichiae, splinter/subungual hemorrhages, Osler nodes (tender subcutaneous nodules on finger/toe pads), and Janeway lesions (hemorrhagic plaques on the palms or soles).

## **DIAGNOSIS AND DIFFERENTIAL**

Suspicion of IE requires hospital admission. Patients at risk for IE are those with unexplained fever and risk factors for the disease, such as injection drug users, patients with prosthetic valves, and those with new or changing murmurs and evidence of arterial emboli.

The necessary components for diagnosis are blood cultures, echocardiogram, and clinical observation. Blood cultures should be drawn prior to administration of antibiotics and from 3 separate sites, with an hour elapsing between the first and last set of cultures. Echocardiography should be performed as soon as possible. Transthoracic two-dimensional echocardiography is typically the initial choice. Although nonspecific for IE, laboratory abnormalities that support the diagnosis are anemia, hematuria, and elevations in C-reactive protein, erythrocyte sedimentation rate, and procalcitonin.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Patients may present with respiratory compromise and require emergent airway stabilization. Dyspnea is often due to decreased cardiac output, diminished lung capacity, altered mental status, or acidosis. Pulmonary edema may be due to left-sided valvular rupture and requires afterload reduction.

2. Intraaortic balloon counterpulsation is indicated for mitral valve rupture but contraindicated for aortic valve rupture.
3. Patients with native valve endocarditis do not require anticoagulation, whereas patients with prosthetic valves already on anticoagulation should be maintained with their current regimen unless requested otherwise by the consultant.
4. Antibiotics should be initiated in patients with suspected endocarditis after appropriate cultures are obtained. [Table 93-1](#) lists empiric treatment regimens. Definitive therapy is based on culture and sensitivity results and typically requires 4 to 6 weeks of antibiotics.

Patient Characteristics	Recommended Agent	Initial Dose
Uncomplicated history	Ceftriaxone	1 to 2 grams IV
	or Nafcillin	2 grams IV
	or Vancomycin	15 milligrams/kilogram IV
	plus Gentamicin	1 to 3 milligrams/kilogram IV
Injection drug use, congenital heart disease, hospital-acquired, suspected MRSA, or on oral antibiotics	Nafcillin	2 grams IV
	plus Gentamicin	1 to 3 milligrams/kilogram IV
	plus Vancomycin	15 milligrams/kilogram IV
Prosthetic heart valve	Vancomycin	15 milligrams/kilogram IV
	plus Gentamicin	1 to 3 milligrams/kilogram IV
	Plus Rifampin	300 milligrams PO

\*Because of controversy in the literature regarding the optimal regimen for empiric treatment, antibiotic selection should be based on patient characteristics, local resistance patterns, and current authoritative recommendations.

**TABLE 93-1** Empiric Therapy of Suspected Bacterial Endocarditis\*

5. Antibiotic prophylaxis against endocarditis has changed dramatically. Prophylactic antibiotics before procedures should be administered for patients only with the highest risk factors: those with a prior history of infective endocarditis; those with a prosthetic heart valve or some component of a prosthetic valve device; unrepaired congenital heart disease; failed repair; or a cardiac transplant recipient with valve regurgitation due to a structurally abnormal valve. The only ED procedures where prophylactic antibiotics should be considered are procedures on known infected skin, such as abscess drainage. Agents



suggested are **dicloxacillin** 2 grams PO, **cephalexin** 2 grams PO, **clindamycin** 600 milligrams IM or IV, or **vancomycin** 1 gram IV, 30 to 60 min before procedure.

6. Lastly, antibiotic prophylaxis is *not* indicated for common emergency department procedures such as local injections, laceration repair, IV placement/ blood draws, endotracheal intubation, and urethral catheterization.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 150, “Infective Endocarditis,” by Richard E. Rothman, Samuel Yang, and Catherine A. Marco.

# CHAPTER 94

## Tetanus and Rabies

Vincent Nacouzi

### TETANUS

Tetanus is an acute, frequently fatal spasmodic disease resulting from a wound infected with the organism *Clostridium tetani*. The disease is exotoxin mediated. In the United States, 69% of Americans > 70 years old not have adequate immunity. The incidence has increased in the 20 to 59 years old.

#### Clinical Features

As a result of an injury, *C. tetani* and its spores are introduced into the wound. Devitalized tissue, if any, favors toxin formation. The incubation varies from 24 hours to more than 30 days. The spores can germinate and release the toxin. Most injuries are unrecognized, and vary from puncture wounds, surgical procedures, abortions, or in neonates because of inadequate umbilical cord care. Clinically, tetanus is categorized into 4 forms: local, generalized, cephalic, and neonatal.

Local tetanus presents with rigidity of muscles in proximity to the injury site and usually resolves without sequelae. Generalized tetanus is the most common and presents with pain and stiffness in the jaw. Later, the rigidity leads to the development of trismus and the characteristic facial expression, *risus sardonicus*, “sarcastic grimace.” Violent spasms and tonic contractions of muscle groups are responsible for the symptoms of the disease including dysphagia, opisthotonos, flexing of the arms, fist clenching, and extension of the lower extremities. Importantly, mentation remains normal, unless laryngospasm and chest rigidity cause respiratory compromise.

In the second week of the illness, a hypersympathetic state develops and manifests as tachycardia, hypertension, sweating, and hyperpyrexia, this difficult to manage hyperactivity contributes to the morbidity and mortality.

*Cephalic tetanus* follows injuries to the head and neck, can result in dysfunction of cranial nerves, most often the seventh nerve, it has a poor prognosis.

*Neonatal tetanus* carries an extremely high mortality rate and results from inadequate maternal immunization and poor umbilical cord care.

## **Diagnosis and Differential**

Tetanus is diagnosed clinically. Prior immunization does not eliminate tetanus as a diagnostic possibility. There are not any confirmatory laboratory tests. The differential diagnosis includes strychnine poisoning, dystonic reactions to phenothiazine, hypocalcemic tetany, rabies, peritonsillar abscess, peritonitis, meningitis, SAH and TMJ disease.

## **Emergency Department Care and Disposition**

Patients with tetanus are best managed in an intensive care unit due to the potential for respiratory compromise. Environmental stimuli must be minimized to prevent precipitation of convulsive spasms. Identification and debridement of the inciting wound and devitalized tissue, after immune globulin administration, is necessary to minimize further toxin production.

1. **Tetanus immune globulin** 3000 to 6000 units IM in a single injection should be given, in the opposite site of the toxoid administration. It should be given before any wound debridement because more exotoxin may be released during wound manipulation.
2. **Tetanus toxoid** (DTap or Td depending on age), 0.5 mL IM at presentation, and 6 weeks and 6 months after presentation (see [Chapter 16](#)).
3. Antibiotics are of questionable value in the treatment of tetanus. If warranted, parenteral **metronidazole** 500 milligrams IV every 6 hours is

the antibiotic of choice. Penicillin is contraindicated because it may potentiate the effects of tetanospasmin.

4. **Midazolam**, 0.05 to 0.15 milligram/kilogram/h IV given as a continuous drip to effect. Often, large quantities of sedatives are needed and, the Glycol vehicle found in diazepam and lorazepam may cause a metabolic acidosis. Midazolam lacks the Glycol, making it the drug of choice. **Lorazepam** 2 milligrams IV to effect, may be used in small quantities.
5. Neuromuscular blockade may be required to control ventilation and muscular spasm and to prevent fractures and rhabdomyolysis. In such cases, **vecuronium** initial bolus of 0.1 milligram/kilogram IV followed infusion at 1 microgram/kilogram/min is the agent of choice. Sedation during neuromuscular blockade is mandatory.
6. The combined  $\alpha$  and  $\beta$ -adrenergic blocking agent, **labetalol** 0.25 to 1 milligram/min continuous IV infusion (0.3 to 1 milligram/kilogram/h in children), has been used to treat the manifestation of sympathetic hyperactivity. **Magnesium sulfate** 70 milligrams/kilogram loading, then 1 to 4 grams/h IV has been advocated as a treatment for this condition. **Morphine sulfate** 0.5 to 1 milligram/kilogram/h is also useful and provides sympathetic control without compromising cardiac output. **Clonidine** 300 micrograms every 8 hours nasogastrically, an  $\alpha$ -receptor agonist, may be helpful in the management of cardiovascular instability.
7. Patients who recover from clinical tetanus *must* undergo active immunization (see [Chapter 16](#) for immunization schedule).

## RABIES

Rabies, a form of encephalitis, is commonly fatal and is transmitted by a viral inoculation from infected animal saliva. Most emergency physicians never encounter clinical rabies, yet must decide on the need for post exposure prophylaxis on a regular basis.

In the United States, dog and cat bites, and exposure to bats are the most common reason for postexposure prophylaxis. The most important source of active rabies is wildlife transmission. Animal bites contracted outside the United States should be considered at high risk for rabies transmission.

High risk rabid wildlife species include bats, skunks, raccoons, cows, dogs, foxes, and cats. Rodents (squirrels, chipmunks, rats, mice, etc) and lagomorphs (rabbits, hares, hamsters etc.) are at very low risk for rabies transmission. Most rabid animals are agitated and cause unprovoked attacks. In the United States, one-third cases are diagnosed postmortem since, the exposure is not often reported.

Human rabies has decreased in the United States. In 60% of the cases in the 1980s, a source of infection was not identified. After inoculation, the virus remains in the wound vicinity for up to 90 days, then ascends to the CNS and spinal cord to replicate. The Incubation averages 35 to 64 days; periods as short as 12 days or as long as 700 days have been reported.

## **Clinical Features**

The initial symptoms of human rabies are nonspecific and last 1 to 4 days: fever, malaise, headache, anorexia, nausea, sore throat, cough, and pain, or paresthesia at the bite site (80%). Subsequently, central nervous system involvement becomes apparent with restlessness and agitation, altered mental status, painful bulbar and peripheral muscular spasms, opisthotonos, and bulbar or focal motor paresis. Alternatively, in 20%, an ascending, symmetric, flaccid, and areflexic paralysis, comparable to the Landry-Guillain-Barré syndrome, may be seen. Hypersensitivity to sensory stimuli and hydrophobia may occur at this stage, with the latter resulting from the sight, sound, swallowing, or even mention of water. Progressively lucid and confused intervals may become interspersed, cholinergic abnormalities may manifest (hyperpyrexia, mydriasis, and increased lacrimation and salivation), and brainstem dysfunction (dysphagia, optic neuritis, and facial palsies) with hyperreflexia may occur. Extensor plantar responses may be positive. Common complications include adult respiratory distress syndrome, diabetes insipidus, syndrome of inappropriate secretion of antidiuretic hormone, hypovolemia, electrolyte abnormalities, pneumonia, and cardiogenic shock with hypotension and dysrhythmia from rabies myocarditis. Coma, convulsions, and apnea are the final manifestations of rabid death.

## **Diagnosis and Differential**

The diagnosis of rabies in the emergency department is clinical. A final diagnosis is made by postmortem analysis of brain tissue. Cerebrospinal fluid and serum antibody titers should be sent to a laboratory skilled in rabies antibody analysis. Elevated cerebrospinal fluid protein and a mononuclear pleocytosis are also seen.

The differential diagnosis includes viral or other infectious encephalitis, polio, tetanus, viral encephalitis, meningitis, CVA, brain abscess, septic cavernous sinus thrombosis, cholinergic poisoning, and the Landry-Guillain-Barré syndrome. The diagnosis is especially difficult without history of exposure but should be considered for patients with a picture of progressive and unexplained encephalitis.

## **Emergency Department Care and Disposition**

The treatment of rabies exposure consists of assessment of risk of rabies, public health and animal control notification, and, if warranted, the administration of specific immunobiological products to protect against rabies.

Remember to call public health officials for advice and guidance as needed.

1. Debridement of devitalized tissue, if any, is important in reducing the viral inoculum. Wounds of special concern should not be sutured because this promotes rabies virus replication.
2. Tetanus should always be considered and primary or reimmunization prophylaxis should be administered (see [Chapter 16](#)).
3. In cases of bites by bats, skunks, raccoons, foxes, and most other carnivores, administer Human Rabies Immune Globulin and start the rabies vaccine series (see below).
4. Bats are a special case. It is currently recommended to consider post-exposure prophylaxis for persons who were in the same room as a bat and who might be unaware that a bite or direct contact had occurred (asleep or

intoxicated). If the bat can be caught and tested by local/state animal control, prophylaxis can be stopped if tests are negative.

5. If the bite was due to domestic dogs, cats, or ferrets with normal behavior the animal should be quarantined for 10 days, which is sufficient for the disease to manifest if the animal is infected. If no signs become apparent, the animal can be considered nonrabid.
6. In cases of livestock, small rodents, lagomorphs (rabbits and hares) large rodents (wood chucks and beavers) and other mammals, consultation should be immediately obtained from local health department officials. If any cases of rabies have been reported or suspected in these animals locally, administer postexposure prophylaxis. If the animal can be captured, it should be sacrificed and tested. In many of these cases, postexposure prophylaxis is initiated and stopped if testing is negative.
7. **Human rabies immune globulin (HRIG)** is administered only once at the outset of therapy or even up to the seventh day. The dose is 20 IU/kg, with half of the dose or more (based on tissue volume constraints) infiltrated locally at the exposure site and the remainder administered intramuscularly. Do not administer the HRIG and vaccine in the same anatomic site.
8. **Human diploid cell vaccine (HDCV)** and **Purified chick embryo cell culture vaccine (PCEV)**, both are for active immunization. The HDCV or PCEV can be administered intramuscularly in 41 mL doses on days 0, 3, 7, and 14 (the formerly recommended day 28 dose has been dropped). Serum sickness reactions have not been reported with PCEV.
9. In adults and children, HRIG and HDCV or PCEV should be administered in the deltoid muscle to avoid failure.
10. State or local officials should be consulted regarding the possibility of rabies in local animal populations before decisions on initiating rabies prophylaxis are made. This action may not be possible before the first treatment but may affect subsequent treatments. Animal bites should be reported to the local animal control unit or police department so that the animal can be quarantined for observation.
11. The Centers for Disease Control and state or county health departments can provide assistance in the management of complications. The most current information available on the rabies home page is produced and

updated regularly by the Centers for Disease Control at [www.cdc.gov/rabies/](http://www.cdc.gov/rabies/).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 151, “Tetanus,” by Joel L. Moll and Donna L. Carden; and Chapter 152, “Rabies,” by David J. Weber, David A. Wohl, and William A. Rutala.



## CHAPTER 95

### Malaria

David M. Cline

Malaria must be considered in any person who has traveled to the tropics and presents with an unexplained febrile illness. Five species of the protozoan *Plasmodium* infect humans: *P falciparum*, *P vivax*, *P ovale*, *P malariae*, and *P knowlesi*. The organism is transmitted by the anopheline mosquito bite and travels hematogenously first to the liver, where asexual reproduction occurs (exoerythrocytic stage). The liver cell ruptures, releasing merozoites that invade erythrocytes, multiply, and cause hemolysis (erythrocytic stage). Malaria also may be transmitted by blood transfusion or passed transplacentally from mother to fetus.

Malaria transmission occurs in large areas of Central and South America, the Caribbean, sub-Saharan Africa, the Indian subcontinent, Southeast Asia, the Middle East, and Oceania (New Guinea, Solomon Islands, etc). More than 50% of all the cases of malaria in the United States, including most cases due to *P falciparum*, arise from travel to sub-Saharan Africa. Resistance of *P falciparum* to chloroquine and other drugs continues to spread (see [Table 95-1](#)). Strains of *P vivax* with chloroquine resistance have been identified. The Centers for Disease Control and Prevention (CDC) has a malaria hotline: 770-488-7788 M-F, 8 am to 4:30 pm EST, and 770-488-7100 after hours, weekends and holidays. Alternatively, the CDC website can be accessed at <http://www.cdc.gov/malaria/> for information on resistance patterns in various countries and information on malaria prophylaxis and treatment. When in doubt, chloroquine resistance for initial treatment should be assumed.

Geographic Region	Areas With Malaria	Countries With Chloroquine-Resistant <i>Plasmodium falciparum</i>
Central America	All countries	Areas east of the Panama Canal
Caribbean	Dominican Republic and Haiti	None
South America		
Temperate	Argentina	None
Tropical	Most countries	All countries except Paraguay
East Asia	China	China
Eastern South Asia	All countries except Brunei and Singapore	All infected areas
Middle South Asia	All countries	All countries
Western South Asia and Middle East	Iraq, Oman, Saudi Arabia, Syria, Turkey, and United Arab Emirates	All countries except Syria and Turkey
Northern Africa	All countries except Tunisia	None
Sub-Saharan Africa	All countries except Reunion and Seychelles	Widespread
Southern Africa	All countries except Lesotho and Saint Helena	Widespread
Oceania	Limited to Papua New Guinea, Solomon Islands, and Vanuatu (small foci elsewhere)	Widespread

**TABLE 95-1** Geographic Distribution of Malaria, Including Resistant Strains

## CLINICAL FEATURES

The incubation period ranges from 1 to 4 weeks. Partial chemoprophylaxis or incomplete immunity can prolong the incubation period to months or even years. Initially malaria manifests with nonspecific fever and malaise, then progresses to chills and high grade fevers; frequent symptoms include headache, myalgia, arthralgia, cough, abdominal pain, nausea, and diarrhea. The patient may have tachycardia, orthostatic dizziness, and extreme weakness. Classically, cycles of fever and chills followed by profuse diaphoresis and exhaustion occur at regular intervals, reflecting hemolysis of infected erythrocytes.

Physical examination findings are typically nonspecific. During a febrile paroxysm, most patients appear acutely ill, with high fever, tachycardia, and tachypnea. Splenomegaly is common. In *P falciparum* infections, hepatomegaly, edema, and icterus often occur. Laboratory features include normocytic normochromic anemia with evidence of hemolysis and thrombocytopenia. The white blood cell count is normal or low.

Complications of malaria can occur rapidly, particularly with *P falciparum*. All forms cause hemolysis and splenomegaly, and splenic rupture may occur. Hypersplenism with subsequent pancytopenia may be seen in advanced cases. Glomerulonephritis, most often in *P malariae* infections, and nephrotic syndrome may occur. Cerebral malaria, characterized by somnolence, coma, delirium, and seizures, has a mortality rate greater than 20%. Other life-threatening complications associated with *P falciparum* include noncardiogenic pulmonary edema and metabolic abnormalities, including lactic acidosis and profound hypoglycemia. Blackwater fever is a severe complication seen almost exclusively in *P falciparum* infections, with massive intravascular hemolysis, jaundice, hemoglobinuria (dark urine), and acute renal failure.

## DIAGNOSIS AND DIFFERENTIAL

The definitive diagnosis is established by identification of the parasite on Giemsa-stained thin and thick smears of peripheral blood. In early infection,

especially with *P falciparum*, parasitemia may be undetectable initially due to intraorgan sequestration. Parasite load in the peripheral circulation fluctuates over time and is highest during an acute rising fever with chills. Therapy should *not* be withheld if malaria is suspected, even though the parasite is not detected on initial blood smears. If plasmodia are not visualized, repeated smears should be taken at least twice daily (preferably during febrile episodes) for 3 days to fully exclude malaria. Once plasmodia are identified, the smear is also evaluated for the degree of parasitemia (percentage of red blood cells infected—which correlates with prognosis), and species type (in particular *P falciparum*) is present. Antigen-detecting rapid diagnostic tests are available in certain areas, and are recommended by the World Health Organization (WHO) provided quality control measures are in place. Further, WHO recommends parasite-based diagnosis except for young children in areas of high transmission when testing availability is limited; in these situations, clinically based diagnoses and treatment are recommended, with monitoring for clinical improvement.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Unless it is certain that a patient could *not* have a chloroquine-resistant case, based on history of geographic exposure, the infection must be assumed to be resistant and treated with one of the chloroquine-resistant regimens listed immediately below.
2. Patients with uncomplicated infection with chloroquine-resistant *P falciparum* can be treated one of several regimens. *Option 1* is **artemether/lumefantrine**, dose twice daily for 3 days, a total of 6 doses. For adults, 20 milligrams/120 milligrams tablets, 4 tablets initially, 4 tablets in 8 hours, then 4 tablets every 12 hours for 2 days. For children, 5 to 15 kilograms, 1 tablet initially, 1 tablet in 8 hours, then 1 tablet every 12 hours for 2 days; 15 to 25 kilograms, 2 tablets initially, 2 tablets in 8 hours, then 2 tablets every 12 hours for 2 days; 25 to 35 kilograms, 3 tablets initially, 3 tablets in 8 hours, then 3 tablets every 12 hours for 2 days; > 35 kilograms, follow adult dosing. *Option 2* is **atovaquone-**

**proguanil.** For adults, give 4 tablets adult strength (250 milligrams/100 milligrams) daily for 3 days. For children > 41 kilograms, give adult dose; 31 to 40 kilograms, 3 adult tablets for 3 days; 21 to 30 kilograms, 2 adult tablets for 3 days; 11 to 20 kilograms, 1 adult tablet for 3 days; 9 to 10 kilograms, 3 pediatric tablets for 3 days; 5 to 8 kilograms, 2 pediatric tablets for 3 days. *Option 3* is **quinine sulfate** 542 milligrams base (= 650 milligrams salt) PO 3 times daily (10 milligrams salt/kilogram/dose maximum) for 3 to 7 days plus doxycycline 100 milligrams PO twice daily for 7 days. Options 1 and 2 are preferred for children. A final option is mefloquine plus doxycycline, but mefloquine has an increased frequency of neuropsychiatric reactions, making it the least favored choice.

3. If *P falciparum* can be excluded (travelers returning from Central America west of the Panama Canal, Haiti, the Dominican Republic, and most of the Middle East), patients with adequate home care and oral hydration can be treated as outpatients with close follow-up, including repeated blood smears to assess treatment response. Recommended treatment for uncomplicated malaria infection due to *P vivax*, *P ovale*, and *P malariae*, and *P knowlesi* is chloroquine plus primaquine phosphate. **For adults: chloroquine** 600 milligrams base (=1 gram salt), then 300 milligrams base (= 500 milligrams salt) in 6 hours, then 300 milligrams base per day for 2 days (total dose 1550 milligrams base); plus **primaquine phosphate** 30 milligrams base per day for 14 days on completion of chloroquine therapy. **For children: chloroquine** 10 milligrams/kilograms base to maximum of 600 milligrams load, then 5 milligrams/ kilogram base in 6 hours and 5 milligrams/kilogram base per day for 2 days, *plus primaquine phosphate* 0.5 milligram/kilogram base for 14 days on completion of chloroquine therapy.
4. Chloroquine has no effect on the exoerythrocytic forms of *P vivax* and *P ovale*, which remain dormant in the liver. Unless treated with primaquine, relapse will occur. Primaquine should be avoided in patients with glucose-6-phosphate dehydrogenase deficiency because of hemolysis.
5. Patients with significant hemolysis or with comorbid conditions that can be aggravated by high fevers or hemolysis are best hospitalized, as are infants and pregnant women. *Plasmodium falciparum* infections are best managed in the hospital, as are patients with more than 3% parasitemia.

6. Patients with complications due to *P falciparum* or with high parasitemia but unable to tolerate oral medication should receive intravenous treatment.
7. For severe malaria, with chloroquine resistant *P falciparum*, there are 2 recommended treatments. *Option 1* is **quinidine**, 6.25 milligrams base (= 10 milligrams salt)/kilogram IV load over 2 hours (maximum, 600 milligrams), then 0.0125 milligram base (= 0.02 milligram salt)/kilogram/min continuous infusion until patient is stabilized and able to tolerate PO therapy (see above). Parenteral quinidine and quinine can cause severe hypoglycemia. They are also myocardial depressants and are contraindicated in patients with heart disease. Cardiac monitoring is required during administration. *Option 2* is **artesunate**, which is available from the CDC quarantine station, follow artesunate with atovaquone-proguanil plus doxycycline as above. The dose of artesunate is 2.4 milligrams/kilogram IV at 0 hour, 12 hours, and 24 hours, and then 2.4 milligrams/kilogram once daily for total of 3 days.
8. Aggressive supportive care should be provided to all hospitalized ill patients, including judicious fluid replacement, correction of metabolic derangements, and advanced support (dialysis, mechanical ventilation, etc).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 153, "Malaria," by John J Szela, Josiah J. Tayali, and Jeffrey D. Band.

## CHAPTER 96

# Foodborne and Waterborne Diseases

David M. Cline

Foodborne disease is an illness that occurs in 2 or more people after the consumption of common food source. Contamination can come from bacteria, viruses, or protozoans. Viruses are the most common source, including Norwalk-type (58% overall, United States), astrovirus, rotaviruses, and enteric adenoviruses. Bacterial sources include nontyphoidal *Salmonella* (11% overall, most common cause for hospitalization and associated death in the United States), *Clostridium perfringens*, *Campylobacter* spp, *Listeria monocytogenes*, *Shigella* spp, Shiga toxin producing *Escherichia coli* (STEC) and *Staphylococcus aureus*. Parasitic causes include *Giardia lamblia*, *Toxoplasma gondii*, *Entamoeba histolytica*, and *Cryptosporidium*. The most common associated foods are poultry, leafy vegetables, and fruits/ nuts. In addition, after eating reef fish that feed on certain dinoflagellates (algae), patients may experience scombroid or ciguatera poisoning which is a toxin induced syndrome.

Waterborne diseases occur from ingestion of, or contact with contaminated water, from swimming pools, hot tubs, spas, or naturally occurring fresh or salt water. Symptoms are can be either GI or dermatologic. Common organisms include the majority of those associated with foodborne illness plus *Vibrio* species, *Aeromonas* species, *Pseudomonas aeruginosa*, *Yersinia* species, Hepatitis A, nontuberculous *Mycobacterium* and less frequent organisms.

## CLINICAL FEATURES

Symptoms of both foodborne and waterborne illness include vomiting, diarrhea, abdominal cramping, fever, dehydration, malaise, and in some, bloody stool. Physical exam may be remarkable for features of dehydration, and in some, stool positive for frank or occult blood. Prolonged illness

beyond 2 weeks suggests protozoan parasites. STEC may be complicated by hemolytic uremic syndrome (decreased urine output, symptoms of anemia), especially after antibiotic treatment. STEC classically presents with vomiting, moderate to marked stomach cramps, diarrhea (often bloody) and mild fever, not over 101°F/38.5°C.

Patients with scombroid fish poisoning or ciguatera poisoning have symptoms similar to foodborne illness described immediately above, 1 to 24 hours after ingestion of reef fish. In addition, patients with scombroid poisoning frequently have flushing and headache due to histamine reaction. Those with ciguatera poisoning may have headaches, muscle aches, paresthesias, or a burning sensation on contact with cold, due to sodium channel mediated nerve depolarizations. Neurologic symptoms may be prolonged beyond the ED visit.

The skin manifestations of waterborne illness vary from simple cellulitis, the painful indurated plaque of *Mycobacterium marinum*, to necrotizing infections which may include hemorrhagic bulla with *Vibrio vulnificus*. Patients with *Aeromonas hydrophila* skin infections often have a history of trauma associated with freshwater exposure, and may have foul smelling wounds.

## **DIAGNOSIS AND DIFFERENTIAL**

Bedside testing for fecal occult blood is the most commonly indicated test; otherwise most patients need no laboratory testing, unless significantly dehydrated or other significant diagnoses are being considered. For those more acutely ill, consider fecal leukocytes, the neutrophil marker lactoferrin, electrolytes, and complete blood count. Stool gram stain may reveal *Campylobacter*. Stool cultures are more likely to be positive in those with positive fecal leukocytes or lactoferrin. STEC and *Vibrio* cultures require specific procedures (check local laboratory guidelines). Reserve ova and parasite testing for those patients with chronic symptoms, immunocompromise, or patients with a confirmed source of parasite. Other considerations include Rotavirus antigen testing in children from daycare settings, daycare workers, or older adults. *Clostridium difficile* antigen



testing may be indicated in those with prolonged symptoms, recent antibiotic use, significant comorbidities, or extremes of age.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

Most cases are self-limited and improve with nonspecific treatment.

1. Initiate oral rehydration fluids initially if tolerated. Intravenous rehydration with normal saline will benefit those significantly dehydrated, or those with continued vomiting.
2. Antiemetics, such as **metoclopramide** 10 milligrams PO or IV, or **ondansetron** 4 milligrams PO or IV may facilitate oral rehydration. Antihistamines, such as **diphenhydramine** 25 milligrams PO or IV, may improve the symptoms of scombroid fish poisoning.
3. **Loperamide** 4 milligrams initially, then 2 milligrams after every unformed stool up to a maximum of 16 milligrams/d is indicated in mild to moderate, nonbloody diarrhea in adults without fever (do not use in patients with STEC).
4. Antibiotics are favored only in those patients with an increasing number of the following features: significant abdominal pain, bloody diarrhea, fever over 101°F/38.5°C, symptom duration > 48 hours, impaired host, positive fecal leukocytes or lactoferrin, however, **antibiotics are contraindicated in patients with STEC**. When treatment is indicated, recommended agents include **ciprofloxacin** 500 milligrams PO twice per day, or **trimethoprim-sulfamethoxazole** double-strength twice daily, for 3 to 5 days. Organism specific antibiotic recommendations can be found in the parent text cited at the end of this chapter.
5. *Vibrio vulnificus* skin infections are treated with **doxycycline** 100 milligrams IV or PO twice daily, plus **ceftazidime** 2 grams IV every 8 hours. *Aeromonas* skin infections are treated with **ciprofloxacin** 500 milligrams twice daily (mild cases) or with **piperacillin-tazobactam** 3.375 grams IV every 6 hours in severe cases. Necrotizing infections require emergent surgical debridement.

6. Most patients can be treated as outpatients, admission is indicated in those appearing toxic, those in whom vomiting cannot be controlled, the immunocompromised, or those at the extremes of age with significant symptoms.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 154, “Foodborne and Waterborne Diseases,” by Patrick L. McGauly and Simon A. Mahler.

# CHAPTER 97

## Zoonotic Infections

Christopher R. Tainter

Zoonotic infections are a class of over 200 specific diseases and infections that are naturally transmitted between vertebrate animals and humans. Transmission may occur via direct contact with an infected animal or animal product, by ingestion of contaminated water or food products, by inhalation, or through arthropod vectors. Pets, farm animals, and common wildlife are the primary reservoirs. Arthropods, in particular ticks, are the primary vectors. These diseases may be caused by myriad organisms including bacteria (eg, *Rickettsia*, *Borrelia*), viruses, and parasites. The high morbidity and mortality rates often associated with these illnesses mandate their careful consideration in patients who present with fever, chills, myalgias, rash, and other nonspecific symptoms. In such cases, specific risk factors for zoonotic infection should be sought: exposure to animals; residence or recent travel in rural areas or underdeveloped countries; history of tick bites or exposure to tick habitat; dressing, skinning, or handling animal skins or raw flesh; animal bites or scratches; or ingestion of animal or dairy products. Worldwide, ticks are the most common vector of disease transmission. Unfortunately, many patients with tick-acquired infections do not recall a history of tick bite. Hence, clinical suspicion should remain high for patients in endemic areas. West Nile Virus infections are discussed in [Chapter 91](#) “Disseminated Viral Infections.”

## ROCKY MOUNTAIN SPOTTED FEVER

Rocky Mountain spotted fever (RMSF) is the most severe tick-borne disease in the United States. It is caused by *Rickettsia rickettsii*, a pleomorphic obligate intracellular coccobacillus. The primary vector for transmission is the *Dermacentor* tick; the usual animal hosts are deer, rodents, horses, cattle, cats, and dogs. Most cases occur between April and

September. More than half of reported cases of RMSF occur in the south to mid-Atlantic states, but cases have been reported in the majority of the continental United States.

## **Clinical Features**

RMSF affects multiple organ systems, and its nonspecific initial presentation renders diagnosis difficult. The classic clinical triad of fever, rash, and history of tick bite is unreliable. Only 50% of infected patients can recall a tick bite and rash is absent in 20% of cases. Initial findings commonly include fever, headache, myalgias, and malaise. Additionally, patients may experience lymphadenopathy, petechiae/purpura, pulmonary infiltrates, jaundice, hepatosplenomegaly, abdominal pain, nausea, vomiting, diarrhea, meningitis/encephalitis, renal failure, and myocarditis.

Patients with RMSF often seek medical attention before onset of the rash, which is seen 2 to 4 days after initial fever. Initially, the rash is maculopapular. It typically begins on the hands, feet, wrists, and ankles (and may involve palms or soles). The rash spreads centripetally up the trunk, usually sparing the face. Infection can also result in a pulmonary capillary vasculitis and associated bronchiolitis; secondary bacterial pneumonia is common.

## **Diagnosis and Differential**

The diagnosis is largely clinical, with confirmation coming after treatment is initiated. In the absence of an alternative explanation, a febrile patient with a headache who was exposed to known tick habitat with high uncut grass, weeds, and low brush is at significant risk for a tick bite, even without seeing a tick on his/her body. Laboratory abnormalities may include neutropenia, thrombocytopenia, elevated liver function tests, and hyponatremia. RMSF can be confirmed, but not in the ED setting, with a rise in antibody titer between acute and convalescent sera, or via skin biopsy of the rash with immunofluorescence antibody testing.

The differential diagnosis for RMSF includes viral illnesses, pneumonia, meningococemia, ehrlichiosis, toxic shock syndrome, scarlet fever, and leptospirosis.

## **Emergency Department Care and Disposition**

1. Recommended treatment for adults is **doxycycline** 100 milligrams po/IV twice daily. **Chloramphenicol** 50 to 75 milligrams/kilogram/d IV in 4 divided doses is an alternative, but is associated with more serious side effects. Treatment should continue for 5 to 10 days, or until 3 days after resolution of fever.
2. Antibiotic therapy for children weighing <45 kilograms is **doxycycline** 2.2 milligrams/kilogram PO twice daily. Doxycycline has been used for short courses in children without significant staining of the teeth, and is recommended as therapy by both the American Academy of Pediatrics and Centers for Disease Control and Prevention as the treatment of choice for all rickettsial diseases for all ages. Alternatives include **chloramphenicol** 50 to 100 milligrams/kilogram/d (3 grams maximum) IV in 4 divided doses.

## **LYME DISEASE**

With approximately 25,000 cases reported annually, Lyme disease remains the most common vector-borne zoonotic infection in the United States. The responsible organism is the spirochete *Borrelia burgdorferi* and the vector is the *Ixodes* deer tick (also known as the *black-legged tick*). In highly endemic areas, the transmission rate from a deer tick bite is about 3%, although **there is almost no risk if duration of attachment is <72 hours**. Cases have been reported in all 48 continental states, but most cases occur in the northeastern, upper mid-Atlantic, and northcentral states. Peak transmission occurs during the summer months.

### **Clinical Features**

Lyme disease is a multiorgan infection that is typically divided into 3 distinct stages. However, not all patients progress through all stages, and stages may either overlap or be separated by periods of remission. The *primary stage* is often characterized by erythema chronicum migrans (ECM), an erythematous plaque with central clearing. ECM forms at the site of the tick bite, usually 2 to 20 days postbite. ECM, which results from local vasculitis, is the most common manifestation of Lyme disease, occurring in 60% to 80% of cases. Untreated, ECM lesions tend to persist 3 to 4 weeks before resolving spontaneously; they may recur in Lyme's secondary stage.

The *secondary stage* corresponds to dissemination of the spirochete within a few days to 6 months after initial infection. Secondary stage Lyme disease is characterized by multiple secondary annular red skin lesions (ECM), fever, adenopathy, neuropathies, cardiac abnormalities, arthritic problems, and multiple annular dermatologic lesions (which occur in up to 50% of infected patients). In the secondary stage, approximately 15% of untreated patients develop neurologic symptoms. These can include headache, neck stiffness, difficulty with mentation, cerebellar ataxia, myelitis, encephalitis, motor or sensory radiculoneuritis, mononeuritis multiplex, and, most commonly, cranial neuritis (usually a **unilateral or bilateral facial nerve palsy**). Oligoarticular arthritis, another complication of the secondary stage, usually affects the large joints (especially the knees); episodes are characteristically separated by months of remission. In 8% of patients, there are cardiac manifestations such as myopericarditis or any type of atrioventricular (AV) block.

The *tertiary stage* occurs years after initial infection. It can be characterized by chronic arthritis, myocarditis, sub-acute encephalopathy, axonal polyneuropathy, and leukoencephalopathy. Symptoms can persist for a decade or more.

## **Diagnosis and Differential**

Diagnosis initially must rely on clinical features. Confirmation may be obtained via polymerase chain reaction (PCR) testing, polyvalent

fluorescence immunoassay, or western immunoblot testing. *B burgdorferi* is difficult to culture. Differential diagnosis depends on clinical manifestation of the disease stage, and may include cellulitis, erythema multiforme, tinea corporis, viral/bacterial meningitis/encephalitis, rheumatic fever, septic arthritis, endocarditis, and other inflammatory/autoimmune and viral syndromes.

## **Emergency Department Care and Disposition**

1. Antimicrobial agents may include : **doxycycline** 100 milligrams PO twice daily, **amoxicillin** 500 milligrams PO 3 times daily, **cefuroxime** 500 milligrams PO twice daily, **erythromycin** 500 milligrams PO 4 times daily, or **ceftriaxone** 1000 milligrams IV daily. Therapy in the primary or early secondary stages should continue for 14 to 21 days. More advanced secondary stage requires treatment for 28 days, and treatment of tertiary stage Lyme disease requires intravenous ceftriaxone or penicillin for 28 to 60 days.
2. First-line treatment for children is **amoxicillin** (20 to 50 milligrams/kilogram/d in 3 divided doses).
3. **A single dose of doxycycline 200 milligrams given within 72 hours of the deer tick bite can prevent Lyme disease**, but this is not routinely suggested due to the low transmission rate and the possibility that prophylactic antibiotic administration may depress the immune response to the disease. Prophylactic antibiotics may be considered in treatment of deer tick bites in areas where Lyme disease is highly endemic, if the nymphal deer tick is at least partially engorged with blood, and if the deer tick has been attached for > 72 hours.

## **EHRlichiosis**

Ehrlichiosis (also called *human monocytic ehrlichiosis*) is a group of zoonotic diseases caused by infection from the *Ehrlichia* genus. These are small, gram-negative pleomorphic coccobacilli that infect circulating leukocytes. The lone star tick, *Amblyomma americanum*, serves as the

primary vector, while the white-tailed deer is the major animal reservoir in the southeastern United States.

## **Clinical Features**

Symptoms usually develop 10 to 14 days after a tick bite. Characteristic clinical features are consistent with a nonspecific febrile illness, and may include fever, malaise, headache, nausea, vomiting, diarrhea, abdominal pain, and arthralgias. A minority of patients progress to serious complications that may include renal failure, respiratory failure, and encephalitis.

## **Diagnosis and Differential**

Laboratory findings may include leukocytopenia, thrombocytopenia, and liver dysfunction. Diagnosis initially must be made on clinical grounds but may be confirmed with an increase in antibody titer between acute and convalescent phases of the illness. The differential diagnosis includes cholecystitis/cholangitis, Lyme disease, babesiosis, malaria, meningitis, RMSF, and typhoid.

## **Emergency Department Care and Disposition**

1. Treatment for adults consists of **doxycycline** 100 milligrams PO or IV twice daily for 7 to 14 days.
2. For children less than 45 kilograms, the dose of **doxycycline** is 2.2 milligrams/kilogram PO twice daily for 14 days.

## **COLORADO TICK FEVER**

Colorado tick fever is an acute viral illness caused by an RNA virus of the *Coltivirus* genus, in the family Reoviridae. *Dermacentor andersoni*, the



wood tick, is the primary vector, and the zoonotic reservoirs are deer, marmots, and porcupines. Only about 300 cases are reported annually. Most cases occur in the western mountainous regions of the United States, above 4000 ft.

## **Clinical Features**

Symptoms begin 3 to 7 days after a tick bite and include fever, chills, headache, myalgias, and photophobia; there may be a macular or petechial rash.

## **Diagnosis and Differential**

Diagnosis is made by clinical features and geography. The differential diagnosis includes other tick-borne illnesses and meningitis.

## **Emergency Department Care and Disposition**

No specific therapy exists, and supportive care usually is sufficient.

# **TULAREMIA**

Tularemia is caused by *Francisella tularensis*, a small, gram-negative, nonmotile coccobacillus. The zoonotic vectors are ticks of the *Dermacentor* and *Amblyomma* genera, and the principal animal reservoirs include rabbits, hares, deer, and dogs. Cases occur throughout the United States (except Hawaii) and Canada. Methods of transmission include tick bites or direct inoculation of broken skin or mucosa from an infected host.

## **Clinical Features**

Clinical features at presentation depend on the route of inoculation. The most common *ulceroglandular* form is characterized by a papule at the site of a tick bite, with painful regional adenopathy. *Glandular* tularemia consists of tender regional adenopathy without a skin lesion. The *typhoidal* form is associated with fever, chills, headache, and abdominal pain. *Ocularoropharyngeal* and *pneumonic* forms result from airborne deposition into the eyes and lungs.

## Diagnosis and Differential

Laboratory findings are nonspecific in all forms of tularemia. Diagnosis can be determined by culture and enzyme-linked immunosorbent assay (ELISA). The multiple clinical variations of tularemia lead to a broad differential diagnosis that should include pyogenic bacterial infection, syphilis, anthrax, plague, Q fever, psittacosis, typhoid, brucellosis, and rickettsial infection.

## Emergency Department Care and Disposition

1. Preferred therapy for adults includes **streptomycin** 10 milligrams/kilogram IM every 12 hours (max. daily dose 2 grams) or **gentamicin** 5 milligrams/kilogram IV or IM divided every 8 hours for 7 to 10 days.
2. The pediatric dose of **streptomycin** is 15 milligrams/kilogram IM every 12 hours (maximum daily dose 2 grams), or **gentamicin** 2.5 milligrams/kilogram IM or IV 2 or 3 times daily for 7 to 10 days.
3. Alternatives include **chloramphenicol** 15 to 25 milligrams/kilogram IV 4 times per day for 14 to 21 days (15 milligrams/kilogram for pediatric, maximum daily dose 4 grams), **doxycycline** 100 milligrams PO twice daily, for 14 days (1 to 2 milligrams/kilogram for pediatric, do not use under age 8, maximum daily dose 200 milligrams), or **ciprofloxacin** 500 to 750 milligrams PO twice daily for 14 days (10 to 15 milligrams/kilogram for pediatric, maximum daily dose 1.5 grams).

# ANTHRAX

Anthrax is an acute bacterial infection caused by *Bacillus anthracis*, an aerobic gram-positive rod that forms central oval spores. Human infection can result from inhalation of spores, inoculation of broken skin, bites from arthropods (fleas), or ingestion of inadequately cooked infected meat.

## Clinical Features

Inhaled or pulmonic anthrax usually results from handling unsterilized, imported animal hides, or imported raw wool. It results in a mediastinitis, rather than in true pneumonia, and is almost always fatal. Initial presentation consists of flulike symptoms, which progress over 3 to 4 days to include marked mediastinal and hilar edema, and respiratory failure. Cutaneous anthrax (wool sorter disease) accounts for 95% of all anthrax infections. It begins with a pruritic macule at the inoculation site (most commonly fingers), which progresses to an ulcerative site with multiple infectious serosanguinous vesicles containing the anthrax bacillus. Gram stain or culture of the vesicular fluid is often diagnostic. The ulcer eventually progresses to a painless black eschar and falls off within 2 weeks. A small minority of untreated patients develop rapidly fatal bacteremia.

## Diagnosis and Differential

Gram stain, direct fluorescent antibody stain, or culture of skin lesions or fluid from vesicle may establish the diagnosis. Blood cultures may also be positive. The differential diagnosis is influenced by exposure and risk factors. For inhalational anthrax, it may include influenza, tuberculosis, and other causes of mediastinitis (bacterial, viral, parasitic, sarcoidosis). With cutaneous anthrax, warfarin necrosis, calciphylaxis, ischemic necrosis, tularemia, plague, spider/ insect bite, mycobacterial infection, ecthyma gangrenosum, and aspergillosis/ mucormycosis should be considered.

## Emergency Department Care and Disposition

1. Aggressive antibiotic therapy is required for inhalational anthrax consisting of **ciprofloxacin** 400 milligrams IV every 12 hours, or **doxycycline** 100 milligrams IV twice per day *plus* **clindamycin** or **rifampin**. Extended treatment for adults is ciprofloxacin 500 milligrams PO every 12 hours or doxycycline 100 milligrams PO every 12 hours. A course of treatment is 60 days.
2. Treatment of cutaneous anthrax consists of **ciprofloxacin** 500 milligrams PO twice per day or **doxycycline** 100 milligrams PO twice per day for 60 days.
3. A vaccine is available for high-risk populations (military personnel and laboratory technicians).
4. Prophylaxis for exposed individuals can be done with either **ciprofloxacin** 500 milligrams PO twice per day or **doxycycline** 100 milligrams PO twice per day for 60 days in combination with a 3-dose vaccination course.

## PLAGUE

Plague (*Yersinia pestis*) is a gram-negative aerobic bacillus of the Enterobacteriaceae family, endemic to the United States. It is found most often in rock squirrels and ground rodents of the southwest but also may be carried by cats or dogs. The rodent flea serves as the primary vector. Transmission to humans occurs through the bite of a flea from an infected animal host.

### Clinical Features

Frequently, an eschar develops at the initial bite wound. This is followed by the development of a painful, sometimes suppurative bubo (enlarged regional lymph node), often in the groin. Sepsis and pneumonia may ensue,

due to hematologic spread of the bacteria. Bubonic plague may present with fever, headache, and buboes, while the pneumonic form is usually associated with cough, chills, dyspnea, and shock. The pneumonic form is highly contagious and can be transmitted from person to person via aerosolized respiratory secretions. It is rapidly fatal if not treated aggressively.

## **Diagnosis and Differential**

Diagnosis must be made on clinical findings in a patient with possible contact with a vector or animal host. Blood culture or culture of suspected sites may reveal organisms, but treatment should be initiated in suspected cases without awaiting these results. The differential diagnosis includes lymphogranuloma venereum, syphilis, staphylococcal or streptococcal lymphadenitis, other causes of pneumonia, or tularemia.

## **Emergency Department Care and Disposition**

1. Recommended antimicrobials are **gentamicin** 5 milligrams/kilogram IV or IM daily for 10 days, or **streptomycin** 15 milligrams/kilogram or 1 gram IV or IM every 12 hours for 10 days.
2. Pediatric dosing of **gentamicin** is 2 to 2.5 milligrams/kilogram/dose IV or IM every 8 hours.
3. Alternatives include **doxycycline** 100 milligrams IV twice daily or 200 milligrams IV daily for 10 days, **ciprofloxacin** 400 milligrams IV twice daily for 10 days, or **chloramphenicol** 25 milligrams/kilogram IV 4 times daily for 10 days.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 155, “Zoonotic Infections” by John T. Meredith.

# CHAPTER 98

## World Travelers

**David M. Cline**

Fever and other symptoms of infection are the most common complaints for returning travelers (see [Table 98-1](#)). The evaluation of infectious disease in the returning traveler requires an understanding of the geographical distribution of infections (see [Table 98-2](#)), risk factors, incubation periods, clinical manifestations, and appropriate laboratory investigations. See Centers for Disease Control and Prevention (CDC) website for further information: <http://wwwnc.cdc.gov/travel/destinations/list.aspx>. Traveler's diarrhea, enteroviral infections, gastroenteritis, giardiasis, salmonellosis, shigellosis are discussed in [Chapter 96](#) "Foodborne and Waterborne Diseases" and in [Chapter 37](#) "Diseases Presenting Primarily with Diarrhea"; malaria is discussed in [Chapter 95](#) "Malaria"; upper respiratory infection and pertussis are discussed in [Chapter 30](#) "Pneumonia, Bronchitis, and Upper Respiratory Infections"; STDs are discussed in [Chapter 87](#) "Sexually Transmitted Diseases"; hepatitis A and B are discussed in [Chapter 48](#) "Hepatic Disorders, and Hepatic Failure"; HIV (human immunodeficiency virus). Infections and acquired immune deficiency syndrome are discussed in [Chapter 92](#); anthrax and plague are discussed in [Chapter 97](#) "Zoonotic Diseases." This chapter covers the most common infectious disease presentations in returning travelers; the reader is referred to the source material, cited at the end of the chapter, for further information, and discussion of less common diseases.

Risk (Frequency)	Diseases/Syndromes (# references to other manual chapters)
High risk (1 in 10)	Traveler's diarrhea (96), upper respiratory illness (30)
Moderate risk (1 in 200)	Chikungunya, dengue fever, enteroviral infection (96), gastroenteritis (96), giardiasis, hepatitis A (48), malaria (95), salmonellosis (96), sexually transmitted diseases (87), shigellosis.
Low risk (1 in 1000)	Amebiasis, ascariasis (roundworm), enterobiasis (pinworm), hepatitis B (48), scabies (155), tuberculosis (31), typhoid/paratyphoid
Very low risk (1 in > 1000)	Anthrax (97), brucellosis, Chagas disease, cysticercosis, hemorrhagic fevers (including yellow fever), Human immunodeficiency virus (92), hookworm, leishmaniasis, leptospirosis, pertussis (30), plague (97), schistosomiasis/Katayama syndrome, typhus/rickettsial disease, trypanosomiasis

**TABLE 98-1** Traveler Risk of Exposure to Infectious Agents

Africa	Malaria, human immunodeficiency virus, TB, hookworm, tapeworm, roundworm, brucellosis, yellow fever (and other hemorrhagic fevers), relapsing fever, schistosomiasis, tick typhus
Central and South America	Malaria, relapsing fever, dengue fever, filariasis, TB, schistosomiasis, Chagas disease, louse-borne typhus
Mexico, Caribbean	Dengue fever, hookworm, malaria, cysticercosis, amebiasis, louse-borne typhus
Australia	Dengue fever
Middle East	Malaria, hookworm, anthrax, brucellosis
China and East Asia	Dengue fever, hookworm, malaria, strongyloidiasis, hemorrhagic fever, scrub typhus

**TABLE 98-2** Common Regional Tropical Illness

## CLINICAL FEATURES

The incubation period for disease is most commonly longer than a traveler's foreign stay, and therefore travelers commonly become febrile/symptomatic upon return. Travel history should include query concerning visits to game parks, farms, caves, health facilities, consumption of exotic foods, activities involving fresh or salt water exposure, insect exposure, sexual activities,

epidemics in the area visited, contact with ill people, as well as pretrip immunizations and prophylactic antibiotics taken. A history chronological disease presentation should be taken including height, quality, and duration of fever and chills. Examination findings such as current temperature, rash, eschar, hepatosplenomegaly, lymphadenopathy, jaundice, and other skin findings should be documented.

## **DIAGNOSIS AND DIFFERENTIAL**

General laboratory assessment includes malaria smear (and dipstick antigen test if available) should be done for all febrile travelers returning from locations with endemic malaria. Complete blood count: look for lymphopenia (dengue, HIV and typhoid) or eosinophilia (parasites, fungal disease) and thrombocytopenia (malaria, dengue, acute HIV, typhoid). Urinalysis may show proteinuria and hematuria in cases of leptospirosis. Blood cultures should be obtained prior to antibiotics. Liver function tests are indicated if patient is jaundiced. Consider specific testing for diseases suspected by symptoms and risk of exposure. Obtain a chest radiograph for respiratory symptoms, and consider a liver ultrasound if amebic liver abscess is suspected.

## **ASSESSMENT, EMERGENCY DEPARTMENT CARE AND DISPOSITION FOR SPECIFIC DISEASES**

Malaria is the most important disease to rule out in returning travelers (discussed in [Chapter 95](#) “Malaria”). The other most common diseases in returning travelers (see also references at the beginning of this chapter) are discussed later in this chapter.

### **Dengue Fever**



Dengue fever is spread by the day-biting *Aedes aegypti* mosquito. Incubation is 4 to 7 days. Symptoms of classic dengue are high fever, headache, nausea, vomiting, myalgias, and rash (late), lasting several days. Dengue fever acquired in Southeast Asia typically is accompanied by hemorrhagic symptoms and often shock; in this form abdominal pain may be marked. Diagnosis is by PCR (1 to 8 days postsymptom onset) or IgM ELISA, after 4 days of symptoms. Daily blood counts are recommended. Outpatient treatment is recommended in mild cases, with oral hydration as tolerated and close follow-up for blood work. Avoid aspirin and NSAIDs. Inpatient treatment for supportive care is recommended if there is a drop in hematocrit or platelets, or hemorrhagic symptoms, or abnormal vital signs.

## **Chikungunya**

Chikungunya is the second most common arbovirus infection in returning travelers, after dengue fever. Also spread by day-biting mosquitos, chikungunya presents very much like classic dengue fever but additionally with generalized arthralgia. From 5% to 30% of patients with go on to have chronic arthropathy. Diagnosis is by PCR (1 to 4 days postsymptom onset) or IgM, after 5 days of symptoms. Treatment is supportive; chloroquine may reduce long term arthralgias but is not standard therapy. NSAIDs are helpful, but should be avoided until dengue fever has been ruled out.

## **Typhoid Fever**

Typhoid fever, or enteric fever, is caused by *Salmonella typhi* and *Salmonella paratyphi*. Transmission is from contaminated food or water, after contact with the infected urine or feces of symptomatic individuals, or asymptomatic carriers. After malaria is ruled out (by lack of potential exposure or by testing), typhoid fever is the most common febrile disease lasting more than 10 days in returning travelers. Incubation is 1 to 3 weeks. Symptoms include fever with headache initially, then high fever with chills, headache, cough, abdominal distention, myalgias, constipation (most common, but some have diarrhea), and prostration. A classic presentation is

bradycardia relative to the height of fever, but is often absent. After several days, a pale red macular rash (“rose spots”) appears on the trunk. Complications include small bowel ulceration, anemia, disseminated intravascular coagulopathy (DIC), pneumonia, meningitis, myocarditis, and renal failure. Remarkable lab findings may include leukopenia and elevated liver enzymes, however not typical. Diagnosis is clinical, confirmation is by stool culture. After initiation of supportive care with fluids and fever control, treatment is **ceftriaxone**, 2 grams IV. IM for 14 days, or **ciprofloxacin** 500 to 750 milligrams PO twice daily for 14 days. For severe typhoid fever complicated by delirium, coma, shock, or DIC, administer **dexamethasone**, 3 milligrams/ kilogram IV load. Blood transfusion may be required in severe cases.

## **Brucellosis**

Brucellosis is caused by the bacteria *Brucella*, most commonly following contact with cattle, goats, camels, dogs, pigs, or after ingestion of unpasteurized milk or cheese. Symptoms include fever, abdominal pain, back pain, fatigue, headache, joint pain, and loss of appetite. Common history is relapsing fever, but can be chronic low grade fever. Examination findings include lymphadenopathy, hepatomegaly, splenomegaly, and may include septic arthritis. Diagnosis is by blood culture, or serology. Consult infectious disease for treatment with **doxycycline**, **rifampicin**, and an aminoglycoside, **streptomycin** or **gentamicin** for 2 weeks.

## **Rickettsial Spotted Fevers including Scrub Typhus**

Rickettsial spotted fevers are transmitted by the bite, body fluid, or feces of ixodid arthropod ticks. Mortality without treatment approaches 25%. Scrub typhus (*Rickettsia orientalis*) and African tick typhus (*R. conorii*) are the most common forms in travelers returning from the Southeast Asia and Africa respectively. Incubation is 3 to 14 days. Symptoms are fever, malaise, myalgias, severe headache, rash (may be absent), nausea, and vomiting followed by lymphadenopathy and splenomegaly. The skin lesion

in scrub typhus starts as a papule at the bite site, which becomes necrotic and forms a crusted black eschar. African scrub typhus is in general, less severe. Diagnosis is clinical; serologic tests confirm the diagnosis after empiric treatment with **doxycycline** 100 milligrams twice daily for 7 to 10 days; chloramphenicol is an alternative.

## **Typhus Epidemic Louse-Borne Typhus**

Epidemic louse-borne typhus, common in Mexico, Guatemala, Ethiopia, and the Himalayas, is caused by *R prowazekii* and should not be confused with the disease caused by *S typhi*. Incubation is 8 to 12 days. Patients may or may not be aware of the louse. Symptoms include high fevers, severe headache, and a maculopapular rash between 4 and 7 days. Diagnosis is clinical; serologic tests confirm the diagnosis after empiric treatment with **doxycycline** 100 milligrams twice daily for 7 to 10 days; chloramphenicol is an alternative.

## **Leptospirosis (Weil Disease)**

Leptospirosis occurs after fresh water exposure to *Leptospira interrogans* or after exposure to infected dogs. Incubation is 2 to 20 days. Symptoms include high fever, severe headache, chills, myalgias, hepatitis with or without jaundice, and conjunctival injection without purulent discharge. Diagnosis requires serology. Mild disease (within 3 days of symptoms) is treated with **amoxicillin** 500 milligrams 3 times daily, or **doxycycline** 100 milligrams twice daily. More severe cases should be treated with **penicillin G**, 5 million units every 6 hours IV, or **ceftriaxone** 1 gram IV/IM daily. Treatment duration is 7 to 14 days.

## **Crimean-Congo Hemorrhagic Fever**

Crimean-congo hemorrhagic fever is a tick-borne viral disease that is rising in frequency in Africa, Asia, eastern Europe and the Middle East.

Agricultural workers are at the greatest risk, but it can be acquired from contact with the blood of patients. Symptoms include sudden onset of fever, headache, myalgia, dizziness, and possibly mental confusion. The hemorrhagic period is short (2 to 3 days), starts the third to fifth day of illness, and may manifest with epistaxis, hemoptysis, GI bleeding, vaginal bleeding, or hematuria. Patients may have thrombocytopenia, elevated liver enzymes and creatinine; prothrombin time and activated partial thromboplastin time may be prolonged. Diagnosis is clinical with confirmation by serology. Treatment is supportive, and may require transfusions and or respiratory support. **Ribavirin** is used in moderate to severe cases, 30 milligrams/kilogram load, then 15 milligrams/kilogram every 6 hours for 4 days, then 7.5 milligrams/ kilogram for 6 days.

## **Yellow Fever**

Yellow fever is caused by a flavivirus, transmitted by a day-biting mosquito, occurring along a broad equatorial belt in South and Central America and Africa. Symptoms range from a mild flulike illness to hemorrhagic fever with 20% mortality. After an incubation period of 3 to 6 days, typical early symptoms include fever, headache, myalgias, conjunctival injection, abdominal pain, prostration, facial flushing, and relative bradycardia, subsequently the classic jaundice, black emesis, and albuminuria is found. Symptoms may progress to shock, multiorgan failure, and bleeding diathesis. Treatment is supportive including transfusion as needed.

## **Cysticercosis**

Cysticercosis is the systemic illness caused by dissemination of the larval form of the pork tapeworm. Humans become infected by ingesting the contaminated food (undercooked pork), or inadvertent contact with contaminated soil. Involvement of almost any tissue can occur. CNS infection is known as neurocysticercosis, and is the most important cause of seizures worldwide. Additional symptoms of neurocysticercosis include

headache, visual or mental status changes, stroke, meningoencephalitis, and obstructive hydrocephalus. Noncontrast CT is shows calcifications of inactive disease, and may reveal hydrocephalus. Therapy is **praziquantel**, 17 milligrams/ kilogram/dose 3 times daily (albendazole also used). Steroids are recommended for those with encephalitis, hydrocephalus, or vasculitis.

## **African Trypanosomiasis (African Sleeping Sickness)**

Sleeping sickness is transmitted by the aggressive tsetse fly. After a bite, a localized inflammatory reaction occurs followed in 2 to 3 days by a painless chancre that increases in size for 2 to 3 weeks, and then gradually regresses. Intermittent fevers follow, with malaise, rash, and eventual CNS involvement occurs, causing behavioral and neurologic changes, encephalitis, coma, and death. Other complications include hemolysis, anemia, pancarditis, and meningoencephalitis. Diagnosis is made by rapid evaluation of blood smears for the mobile parasite. Consult infectious disease expert for diagnosis and treatment with **suramin** and other agents.

## **Chagas Disease (American Trypanosomiasis)**

The protozoan *T. cruzi* is endemic in regions of Latin America and is reported as far north as Texas. It is spread by the reduviid “kissing bug” or “assassin” bug. The bug typically bites nocturnally after emerging from rural adobe walls or thatched roofs. Symptoms of the acute phase are unilateral periorbital edema (Romaña sign) or painful cutaneous edema at the site of skin penetration (chagoma), and is followed by a toxemic phase with parasitemia causing lymphadenopathy and hepatosplenomegaly. The acute phase diagnosis is made by examination of peripheral blood smears demonstrating motile parasites, or by blood culture. In the chronic phase, serologic tests or tissue biopsy are useful. Recommended treatment is **nifurtimox** (consult infectious disease).

## **Leishmaniasis (Visceral)**

*Leishmania* is an intracellular protozoan transmitted by *Lutzomyia* or *Phlebotomus* sandflies. Leishmaniasis should be suspected in the military and their families living proximal to jungles, adventure travelers, field biologists, and emigrants from endemic zones. The disease has a variety of syndromic presentations, the most important of which is visceral leishmaniasis, or Kala-azar, or Black Fever. It is typified by a pentad of fever, weight loss, hepatosplenomegaly, pancytopenia, and hypergammaglobulinemia. Treat visceral disease with pentavalent antimonials, either **sodium stibogluconate** (available through the CDC) or **meglumine antimonate**, available in some European countries.

### **Schistosomiasis (Snail Fever)**

Schistosomiasis should be suspected in travelers presenting with GI symptoms exposed to freshwater. The larvae are released into fresh water by snails. Soon after exposure, “swimmers itch” occurs with a macular-papular pruritic dermatitis over the lower legs, which can last for days. Four to 8 weeks later, fever occurs with headache, cough, urticaria, diarrhea, hepatosplenomegaly, and hypereosinophilia (Katayama fever). Worms mature in the venous blood and (if untreated) deposit eggs in the bladder, GI tract, brain, skin, and liver. Diagnosis is suspected from eosinophilia and microscopic identification of eggs in midday urines or stools. Treatment is with **praziquantel**, 20 milligrams/kilogram, 2 doses in a single day, except with GI involvement, where 3 doses in a single day are suggested.

### **Amebiasis**

Pathogenic species such as *Entamoeba histolytica* are endemic to Asia, Africa, and Latin America. Amebiasis is typically spread by asymptomatic carriers whose excrement contains encysted organisms. Incubation is 1 to 3 weeks for colitis, and weeks to months for liver abscess. Symptoms include alternating constipation with diarrhea, over weeks, to abdominal pain, fever, dehydration, and weight loss. Complication such as liver abscess cause

fever, right upper quadrant pain, chronic vague abdominal pain, and weight loss. Stool for ova and parasites is diagnostic (specimen should be examined within 30 min of collection). Ultrasound should identify liver abscess. Most common treatment is with **metronidazole**, 500 to 750 milligrams 3 times daily for 10 days.

## **Ascariasis**

Infection with *Ascaris lumbricoides* should be suspected following ingestion of street vendor foods or vegetables fertilized by “night soil” (human feces) or animal feces. Symptoms may include a dry cough or pneumonia as young worms are expectorated and migrate from the lungs to the esophagus and gut. A large worm burden can lead to malnutrition and weakness, and a mass of worms may lead to bowel obstruction. Diagnosis is with stool examination and serology. Treatment is with **mebendazole**, 100 milligrams daily for three days, or **albendazole**, 400 milligrams twice a day for 3 days or 500 milligrams single dose, or **ivermectin**, 150 to 200 micrograms/kilogram single dose. The single dose regimens are used, but have lower cure rates.

## **Enterobiasis (Seatworm or Pinworm)**

Infection is typically from fecal-oral contact from contaminated objects. Presentation is intense perianal itching. Diagnosis is with cellophane tape swab of anus to look for worms. Treatment is with **mebendazole**, 100 milligrams single dose and repeat in 2 weeks, or **albendazole**, 400 milligrams single dose and repeat in 2 weeks, or pyrantel pamoate, 11 milligrams/ kilogram (up to 1 gram) single dose and repeat in 2 weeks.

## ***Ancylostoma duodenale* and *Necator americanus* (Hookworm)**

Infection follows exposure to contaminated soil; larvae penetrate skin. Worms may migrate to the lungs, may be coughed up, and access the GI

tract after being swallowed. Symptoms include abdominal pain; severe anemia; and cutaneous larva migrans, red, wormlike burrows visible underneath the skin. Treatment is with **albendazole** 400 milligrams single dose (preferred), or **mebendazole** 100 milligrams twice daily for 3 days, or **Pyrantel pamoate**, 11 milligrams/kilogram (maximum, 1 gram) daily for 3 days.

***Taenia solium* (Pork Tapeworm), *Taenia saginata* (Beef Tapeworm), *Diphyllobothrium latum* (Fish Tapeworm)**

Infection follows ingestion of undercooked pork, beef, or fish. Symptoms include diarrhea, abdominal pain, bowel obstruction, and taenia cysts in eye, heart, and brain (see cysticercosis above). Diagnosis is by stool examination or serology (may be negative if cysts are calcified). Treatment is with **praziquantel**, 5 to 10 milligrams/kilogram single dose.

For discussion of other diseases that may be acquired during travel, or other parasites, see the chapter referenced immediately below.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 156, “World Travelers,” by Michael J. VanRooyen and Raghu Venugopal.



# CHAPTER 99

## The Transplant Patient

David M. Cline

Management of the transplant patient in the emergency department can be divided into 3 general areas: disorders specific to the transplanted organ; disorders common to all transplant patients due to their immunosuppressed state or antirejection medication; and disorders unrelated to their transplant, yet special care is required due to their medications or altered physiology. Disorders specific to the transplanted organ are manifestations of acute rejection, surgical complications specific to the procedure performed, and altered physiology (most important in cardiac transplantation). The most common presentations of transplant patients to the emergency are: infection (39%) followed by noninfectious GI/GU pathology (15%), dehydration (15%), electrolyte disturbances (10%), cardiopulmonary pathology (10%) or injury (8%), and rejection (6%). Before prescribing any new drug for a transplant recipient, the treatment plan should be discussed with a representative from the transplant team.

### POSTTRANSPLANT COMPLICATIONS

### INFECTIOUS

Predisposing factors to infections posttransplant include ongoing immunosuppression in all patients and the presence of diabetes mellitus, advanced age, obesity, and other host factors. [Table 99-1](#) lists the broad array of potential infections and the time after transplant they are most likely to occur.

<p><b>First month posttransplant</b></p> <p>Wound infection/anastomotic leaks: (<i>MRSA</i>, gram-negative bacteria)  Pneumonia: <i>Pseudomonas</i>, <i>Klebsiella</i>, <i>Legionella</i>, <i>Aspergillus</i>  UTI: gram-negative bacteria, enterococcus, <i>Candida</i>  Line-related sepsis: <i>Staphylococcus aureus</i>, <i>Staphylococcus epidermidis</i>, gram-negative bacteria, <i>Candida</i>  Abdomen: VRE, <i>Clostridium difficile</i>, intraabdominal infections (liver transplant)  Viral: disseminated/local, HSV, RSV (HSCT patients)  Febrile neutropenia: <i>Streptococcus viridans</i> (HSCT patients)  Oral: candidal pharyngitis, esophagitis</p>
<p><b>Second to sixth month posttransplant</b></p> <p>Pneumonia: CAP, <i>Nocardia</i>, CMV, <i>Aspergillus</i>, TB, <i>Pneumocystis</i>, <i>Listeria</i>  Meningitis: <i>Listeria</i>, <i>Cryptococcus</i>  UTI/cystitis/nephritis: common pathogens, polyomavirus BK  Abdominal: colitis: <i>C difficile</i>, visceral leishmaniasis  Oral: candidal pharyngitis, esophagitis  Viral: CMV, EBV, HSV, VZV, Adenovirus, influenza, Hepatitis A, B, C  Fever: toxoplasmosis, common presentation of most listed pathogens</p>
<p><b>Beyond sixth months posttransplant</b></p> <p>Pneumonia: CAP, <i>Aspergillus</i>, CMV, <i>Pneumocystis</i>, <i>Listeria</i>, <i>Nocardia</i>  UTI/cystitis: common pathogens, Polyomavirus BK  Meningitis: <i>Listeria</i>, <i>Cryptococcus</i>  Oral: candidal pharyngitis, esophagitis  Viral: CMV (chorioretinitis, colitis) VZV, HSV, hepatitis C, B, West Nile</p>

Key: CAP = community acquired pneumonia, CMV = cytomegalovirus, EBV = Epstein Barr Virus, HBV = hepatitis B virus, HCV = hepatitis C virus, HSCT = hemapoietic stem cell transplant, HSV = herpes simplex virus, MRSA = methicillin-resistant *Staphylococcus aureus*, PTLD = post-transplantation lymphoproliferative disorder, RSV = respiratory syncytial virus, TB = tuberculosis, UTI = urinary tract infection, VRE = vancomycin-resistant *Enterococcus faecalis*, VZV = varicella-zoster virus.

**TABLE 99-1** Infections in Transplant Patients Stratified by Posttransplant Period

## Clinical Features

The initial presentation of a potentially life-threatening infectious illness may be quite subtle in transplant recipients. As many as 50% of transplant patients, with serious infections, will not have fever. A nonproductive cough with little or no findings on physical examination may be the only clue to emerging *Pneumocystis jiroveci* pneumonia or cytomegalovirus (CMV) pneumonia. Urinary tract infections are a very common cause of fever in this group of patients.

## Diagnosis and Differential

Blood counts, inflammatory markers, baseline tests of renal and liver function may be helpful in this group of complex patients. The threshold for obtaining chest radiographs for these patients should be low. Cultures of all appropriate fluids, including blood, are essential before (or simultaneous with) initiating antimicrobial therapy. Central nervous system infections such as meningitis (*Listeria monocytogenes* and cryptococci) should be considered. Complaints of recurrent headaches, therefore, with or without fever, should be investigated vigorously, first with a structural study to exclude a mass lesion (central nervous system lymphomas occur with increased frequency, too) and then with a lumbar puncture. Liver transplant patients are especially susceptible to intraabdominal infections during the first postoperative month. Lung transplant patients are especially prone to pneumonia. Cardiac transplant patients may develop mediastinitis during the first postoperative month.

## Emergency Department Care and Disposition

1. Drug choice, dose, and ultimate management should be accomplished in consultation with the transplant team. The following recommended drugs are listed for the event of urgent patient need due to instability or delay in reaching the transplant team.
2. For skin and superficial wounds, a broad spectrum antibiotic plus an agent specific to MRSA is recommended. Therefore, **imipenem** 500 milligrams IV every 6 hours, *or* **meropenem** 1 gram IV every 8 hours, *or* **piperacillin/ tazobactam** 3.375 IV every 6 hours can be initiated *plus* **vancomycin** 1 gram IV every 12 hours *or* **linezolid** 600 milligrams IV every 12 hours are recommended.
3. Pneumonia may be caused by a wide variety of organisms from common to atypical to opportunistic. Treatment options include **imipenem** 500 milligrams IV every 6 hours, **meropenem** 1 gram IV every 8 hours, **cefotaxime** 1 to 2 grams IV every 6 to 8 hours plus **gentamicin** 1 to 2 milligrams/kilogram IV every 8 hours, *or* **piperacillin/tazobactam** 3.375

grams IV every 6 hours. Add MRSA specific therapy, listed above, and fungal therapy, listed below, if suspected.

4. Intraabdominal infection may be due to enteric gram-negative aerobic, obligate anaerobic bacilli and facultative bacilli, and enteric gram-positive streptococci. Recommended coverage is to combine **metronidazole** 500 milligrams IV every 12 hours *plus* one of the following agents: **imipenem** 500 milligrams IV every 6 hours, **meropenem** 1 gram IV every 8 hours, **doripenem** 500 milligrams IV every 8 hours, **piperacillin/ tazobactam** 3.375 grams IV every 6 hours. Ampicillin-sulbactam is not recommended for use because of high rates of resistance to this agent among community-acquired *E coli*.
5. Meningitis is frequently due to *L monocytogenes*, and patients with suspected meningitis should be treated **cefotaxime** 2 grams IV every 4 to 6 hours plus **vancomycin** 1 gram IV every 12 hours. The addition of vancomycin should be considered.
6. The initial treatment of suspected fungal disease is **fluconazole** 400 milligrams daily IV; amphotericin B 0.7 milligram/kilogram/d IV, has been a mainstay of treatment, but has more toxicity than fluconazole. Oral or esophageal *Candida*, treat with **fluconazole** 200 milligrams day 1, then 100 milligrams PO daily.
7. Suspected CMV disease is treated with **ganciclovir**, with a dose of 5 milligrams/kilogram IV twice daily; in bone marrow transplant patients, add immunoglobulin.
8. Varicella and herpes simplex virus are typically treated with **acyclovir** 800 milligrams IV 5 times a day for dissemination or ocular involvement. Acyclovir has renal excretion, and the dose must be adjusted for renal insufficiency. Alternatives include **valacyclovir** 1000 milligrams every 8 hours, and **famciclovir** 500 milligrams every 8 hours.
9. Epstein-Barr virus is typically treated with a reduction in the immunosuppression regimen. Both acyclovir and ganciclovir have also been used, but not routinely.
10. Treatment of choice for *Pneumocystis jiroveci* pneumonia starts with prednisone 80 milligrams/day followed immediately by antimicrobial therapy. First choice is **trimethoprim/sulfamethoxazole** (TMP-SMX), TMP 15 milligrams/kilogram/d IV divided every 8 hours while critically ill. Oral therapy is TMP-SMX double strength (DS) 2 tablets PO every 8

hours for 3 weeks of total therapy. **Pentamidine** 4 milligrams/kilogram/d IV or IM for 3 weeks, or **clindamycin** 600 milligrams IV *plus* **primaquine** 30 milligrams orally daily are reserved as alternative therapies if TMP-SMX is not tolerated.

11. Toxoplasmosis can be treated initially with **pyrimethamine** 200 milligrams PO initially then 50 to 75 milligrams PO daily plus **sulfadiazine** 1 to 4 grams PO daily plus **folinic acid** 10 milligrams PO daily.
12. Urinary tract infections (see [Chapter 54](#)), invasive gastroenteritis (due to *Salmonella*, *Campylobacter*, and *Listeria*, see [Chapter 37](#) and [Chapter 96](#)), and diverticulitis (see [Chapter 44](#)) can be treated with the usual antimicrobial agents.

## COMPLICATIONS OF IMMUNOSUPPRESSIVE AGENTS

Therapeutic immunosuppression is accompanied by a number of adverse effects and complications. These adverse effects are typically gradual in onset, but may be life threatening such as pancreatitis, bleeding, hypoglycemia or hyperglycemia, bradycardia or tachycardia, hyperkalemia, hypertension or hypotension, cardiotoxicity, pulmonary edema, seizures, thromboembolic events, and thrombocytopenia. Side effects such as fever or rigors may also be confused for life-threatening infections. A headache syndrome often indistinguishable from migraine is common in transplant recipients and usually develops within the first 2 months of immunosuppression. An important differential must include infectious causes and malignancy when headache first presents and usually requires computed tomography of the head with subsequent biochemical analysis of cerebro-spinal fluid. As the number of immunosuppressive drugs has increased dramatically, a complete listing of adverse effects is beyond the scope of this manual. The reader is referred to the parent textbook, referenced at the end of this manual chapter, or to web resources, or a personal digital assistant, for a more complete listing of side effects of these medications.

Any illness that prevents transplant patients from taking or retaining their immunosuppressive therapy warrants hospital admission for IV therapy, preferably at a transplant center. Starting even simple medications can precipitate complications. For example, nonsteroidal anti-inflammatory drugs may increase nephrotoxicity. In general, any new medications should be discussed with a representative of the patient's transplant team.

## **CARDIAC TRANSPLANTATION**

Transplantation results in a denervated heart that does not respond with centrally mediated tachycardia in response to stress or exercise but does respond to circulating catecholamines and increased preload. Patients may complain of fatigue or shortness of breath with the onset of exercise, which resolves with continued exertion as an appropriate tachycardia develops.

The donor heart is implanted with its sinus node intact to preserve normal atrioventricular conduction. The normal heart rate for a transplanted heart is 90 to 100 beats/min. The technique of cardiac transplantation also results in the preservation of the recipient's sinus node at the superior cavoatrial junction. The atrial suture line renders the 2 sinus nodes electrically isolated from each other. Thus, electrocardiograms frequently will have 2 distinct P waves. The sinus node of the donor heart is easily identified by its constant 1:1 relation to the QRS complex, whereas the native P wave marches independently through the donor heart rhythm.

### **Clinical Features**

Because the heart is denervated, myocardial ischemia does not present with angina. Instead, recipients present with heart failure secondary to silent myocardial infarctions or with sudden death. Transplant recipients who have new onset shortness of breath, chest fullness, or symptoms of congestive heart failure should be evaluated, in routine fashion with an electrocardiogram and serial cardiac enzymes levels, for the presence of myocardial ischemia or infarction.

Although most episodes of acute rejection are asymptomatic, symptoms can occur. The most common presenting symptoms are dysrhythmias and generalized fatigue. The development of atrial or ventricular dysrhythmia in a cardiac transplant recipient (or congestive heart failure) must be assumed to be due to acute rejection until proven otherwise. In children, rejection may present with low-grade fever, fussiness, and poor feeding.

## **Emergency Department Care and Disposition**

1. Rejection: Management of acute rejection is **methylprednisolone** 1 gram IV after consultation with a representative from the transplant center. Treatment for rejection without biopsy confirmation is contraindicated except when patients are hemodynamically unstable.
2. Dysrhythmias: If patients are hemodynamically compromised by dysrhythmias, empiric therapy for rejection with **methylprednisolone** 1 gram IV may be given after consultation. Atropine has no effect on the denervated heart; isoproterenol is the drug of choice for bradycardia in these patients. Patients who present in extremis should be treated with standard cardiopulmonary resuscitation measures.
3. Hypotension: Low-output syndrome, or hypotension, should be treated with inotropic agents such as dopamine or dobutamine when specific treatment for rejection is instituted.
4. Hospitalization: Transplant patients suspected of having rejection or acute illness should be hospitalized, preferably at the transplant center, if stable for transfer.

## **LUNG TRANSPLANTATION**

### **Clinical Features**

Clinically, the patient suffering rejection may have a cough, chest tightness, fatigue, and fever ( $> 0.5^{\circ}\text{C}$  above baseline). Acute rejection may manifest with frightening rapidity, causing a severe decline in patient status in only 1 day. Isolated fever may be the only finding. Spirometry may show a 15%

drop in forced expiratory volume in 1 second, the patient may be newly hypoxic, and examination may show rales and adventitious sounds. Chest radiograph may demonstrate bilateral interstitial infiltrates or effusions but may be normal when rejection occurs late in the course. The longer a patient is from transplant, the less classic a chest radiograph may appear for acute rejection. Infection, such as interstitial pneumonia, may present with a clinical picture similar to acute rejection. Diagnostically, bronchoscopy with transbronchial biopsy is usually needed not only to confirm rejection but also to exclude infection.

Two late complications of lung transplant are obliterative bronchiolitis and posttransplant lymphoproliferative disease (PTLD). Obliterative bronchiolitis presents with episodes of recurrent bronchitis, small airway obliteration, wheezing, and eventually respiratory failure. PTLD is associated with Epstein-Barr virus and presents with painful lymphadenopathy and otitis media (due to tonsillar involvement) or may present with malaise, fever, and myalgia.

## **Diagnosis and Differential**

Evaluation of the lung transplant patient should include chest radiograph, pulse oximetry, arterial blood gas analysis (if CO<sub>2</sub> retention is suspected), spirometry, complete blood cell count, serum electrolytes, creatinine and magnesium levels, and appropriate drug levels.

## **Emergency Department Care and Disposition**

1. Rejection: After consultation with the transplant center representative, and infection is excluded, **methylprednisolone** 500 to 1000 milligrams IV should be given for acute rejection. Patients who have a history of seizures associated with the administration of high-dose glucocorticoids also will need concurrent benzodiazepines to prevent further seizure episodes.
2. Late complications: Obliterative bronchiolitis is treated with increased immunosuppression including high-dose steroids, whereas PTLD is



treated with reduced immunosuppression and other therapy such as rituximab. These decisions should be made in consultation with specialists from the transplant center.

## **RENAL TRANSPLANT**

### **Clinical Features**

Diagnosis and treatment of acute rejection is most critical. Without timely recognition and intervention, allograft function may deteriorate irreversibly in a few days.

Renal transplant recipients, when symptomatic from acute rejection, complain of vague tenderness over the allograft (in the left or right iliac fossa). Patients also may describe decreased urine output, rapid weight gain (from fluid retention), low-grade fever, and generalized malaise. Physical examination may disclose worsening hypertension, allograft tenderness, and peripheral edema. The absence of these symptoms and signs, however, does not exclude the possibility of acute rejection. With improved methods of maintenance immunosuppression, the only clue may be an asymptomatic decline in renal function.

### **Diagnosis and Differential**

Even a change in creatinine levels from 1.0 milligrams/dL to 1.2 or 1.3 milligrams/dL may be important. When such changes in creatinine levels are reproducible, a careful workup consists of complete urinalysis, possibly renal ultrasonography, and levels of immunosuppressive drugs if available, in addition to a careful history and examination. It is critical to interpret changes in renal function in the context of prior data (eg, trends of recent serum creatinine levels, recent history of rejection, or other causes of allograft dysfunction). Evaluation should consider the multiple etiologies of decreased renal function in the renal transplant recipient. The 2 most

common causes, apart from acute rejection causing an increase in creatinine, are volume contraction and cyclosporine-induced nephrotoxicity.

## **Emergency Department Care and Disposition**

1. Rejection: After consultation with the transplant center representative, treatment of allograft rejection consists of high-dose glucocorticoids, typically **methylprednisolone** 500 milligrams IV.

# **LIVER TRANSPLANT**

## **Clinical Features**

Although frequently subtle in presentation, a syndrome of acute rejection includes fever, liver tenderness, lymphocytosis, eosinophilia, liver enzyme elevation, and a change in bile color or production. In the perioperative period, the differential diagnosis must include infection, acute biliary obstruction, or vascular insufficiency. Diagnosis can be made with certainty only by hepatic ultrasound and biopsy, which usually requires referral back to the transplant center for management and follow up.

Two possible surgical complications in liver transplant patients are biliary obstruction or leakage and hepatic artery thrombosis. Biliary obstruction follows 3 typical presentations. The most common is intermittent episodes of fever and fluctuating liver function tests. The second is a gradual worsening of liver function tests without symptoms. Third, obstruction may present as acute bacterial cholangitis with fever, chills, abdominal pain, jaundice, and bacteremia. It can be difficult to distinguish clinically from rejection, hepatic artery thrombosis, CMV infection, or a recurrence of a preexisting disease, especially hepatitis.

If a biliary complication is suspected, all patients should have a complete blood count; serum chemistry levels; liver function tests; basic coagulation studies; and lipase levels; cultures of blood, urine, bile, and ascites, if present; chest radiograph; and abdominal ultrasound. Ultrasound looks for



conditions. The most common complication of HSCT is graft-versus-host disease, affecting approximately 50% of HSCT patients.

### **Clinical Features (graft-versus-host disease)**

A HSCT recipient presenting to the ED with nonspecific rash (see [Fig. 99-1](#)) should be suspected of having graft-versus-host disease. The rash may be pruritic or painful, frequently demonstrating a brownish hue and slight scaling. The distribution varies greatly but often affects palms and soles initially, and later progresses to cheek, ears, neck, trunk, chest, and upper back. In the more severe forms, skin involvement is erythrodermic or may show bullae formation. Mucositis has been reported to occur in 35% to 70% of patients. As many as 90% of patients undergoing combined chemotherapy and radiotherapy develop severe skin disease.



**FIGURE 99-1.** Rash of acute cutaneous graft-versus-host disease. The maculopapular lesions have acquired a brownish hue and there is slight

scaling. (Reproduced with permission from Wolff KL, Johnson R, Suurmond R. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*, 6th ed. © 2009, McGraw-Hill Companies Inc. All rights reserved.)

The second most common presentation is gastrointestinal with diarrhea. Upper GI symptoms such as anorexia, nausea, and emesis are common. The patient may develop painful cramping, ileus, and, sometimes, life-threatening hemorrhage from the colon.

### **Diagnosis and Differential (graft-versus-host disease)**

The diagnosis of graft-versus-host disease is made on clinical grounds initially. The patient with serious GI hemorrhage in the early posttransplant period may have coagulation deficits, especially thrombocytopenia. The differential diagnosis of GI bleeding in this setting includes all the usual causes of GI bleeding in addition to infection (viral, fungal, or bacterial). Liver involvement presents with hyperbilirubinemia and increases in alkaline phosphatase and transaminase levels.

### **Emergency Department Care and Disposition**

1. Most patients with graft-versus-host disease will need supportive care in consultation with the patient's transplant team for management including possible admission or transfer to the transplant center.
2. Initiation of **prednisone** 60 milligrams PO daily, or **methylprednisolone** at 1 to 2 milligrams/kilogram IV daily until clinical improvement is seen is the usual management.
3. If other immunosuppressants that have recently been tapered or discontinued are, generally, increased or reinstated.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 295, "The Transplant Patient," by Raymond M. Fish and Malek G. Massad.

## **SECTION 11 Toxicology and Pharmacology**

## **CHAPTER 100**

# **General Management of the Poisoned Patient**

L. Keith French

Knowledge of appropriate decontamination techniques and timely administration of antidotes coupled with excellent supportive care may positively alter the outcome of poisoned patients.

## **CLINICAL FEATURES**

A detailed history is essential in the evaluation of a potentially poisoned patient. In the conscious, cooperative person, the specific agent(s), time, route, amount, and intent of exposure need to be documented. In the uncooperative or altered patient, adjunctive information from friends, family, prehospital providers, police, or bystanders may provide more accurate details. Environmental clues such as drug paraphernalia, empty pill bottles, odors, or suicide notes may aid in the diagnosis. If available, review hospital records for recent prescriptions or any history of psychiatric illness. Loose pills with imprint codes may be identified by the pharmacy or poison center.

A thorough exam begins with a completely disrobed patient. Search clothing and personal possessions, but remain vigilant while doing so as to avoid potential injury from needles or chemicals. Review vital signs and perform a comprehensive physical examination. Focus on the general appearance, level of consciousness, pupil size, mucous membranes, respiratory rate, breath sounds, presence of bowel sounds, skin temperature, and muscle tone as the combination of findings may suggest a specific toxidrome ([Table 100-1](#)).

Toxidrome	Representative Agent(s)	Most Common Findings	Additional Signs and Symptoms	Potential Interventions
Opioid	Heroin Morphine Oxycodone	Central nervous system depression; miosis, respiratory depression	Hypothermia, bradycardia Death may result from respiratory arrest, acute lung injury	Ventilation or naloxone
Sympathomimetic	Cocaine Amphetamine	Psychomotor agitation, mydriasis, diaphoresis, tachycardia, hypertension, hyperthermia	Seizures, rhabdomyolysis, myocardial infarction Death may result from seizures, cardiac arrest, hyperthermia	Cooling, sedation with benzodiazepines, hydration
Cholinergic	Organophosphate insecticides Carbamate insecticides	Muscarinic effects (salivation, lacrimation, diaphoresis, nausea, vomiting, urination, defecation, bronchorrhea) Nicotinic effects (muscle fasciculations and weakness)	Bradycardia, miosis/mydriasis, seizures, respiratory failure, paralysis Death may result from respiratory arrest from paralysis, bronchorrhea, or seizures	Airway protection and ventilation, atropine, pralidoxime
Anticholinergic	Scopolamine Atropine	Altered mental status, mydriasis, dry flushed skin, urinary retention, decreased bowel sounds, hyperthermia, dry mucous membranes	Seizures, dysrhythmias, rhabdomyolysis Death may result from hyperthermia and dysrhythmias	Physostigmine (if appropriate), sedation with benzodiazepines, cooling, supportive management
Salicylates	Aspirin Oil of wintergreen	Altered mental status, respiratory alkalosis, metabolic acidosis, tinnitus, hyperpnea, tachycardia, diaphoresis, nausea, vomiting	Low-grade fever, hematuria Death may result from acute lung injury or cerebral edema	Multidose activated charcoal, alkalinization of urine with potassium repletion, hemodialysis
Sedative-hypnotic	Barbiturates Benzodiazepines	Depressed level of consciousness, slurred speech, ataxia	Stupor to coma, depressed respirations, apnea, bradycardia	Ventilatory support



Hypoglycemic	Sulfonylureas Insulin	Altered mental status, diaphoresis, tachycardia, hypertension	Paralysis, slurring of speech, bizarre behavior, seizures Death may result from seizures	Glucose-containing solution IV and oral feedings If possible, frequent glucose measurement, octreotide
Hallucinogenic	Phencyclidine Lysergic acid diethylamide Psilocybin Mescaline	Hallucinations, dysphoria, anxiety	Hyperthermia, mydriasis, nausea, sympathomimetic symptoms	Generally supportive
Serotonin	SSRIs Meprobamate A variety of drug interactions with dextromethorphan, monoamine oxidase inhibitors, tricyclic antidepressants, other SSRIs, and amphetamines	Altered mental status, increased muscle tone, hyperreflexia, hyperthermia	Intermittent whole-body tremor Death may result from hyperthermia	Cooling, sedation with benzodiazepines, supportive management, theoretical benefit of cyproheptadine
Extrapyramidal	Haloperidol Phenothiazines Risperidone Clozapine	Dystonia, torticollis, tremor, muscle rigidity	Choreoathetosis, hyperreflexia, seizures	Diphenhydramine Benztropine Benzodiazepines

Key: SSRI = selective serotonin reuptake inhibitor.

**TABLE 100-1** Common Toxidromes

## DIAGNOSIS AND DIFFERENTIAL

A diagnosis of poisoning is established primarily through the history and physical examination. While specific toxicology screens are often available, in general, these are of limited utility and seldom impact care and outcome. False negative and positive results on urine drug screening may be confusing and potentially distract the clinician. Because acetaminophen and aspirin are easily obtained, often combined in over-the-counter products, and have specific therapies, consider empiric testing in all potentially poisoned patients. Also consider blood glucose, an arterial blood gas analysis, ECG, urine pregnancy test, electrolytes, and liver function panel.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Gross decontamination should occur **prior** to patient entry into the ED. Care providers need to protect themselves with properly fitted gloves, masks, and gowns. If a mass casualty exposure exists, staging centers may have already initiated surface decontamination. Ensure that clothing and jewelry are removed. Wash the skin with copious amounts of water.
2. The primary goal in management is **resuscitation**. Focus on assessment and stabilization of the airway, breathing, and circulation. Place patients on cardiac monitors and obtain an ECG. Oxygen saturations and core temperature are important vital signs to note. Obtain bedside point of care testing for blood glucose concentrations. Administer **oxygen**. Treat hypotension initially with fluids before initiating pressors. Treat ventricular dysrhythmias according to standard ACLS/PALS protocols.
3. Early **endotracheal intubation** may be necessary given the anticipated clinical course of some toxidromes. Initiate airway protection prior to GI decontamination.
4. The proper and timely use of antidotes ([Table 100-2](#)) is paramount in the management of many poisoned patients. Rarely, however, does antidotal

therapy trump standard resuscitation steps. Focus first on IV or IO access, oxygenation, ventilation, fluid administration, and airway protection.

Antidote	Dose		Indication
	Pediatric	Adult	
N-acetylcysteine	140 milligrams/kilogram PO load, followed by 70 milligrams/kilogram PO every 4 h for 17 total doses or 150 milligrams/kilogram IV load over 60 min, followed by 50 milligrams/kilogram IV over 4 h and then 100 milligrams/kilogram IV over 16 h		Acetaminophen
Activated charcoal	1 gram/kilogram PO	50 to 100 grams	Most ingested poisons
Antivenom Fab	4 to 6 vials IV initially over 1 h, may be repeated to gain control of progressive symptoms		Envenomation by Crotalidae
Calcium chloride 10% (27.2 milligrams/mL elemental calcium)	0.2 to 0.25 mL/kg IV	10 mL IV	Calcium channel antagonists
Calcium gluconate 10% (9 milligrams/mL elemental calcium)	0.6 to 0.8 mL/kg IV	10 to 30 mL IV	Hypermagnesemia Hypocalcemia
Cyanide antidote kit Amyl nitrite	Not typically used	1 ampule in oxygen chamber of ventilation bag 30 s on/30 s off	Cyanide
Sodium nitrite (3% solution)	0.53 mL/kg IV	10 mL IV	Cyanide Hydrogen sulfide
Sodium thiosulfate (25% solution)	1.65 mL/kg IV	50 mL IV	Cyanide
Deferoxamine	30 milligrams/kilogram IM (1 gram maximum) or 15 milligrams/kilogram IV (maximum dose, 1 gram/day)	2 grams IM or 15 milligrams/kilogram IV (maximum dose, 6 to 8 grams/day)	Iron

Dextrose (glucose)	0.5 gram/kilogram IV	1 gram/kilogram IV	Insulin Oral hypoglycemics
Digoxin Fab			
Acute	1 to 2 vials IV	5 to 10 vials IV	Digoxin and other cardioactive steroids
Chronic	1 to 2 vials IV	3 to 6 vials IV	Cardioactive steroids
Ethanol (10% for IV administration)	10 mL/kg IV over 30 min, then 1.2 mL/kg/h*	10 mL/kg IV over 30 min, then 1.2 mL/kg/h*	Ethylene glycol Methanol
Folic acid/leucovorin	1 to 2 milligrams/kilogram IV every 4 to 6 h	1 to 2 milligrams/kilogram IV every 4 to 6 h	Methotrexate (only leucovorin)
Fomepizole	15 milligrams/kilogram IV, then 10 milligrams/kilogram every 12 h	15 milligrams/kilogram IV, then 10 milligrams/kilogram every 12 h	Methanol Ethylene glycol Disulfiram-ethanol interaction
Flumazenil	0.01 milligram/kilogram IV	0.2 milligram IV	Benzodiazepines
Glucagon	50 to 150 micrograms/kilogram IV	3 to 10 milligrams IV	Calcium channel antagonists β-blockers
Hydroxocobalamin	70 milligrams/kilogram IV (not to exceed 5 grams over 30 min); can be repeated up to 3 times Administered in combination with sodium thiosulfate		Cyanide Nitroprusside
IV lipid emulsion 20%	1.5 mL/kg IV bolus over 1 min (may be repeated 2 times at 5-min intervals), followed by 0.25 mL/kg/min IV	100 mL IV bolus over 1 min, followed by 400 mL IV over 20 min	IV liposoluble Rescue therapy for calcium channel antagonists and β-blockers
Methylene blue	1 to 2 milligrams/kilogram IV Neonates: 0.5 to 1.0 milligram/kilogram	1 to 2 milligrams/kilogram IV	Oxidizing chemicals (eg, nitrites, benzocaine, sulfonamides)
Octreotide	1 microgram/kilogram SC every 6 h	50 to 100 micrograms SC every 6 h	Refractory hypoglycemia after oral hypoglycemic agent ingestion

Naloxone	As much as is needed Typical starting dose is 0.01 milligram IV	As much as needed Typical starting dose is 0.4 to 2.0 milligrams IV	Opioid Clonidine
Physostigmine	0.02 milligram/kilogram IV	0.5 to 2.0 milligrams slow IV over 2 to 5 min	Anticholinergic agents (not cyclic antidepressants)
Pralidoxime (2-PAM)	20 to 40 milligrams/kilogram IV over 5 to 10 min, followed by 20 milligrams/kilogram/h infusion	1 to 2 grams IV over 5 to 10 min, followed by 500 milligrams/h infusion	Cholinergic agents
Protamine	1 milligram neutralizes 100 units of unfractionated heparin, administered over 15 min 0.6 milligram/kilogram IV (empiric dose)	25 to 50 milligrams IV (empiric dose)	Heparin
Pyridoxine	1.0 gram for 1.0 gram of ingestion if amount of isoniazid is known 70 milligrams/kilogram (maximum 5 grams) IV	5 grams IV	Isoniazid Cyanide esters Hydrazine
Sodium bicarbonate	1 to 2 mEq/kg IV bolus followed by 2 mEq/kg/h IV infusion	1 to 2 mEq/kg IV bolus followed by 2 mEq/kg/h IV infusion	Sodium channel blockers For urinary alkalization
Thiamine	5 to 10 milligrams IV	100 milligrams IV	Wernicke syndrome Vitamin B <sub>1</sub> deficiency
Vitamin K <sub>1</sub>	1 to 5 milligrams/day PO	20 milligrams/d PO	Anticoagulant rodenticides

\*This is an approximation. Dose should be titrated to level (see Chapter 104, Antidotes).

**TABLE 100-2** Common Antidotes: Initial Dosages and Indications

5. Altered mental status and coma are common presentations of many intoxicants. Reasonable empiric treatments include supplemental oxygen, **naloxone** (0.2 to 2.0 milligrams IV/IO/IM), **glucose** (1 to 1.5 grams/kilogram IV/IO), and **thiamine** (10 to 100 milligrams IV/IO in the adult patient). The routine use of flumazenil in the treatment of an undifferentiated, obtunded patient is potentially dangerous and not recommended. The dogma that thiamine administration must precede glucose administration is unfounded.
6. Seizures from toxins generally respond to benzodiazepines or barbiturates. **Lorazepam** (0.05 to 0.1 milligram/kilogram IV/IO in children or 1 to 2 milligrams IV/IO in adults) is a reasonable first-line agent. Phenytoin is generally not effective in toxin-induced seizures and may exacerbate dysrhythmias in some poisonings.
7. Once stabilized, **surface decontamination** is the next priority in care. If not previously done, completely disrobe the patient. Dermal toxins must be removed from the skin by irrigation. Ocular exposure often requires pain control with topical agents such as 0.5% tetracaine. Copiously irrigate the eye with isotonic crystalloid. This may require several liters before restoration of physiologic pH.
8. Gastrointestinal decontamination is achieved via removal of the toxin from the stomach, binding toxin within the GI tract, or enhancing transit time through the gut. The particular method(s) utilized, if any, depends on the route, timing, amount, and nature of the toxin. With all modalities, the patient must be able to protect their airway during the process. If this is not possible, strongly consider endotracheal intubation to help reduce the risk of aspiration.
  - a. Inducing emesis with syrup of ipecac is no longer recommended for routine use. **Orogastric lavage** is now less frequently used in the management of poisoned patients due to potentially serious consequences and limited data demonstrating improvement in clinical outcomes. This procedure requires advancement of a large, typically 36F to 40F orogastric tube into the stomach while the patient lies in a left lateral position. The head of the bed should be titled down 20°. Aliquots of roughly 250 mL of room-temperature fluid are instilled into the stomach then removed via gravity or suction. The procedure continues until effluent is clear. Activated charcoal (AC) should be

instilled through the tube before removal. This method is generally not useful beyond 1 to 2 hours postingestion.

b. **Activated charcoal** binds a large number of xenobiotics and prevents their absorption across the GI tract. The dose is typically 1 gram/ kilogram in children or 25 to 50 grams in adults. The minimal dose should be no less than a 10:1 ratio of AC to drug. Only the first dose of AC should be used with a cathartic, and only if diarrhea is not expected. An awake, alert, and cooperative patient may drink the mixture. Alternatively, AC can be infused through an NG tube.

c. **Whole-bowel irrigation (WBI)** is best accomplished through placement of an NG tube and instilling **polyethylene glycol** at a rate 1 to 2 L/h until rectal effluent is clear. Indications for WBI include sustained-release tablets, certain metals, and drugs carried by body stuffers/packers ([Table 100-3](#)). Contraindications include diarrhea, decreased bowel sounds, or intestinal obstruction.

Sustained- or delayed-release formulations
Agents with potential for bezoar formation
Iron and other heavy metals
Paint chips containing lead
Lithium
Drugs carried by body stuffers and body packers

**TABLE 100-3** Ingestions for Which Whole-Bowel Irrigation May Be Helpful

9. Considerations for enhanced elimination depend on the specific toxin and response to standard treatment. Urinary **alkalinization** and **hemodialysis** are the 2 most frequently utilized modalities. Forced diuresis has essentially no role in enhancing elimination.
10. Disposition depends on the nature of the intoxicant. Medical management is the priority and special attention must be made for exposures that may result in delayed toxicity.
11. Consider early consultation with a toxicologist and Poison Center for all poisonings. Consult with a mental health specialist for all intentional overdoses. Consider neglect or abuse in pediatric exposures.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 170, “General Management of Poisoned Patients,” by



Jason B. Hack and Robert S. Hoffman.

# CHAPTER 101

## Anticholinergic Toxicity

O. John Ma

### CLINICAL FEATURES

Clinical findings include hypotension or hypertension, tachycardia, hypoactive or absent bowel sounds, urinary retention, flushed skin, hyperthermia, dry skin and mucus membranes, mydriasis, confusion, agitation, disorientation, and auditory and visual hallucinations. [Table 101-1](#) compares muscarinic and antimuscarinic effects.

Organ	Stimulation or Muscarinic Effect	Antagonism or Antimuscarinic Effect
Brain	Complex interactions, possible improvement in memory	Complex interactions, impairs memory, produces agitation, delirium, hallucinations, and fever
Eye	Constricts pupil (miosis), decreases intraocular pressure, increases tear production	Dilates pupil (mydriasis), loss of accommodation (blurred vision), increases intraocular pressure
Mouth	Increases saliva production	Decreases saliva production, dry mucous membranes
Lungs	Bronchospasm, increases bronchial secretions	Bronchodilation
Heart	Bradycardia, slows atrioventricular conduction	Tachycardia, enhances atrioventricular conduction
Peripheral vasculature	Vasodilation (modest)	Vasoconstriction (very modest)
GI	Increases motility, increases gastric acid production, produces emesis	Decreases motility, decreases gastric acid production
Urinary	Stimulates bladder contraction and expulsion of urine	Decreases bladder activity, promotes urinary retention
Skin	Increases sweat production	Decreases sweat production (dry skin), cutaneous vasodilation (flushed appearance)

**TABLE 101-1** Muscarinic and Antimuscarinic Effects

## DIAGNOSIS AND DIFFERENTIAL

The diagnosis is primarily clinical. In isolated anticholinergic toxicity, routine laboratory studies should be normal, and routine toxicology screening is often of little value. Nonetheless, electrolytes, glucose, creatine phosphokinase, and pulse oximetry should be obtained. The differential diagnosis includes viral encephalitis, Reye syndrome, head trauma, other intoxications, neuroleptic malignant syndrome, delirium tremens, acute psychiatric disorders, and sympathomimetic toxicity.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

Treatment is primarily supportive. The goal is to prevent life-threatening complications, which include status epilepticus, hyperthermia, cardiovascular collapse, and rhabdomyolysis.

1. The patient should be placed on a cardiac monitor and intravenous or intraosseous access secured.
2. **Activated charcoal** may decrease drug absorption, even beyond 1 hour of ingestion.
3. Temperature monitoring is essential. Hyperthermia is treated conventionally.
4. Hypertension usually does not require intervention, but should be treated conventionally as necessary.
5. Standard antiarrhythmics are usually effective, but avoid class IA medications (eg, procainamide). Treat dysrhythmias, widened QRS complexes, and hypotension from sodium blocking agents (eg, cyclic antidepressants) with IV sodium bicarbonate 1 mEq/kg.
6. Treat agitation with benzodiazepines (**lorazepam 2 to 4 milligrams IV or 0.1 milligram/kilogram**). Phenothiazines should be avoided.

7. Treat seizures with benzodiazepines (lorazepam 2 milligrams IV).
8. **Physostigmine** treatment is controversial. It is indicated if conventional therapy fails to control seizures, agitation, unstable dysrhythmias, coma with respiratory depression, malignant hypertension, or hypotension. The initial dose is 0.5 to 2 milligrams IV (0.02 milligram/kilogram in children, maximum dose 0.5 milligram/dose), slowly administered over 5 min. When effective, a significant decrease in agitation may be apparent within 15 to 20 min. Physostigmine may worsen cyclic antidepressant toxicity and lead to bradycardia and asystole. It is contraindicated in patients with cardiovascular or peripheral vascular disease, bronchospasm, intestinal or bladder obstruction, cardiac conduction disturbances, and suspected concomitant sodium channel antagonist poisoning. The patient should be observed for cholinergic excess.
9. Patients with mild anticholinergic toxicity can be discharged after 6 hours of observation if their symptoms have resolved. More symptomatic patients should be admitted for 24 hours of observation. Patients who receive physostigmine usually require, at least, a 24-hour admission.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 196, "Anticholinergics," by Paul M. Wax and Amy C. Young.

# CHAPTER 102

## Psychopharmacologic Agents

C. Crawford Mechem

### CYCLIC ANTIDEPRESSANTS

Cyclic antidepressants inhibit reuptake of norepinephrine and serotonin and antagonize postsynaptic serotonin receptors. They can produce severe toxicity in overdose.

#### Clinical Features

Toxicity may present with altered mental status, seizures, cardiac conduction or rhythm disturbances, hypotension, respiratory depression, and, in severe cases, coma.

#### Diagnosis

ECG changes include sinus tachycardia; right axis deviation of the terminal 40 milliseconds; PR, QRS, and QT interval prolongation; right bundle-branch block; A-V blocks; and the Brugada pattern.

#### Emergency Department Care and Disposition

Care is primarily supportive.

1. Obtain IV access and initiate cardiac rhythm and ECG monitoring.
2. Patients should receive 1 gram/kilogram of **activated charcoal** PO. This may be preceded by gastric lavage in patients presenting <1 hour after a

large ingestion.

3. Hypotension is treated with isotonic crystalloids. If no response, administer **sodium bicarbonate** as an IV bolus of 1 to 2 mEq/kg, repeated until the patient improves or until blood pH is 7.50 to 7.55. A continuous IV infusion (150 mEq added to 1 L of 5% dextrose in water) may be used at a rate of 2 to 3 mL/kg/h. **Norepinephrine** is indicated if hypotension persists.
4. Treat conduction disturbances and ventricular dysrhythmias with **sodium bicarbonate**. **Synchronized cardioversion** may be indicated for unstable patients. Treat torsades de pointes with 2 grams of IV **magnesium sulfate**.
5. Control agitation with **benzodiazepines**.
6. Treat seizures with benzodiazepines. **Phenobarbital**, starting at 15 milligrams/kilogram IV, may be required for refractory seizures.
7. Patients who remain asymptomatic after 6 hours do not need admission for toxicologic reasons. Admit symptomatic patients to a monitored bed or intensive care unit (ICU).

## **ATYPICAL ANTIDEPRESSANTS, SEROTONIN REUPTAKE INHIBITORS, AND SEROTONIN SYNDROME**

Newer antidepressants include trazodone, bupropion, mirtazapine, selective serotonin reuptake inhibitors, and serotonin/norepinephrine reuptake inhibitors. They are safer than older agents but can still cause toxicity, including the serotonin syndrome.

### **TRAZODONE**

#### **Clinical Features**

Symptoms of toxicity include central nervous system depression, ataxia, dizziness, seizures, orthostatic hypotension, vomiting, and abdominal pain.

ECG abnormalities include QT interval prolongation, sinus bradycardia and tachycardia, and torsades de pointes.

## **Emergency Department Care and Disposition**

Supportive care is generally sufficient in isolated overdoses.

1. Initiate cardiac rhythm monitoring and obtain a 12-lead ECG.
2. Single-dose **activated charcoal** is recommended. **Gastric lavage** followed by activated charcoal may be beneficial for trazodone ingestions > 2 grams if early after ingestion.
3. Treat hypotension with isotonic IV fluids, followed by **norepinephrine**.
4. Treat torsades de pointes with IV **magnesium sulfate**.
5. Discharge patients who remain asymptomatic for at least 6 hours, with psychiatric evaluation as indicated. Admit those with neurologic and/or cardiac symptoms for > 6 hours after ingestion to a monitored bed.

## **BUPROPION**

### **Clinical Features**

Toxicity manifests as agitation, dizziness, tremor, vomiting, drowsiness, and tachycardia. Seizures are more common than with other atypical antidepressants. ECG changes include sinus tachycardia, QRS interval widening, and QT interval prolongation.

### **Emergency Department Care and Disposition**

Seizures should be anticipated. Cardiotoxicity is unlikely in isolated overdoses.

1. Start a peripheral IV line and initiate cardiac rhythm monitoring.

2. GI decontamination is recommended if done within 1 hour of ingestion. Consider **whole-bowel irrigation** in overdoses of sustained-release products.
3. Treat seizures with **benzodiazepines**, followed by **phenobarbital**.
4. Observe asymptomatic patients for 8 hours. Monitor patients ingesting >450 milligrams of sustained-release bupropion for up to 24 hours. Admit those with seizures, persistent tachycardia, or lethargy.

## **MIRTAZAPINE**

### **Clinical Features**

Toxicity causes sedation, confusion, sinus tachycardia, and hypertension. Coma and respiratory depression are seen in severe cases or with coingestion of other sedatives.

### **Emergency Department Care and Disposition**

1. Isolated overdoses can generally be managed with supportive care.
2. Single-dose **activated charcoal** is recommended for GI decontamination.
3. Admit symptomatic patients to a monitored bed. Discharge asymptomatic patients after 6 hours.

## **SELECTIVE-SEROTONIN INHIBITORS**

## **REUPTAKE**

### **Clinical Features**

Signs and symptoms may include vomiting, sedation, tremor, sinus tachycardia, mydriasis, seizures, diarrhea, and hallucinations. Sinus bradycardia is more common with fluvoxamine than with other SSRIs. QRS and QT interval prolongation has been reported in citalopram ingestions.



## Emergency Department Care and Disposition

Supportive care is generally sufficient.

1. Establish IV access and initiate cardiac monitoring.
2. Single-dose **activated charcoal** is appropriate for most ingestions.
3. **Benzodiazepines** are recommended for management of seizures.
4. Observe patients for at least 6 hours. Admit patients who are tachycardic, lethargic, or have conduction abnormalities on ECG 6 hours after ingestion.

## SEROTONIN/NOREPINEPHRINE REUPTAKE INHIBITORS

### Clinical Features

Serotonin/norepinephrine reuptake inhibitors (SNRIs) include venlafaxine, duloxetine, and desvenlafaxine. Overdose may cause hypertension, diaphoresis, tremor, mydriasis, sedation, and seizures. ECG changes include sinus tachycardia and QRS or QT interval widening.

### Emergency Department Care and Disposition

There are no established guidelines for treating SNRI overdoses.

1. Initiate peripheral IV access and cardiac monitoring.
2. Single-dose **activated charcoal** is appropriate in most cases. **Gastric lavage** may be beneficial with early presentation after large ingestions.
3. **Benzodiazepines** are the anticonvulsants of choice.
4. Treat hypotension with fluids and a direct-acting  $\alpha$ -agonist.
5. All patients require at least 6 hours of observation, longer for those ingesting extended-release preparations. Admit symptomatic patients to a monitored bed.

# SEROTONIN SYNDROME

Serotonin syndrome is a potentially life-threatening adverse reaction to serotonergic medications. It can be produced by any drug or combination of drugs that increase central serotonin neurotransmission, most commonly antidepressants.

## Clinical Features

Signs and symptoms are altered mental status, hyperthermia, seizures, and increased muscle tone, in particular myoclonus. Hyperthermia is the most common cause of death.

## Diagnosis

Symptoms are nonspecific, and there are no confirmatory laboratory tests. Diagnostic criteria emphasize exposure to a serotonergic drug and presence of myoclonus.

## Emergency Department Care and Disposition

Treatment is supportive. Watch patients for rhabdomyolysis and metabolic acidosis.

1. Endotracheal intubation and ventilatory support may be required in severe cases.
2. Use **benzodiazepines** to decrease discomfort and promote muscle relaxation.
3. The antiserotonergic agent **cyproheptadine** may be given at an initial dose of 4 to 12 milligrams PO, repeated at 2 hours intervals if no response.
4. Admit all patients until symptoms resolve. Severely ill patients require admission to an ICU. Discontinue serotonergic drugs.

# MONOAMINE OXIDASE INHIBITORS

Monoamine oxidase inhibitors (MAOIs) are used to treat refractory depression. They cause accumulation of neurotransmitters such as norepinephrine in presynaptic nerve terminals and increased systemic availability of dietary amines, such as tyramine. MAOIs can lead to fatal food and drug interactions and cause severe toxicity in overdose.

## Clinical Features

Symptoms include headache, agitation, palpitations, and tremor. Signs include sinus tachycardia, hyperreflexia, fasciculations, mydriasis, hyperventilation, nystagmus, flushing, muscle rigidity, and hypertension. Coma, seizures, brady-cardia, hypotension, hypoxia, and hyperthermia may develop. Death usually results from multiorgan failure.

## Diagnosis and Differential

Diagnosis is made on clinical grounds. Laboratory tests can identify complications, including rhabdomyolysis, renal failure, hyperkalemia, metabolic acidosis, and disseminated intravascular coagulation. The differential diagnosis includes drugs and conditions that produce a hyperadrenergic state, altered mental status, and/or muscle rigidity.

## Emergency Department Care and Disposition

Treatment consists of supportive care and management of complications.

1. Obtain IV access, and initiate cardiac rhythm monitoring.
2. Give all patients **activated charcoal**. Gastric lavage is recommended if it can be performed within 1 hour of ingestion.

3. Hypertension may be treated with **phentolamine**, 2.5 to 5.0 milligrams IV every 10 to 15 min, followed by an infusion. Alternative agents are **nitroprusside** or **fenoldopam**.
4. **Nitroglycerin** is indicated for anginal chest pain and signs of myocardial ischemia.
5. Treat hypotension with isotonic IV fluid boluses, followed by **norepinephrine**.
6. Treat ventricular dysrhythmias with **lidocaine** or **procainamide**.
7. Treat bradycardia with **atropine**, **isoproterenol**, **dobutamine**, and **pacing**.
8. Treat seizures with **benzodiazepines**. General anesthesia and muscle paralysis using vecuronium, with ongoing EEG monitoring, may be necessary.
9. Treat hyperthermia with benzodiazepines to reduce muscle rigidity plus cooling measures. Chemical paralysis with a nondepolarizing agent may be needed for severe rigidity. **Dantrolene**, 1.0 to 2.5 milligrams/ kilogram IV every 6 hours, is another option.
10. Patients who have ingested >1 milligram/kilogram require ICU admission. Those who have ingested less can be admitted to a monitored bed. Observe asymptomatic patients for 24 hours.

## ANTIPSYCHOTICS

Antipsychotics are used to treat psychosis, agitation, nausea, headaches, hiccups, and involuntary motor disorders. Their action involves blockade of dopamine receptors.

### Clinical Features

Central nervous system effects include lethargy, ataxia, dysarthria, confusion, and coma. Seizures are more common with loxapine and clozapine. Anticholinergic toxicity may be seen. Cardiovascular manifestations include orthostatic hypotension; sinus tachycardia; PR, QRS,

and QT interval prolongation; ST and T wave abnormalities; and right axis deviation of the terminal 40 milliseconds of the QRS complex.

Neuroleptic malignant syndrome is a rare but potentially fatal idiosyncratic reaction to antipsychotic agents. It presents with fever, muscular rigidity, autonomic dysfunction, and altered mental status ([Table 102-1](#)). Death results from complications of muscle rigidity, such as rhabdomyolysis, renal failure, or cardiac or respiratory failure.

	Caroff and Mann <sup>1</sup>	Levenson <sup>2</sup>	American Psychiatric Association <sup>3</sup>
<b>Major criteria</b>	Fever >38°C (100.4°F) Muscle rigidity	Fever Muscle rigidity Elevated CK level	Fever Muscle rigidity
<b>Minor criteria</b>	Change in mental status Tachycardia Hypertension or hypotension Tachypnea or hypoxia Diaphoresis or salivary Tremor Incontinence Increased CK level or myoglobinuria Leukocytosis Metabolic acidosis	Tachycardia Abnormal blood pressure Tachypnea Leukocytosis Diaphoresis Altered mental status	Diaphoresis Dysphagia Tremor Incontinence Altered mental status Miosis Tachycardia Labile blood pressure Leukocytosis Elevated CK level
<b>Diagnostic requirement</b>	Both major and at least five minor criteria must be present, and treatment with an antipsychotic must have been within 7 d of symptom onset (or 2 to 4 weeks with a depot agent).	All 3 major criteria or 2 major and 4 minor criteria must be present.	Both major and at least 2 minor criteria must be present.

Key: CK = creatine kinase.

<sup>1</sup>Caroff SH, Mann SC: Neuroleptic malignant syndrome. *Med Clin North Am* 77:185, 1993.

<sup>2</sup>Levenson J: Neuroleptic malignant syndrome. *Am J Psychiatry* 142:1157, 1985.

<sup>3</sup>American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed, text revision. Washington DC, American Psychiatric Association, 2000, pp. 795-798.

**TABLE 102-1** Diagnostic Criteria for Neuroleptic Malignant Syndrome

## **Diagnosis**

Diagnostic studies should include a complete blood count, basic chemistries, a pregnancy test for women of childbearing age, and an ECG. Also obtain a creatine kinase level and liver function tests in patients with neuroleptic malignant syndrome.

## **Emergency Department Care and Disposition**

Treatment is largely supportive.

1. Establish IV access, and initiate cardiac rhythm monitoring.
2. Ventilatory support may be necessary for patients with respiratory depression.
3. Treat seizures with a **benzodiazepine**.
4. Treat hypotension with fluid resuscitation and **norepinephrine**.
5. Treat intraventricular conduction delay and ventricular dysrhythmias with IV **sodium bicarbonate**. Lidocaine is an alternative for ventricular dysrhythmias.
6. Patients with a QTc interval of  $> 500$  milliseconds are at increased risk for torsades de pointes. Treat with **magnesium sulfate**, 2 to 4 grams IV over 10 min.
7. Treatment of neuroleptic malignant syndrome includes cooling measures and benzodiazepines to reduce muscle rigidity. Intubation and paralysis with a nondepolarizing agent may facilitate cooling. Consider **dantrolene** (1.0 to 2.5 milligrams/kilogram IV load) when muscle rigidity is pronounced.
8. Observe patients for 6 hours postingestion. Admit symptomatic patients to a monitored bed. Patients with neuroleptic malignant syndrome warrant ICU admission.

# LITHIUM

Lithium is used to treat bipolar disorder and mania. Toxicity results from overdose or altered renal clearance. Death is usually from respiratory or cardiac failure.

## **Clinical Features**

Patients may present with muscle fasciculations or weakness, ataxia, agitation, peripheral neuropathy, lethargy, or coma. Acute renal failure may be noted, especially in the elderly and those with preexisting renal impairment, diabetes, hypertension, or dehydration. Gastrointestinal symptoms are common in acute and chronic toxicity. Cardiac abnormalities are more common in acute toxicity and include conduction disturbances and ventricular dysrhythmias.

## **Diagnosis**

Acute overdose causes more GI than neurologic toxicity. Serum lithium levels may not correlate well with symptoms. Patients with chronic toxicity display more neurologic effects. In this setting, serum lithium levels correlate better with toxicity. Acute-on-chronic ingestions have aspects of both. A low or negative anion gap, elevated osmolar gap, and severe leukopenia may be noted. ECG abnormalities include QT interval prolongation and ST and T wave changes.

## **Emergency Department Care and Disposition**

Stabilization includes securing the airway and ventilatory and hemodynamic support.

1. Obtain IV access, and initiate cardiac rhythm and ECG monitoring.



2. Initial laboratory studies should include renal function tests, electrolyte levels, complete blood count, and serum levels of lithium and other possible ingestants.
3. Treat seizures with IV **benzodiazepines**, followed by **phenobarbital**.
4. Activated charcoal does not bind lithium but may be helpful for other ingestions.
5. Consider **gastric lavage** with a large-bore tube for ingestions > 4 grams if it can be performed within 1 hour of exposure. Whole-bowel irrigation may be helpful, especially for sustained-release lithium products.
6. IV administration of normal saline is important. In most adults, a 2-L IV bolus is given over 30 to 60 min followed by a 200-mL/h continuous infusion.
7. Indications for **hemodialysis** are serum lithium levels of > 4 mEq/L in acute overdose, 3.5 mEq/L in chronic toxicity, or no change in lithium level after 6 hours of IV normal saline. Patients with renal failure, rising lithium levels, and those who have ingested sustained-release preparations may also benefit. The goal of dialysis is a lithium level <1 mEq/L.
8. Monitor patients with acute ingestions for 4 to 6 hours. Admit those with lithium levels > 1.5 mEq/L and patients who have ingested a sustained-release preparation. Treat patients with mild chronic toxicity with IV normal saline for 6 to 12 hours, and discharge or refer for psychiatric evaluation once their lithium level decreases to <1.5 mEq. Admit patients with more severe chronic toxicity.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 171, "Cyclic Antidepressants," by Kirk C. Mills; Chapter 172, "Atypical Antidepressants, Serotonin Reuptake Inhibitors, and Serotonin Syndrome," by Kirk C. Mills and Keenan M. Bora; Chapter 173, "Monoamine Oxidase Inhibitors," by Kirk C. Mills; Chapter 174, "Antipsychotics," by Michael Levine and Frank LoVecchio; and Chapter 175, "Lithium," by Sandra M. Schneider and Daniel J. Cobaugh.

# CHAPTER 103

## Sedatives and Hypnotics

L. Keith French

Sedative and hypnotic medications are commonly used pharmaceuticals. The three classes include barbiturates, benzodiazepines, and nonbenzodiazepines (buspirone, carisoprodol, meprobamate, chloral hydrate,  $\gamma$ -hydroxybutyrate, melatonin, ramelteon, zaleplon, zolpidem, and zopiclone).

### BARBITURATES

#### Clinical Features

Among the sedative-hypnotic class of medications, barbiturates are associated with the greatest morbidity and mortality. Owing to safer alternatives for seizure management, the clinical use of barbiturates has declined.

Barbiturates cause a dose-dependent spectrum of neuronal depression. With mild to moderate ingestions, toxicity resembles that of ethanol or other sedative-hypnotic medications: confusion, ataxia, slurred speech, drowsiness, and disinhibition. Gastrointestinal motility may be slowed. Severe intoxication follows a 10-fold overdose and loss of deep tendon and corneal reflexes may occur. Hypothermia, hypotension, and respiratory depression are common. Complicating features of the toxicity include hypoglycemia, aspiration pneumonia, pulmonary edema, and acute lung injury.

Barbiturate withdrawal syndrome may occur in the habituated patient who suddenly stops taking their medication. The syndrome occurs within 24 hours of cessation and begins with mild symptoms, which may become severe over the next 2 to 8 days. Short-acting barbiturates generally cause a more robust withdrawal syndrome than the long-acting products. Minor

symptoms include anxiety, restlessness, depression, insomnia, anorexia, nausea, and vomiting. Major symptoms include psychosis, hallucinations, delirium, generalized seizures, hyperthermia, and cardiovascular collapse. Barbiturate withdrawal has a high mortality and gradual inpatient withdrawal of the addicting agent is recommended.

## **Diagnosis and Differential**

Serum barbiturate levels may help establish an etiology for altered mental status in a comatose patient; however, decisions regarding management are based primarily on clinical grounds. Mixed sedative-hypnotic ingestions may be inappropriately ascribed to the barbiturate alone. Due to variability among patients with barbiturate overdoses, heart rate, pupil size and reactivity, and nystagmus are not clinically distinguishing signs. Skin bullae are rarely evident and are not specific to barbiturates. Myocardial depression is more common with barbiturates than benzodiazepines.

Bedside glucose measurement is imperative in the patient with altered mental status and may help narrow the differential diagnosis. Other useful diagnostic testing include arterial blood gas analysis, liver function tests, urine toxicology screen, salicylate and acetaminophen concentrations, blood urea nitrogen and creatinine levels, complete blood count, and creatinine phosphate kinase.

## **Emergency Department Care and Disposition**

Treatment begins with airway management and supportive care. Once pulmonary and cardiovascular function have been adequately assessed and stabilized, enhancing elimination can be considered.

1. Stabilize the airway. **Endotracheal intubation** in the severely poisoned patients is commonly required and should be initiated early in the ED course. Due to the potential for myocardial depression, place 2 large-bore IVs and initiate fluid resuscitation with isotonic saline for hypotension.

2. Consider empiric treatment with naloxone and thiamine early in the management.
3. **Dopamine** or **norepinephrine** may be needed if fluid boluses fail to reverse hypotension. Hypothermia between 30°C (86°F) and 36°C (96.8°F) requires standard rewarming techniques.
4. **Activated charcoal** helps reduce absorption. In the awake, cooperative patient, 50 to 100 grams orally (1 gram/kilogram in children) should be administered. For sedated or unconscious patients, airway protection should precede the administration of activated charcoal. Multidose charcoal will reduce serum levels, but has not been shown to change clinical outcome.
5. Forced diuresis is not indicated due to risks of sodium and fluid overload and a lack of proven efficacy.
6. Urinary alkalization is not considered first-line therapy. While it may enhance the clearance of phenobarbital and primidone, it is less effective than multidose charcoal alone, and has no role in the management of short-acting barbiturates.
7. **Hemodialysis**, hemoperfusion, and hemodiafiltration are reserved for patients who deteriorate despite aggressive medical support, but are only effective with phenobarbital toxicity.
8. Disposition depends on the degree of intoxication: evidence of toxicity greater than 6 hours from time of arrival requires hospital admission. Obtain psychiatric consult for intentional overdose. Toxicology or poison center consultation is recommended to assist with management.

## **BENZODIAZEPINES**

### **Clinical Features**

Benzodiazepine overdoses are common but carry a low mortality rate in isolation. There is, however, variation in the clinical outcome among agents due to differences in potency. Parenteral administration in the ED may produce significant complications. Benzodiazepines, when mixed with

other sedative-hypnotics, may produce profound toxicity including respiratory depression, hypotension, and death.

The primary effect of benzodiazepines is neurologic and is characterized by somnolence, dizziness, slurred speech, confusion, ataxia, incoordination, and general impairment in intellectual function. Neurologic effects in the elderly, very young, or malnourished may be prolonged or enhanced. Disinhibition, extrapyramidal reactions, and paradoxical excitation are uncommon but reported. Short-term, anterograde amnesia is a common, sometimes desirable, effect with the administration of certain benzodiazepines.

Chronic use of benzodiazepines is associated with physiologic addiction. Withdrawal from benzodiazepines may occur following abrupt cessation. Symptoms are more intense following withdrawal from short acting agents. Clinical findings may mimic alcohol withdrawal and include anxiety, irritability, insomnia, nausea, vomiting, tremor, and sweating. Serious manifestations include hallucinations, psychosis, disorientation, and seizures. Treatment begins with reintroduction of a benzodiazepine with subsequent, gradual tapering.

## **Diagnosis and Differential**

There is limited value in toxicological testing as serum levels do not correlate well with clinical findings. Qualitative urine screening is unreliable and a positive test does not prove causation of clinical signs as clinical features are nonspecific and may be seen with overdose of any sedative-hypnotic.

## **Emergency Department Care and Disposition**

1. Priorities include assessment and stabilization of the airway, breathing, and circulation (see the preceding section on barbiturates for guidance regarding initial management, resuscitation and laboratory monitoring).
2. Do not induce emesis. **Activated charcoal** (1 gram/kilogram in children or 25 to 50 grams in adults) will bind benzodiazepines. Exercise caution

in the sedated patient, and secure the airway before administration. Multidose charcoal is not indicated. Gastric lavage, forced diuresis, enhanced elimination, hemodialysis and hemoperfusion are ineffective and unnecessary.

3. **Flumazenil** is a unique, selective antagonist of the central effects of benzodiazepines. Unlike naloxone, flumazenil should not be used empirically for the undifferentiated sedative-hypnotic toxidrome as it may precipitate seizures ([Table 103-1](#)). The ED applications of flumazenil are limited to the setting of respiratory depression following procedural sedation with benzodiazepines. The dose is 0.2 milligram IV and titrated every min to a maximum total dose of 3 (0.01-0.02 milligram/ kilogram in children). The half-life of flumazenil is approximately 1 hour and rebound sedation can occur in the setting of overdose with long-acting benzodiazepines.

Overdose of unknown agents Suspected or known physical dependence on benzodiazepines Suspected cyclic antidepressant overdose Coingestion of seizure-inducing agents Known seizure disorder Suspected increased intracranial pressure
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**TABLE 103-1** Contraindications to Flumazenil

4. In general, care is supportive. Admit patients with significant alterations of mental status, respiratory depression, or hypotension. Consultation with mental health specialists may be appropriate.

## **NONBENZODIAZEPINE HYPNOTICS**

## **SEDATIVE-**

### **Clinical Features**

The hallmark of all sedative-hypnotic medications, regardless of subclass, is sedation. Exposure to nonbenzodiazepine agents is common and coingestion with other sedatives may be synergistic and produce profound

sedation. Three agents, ethchlorvynol, glutethimide, and methaqualone, have recently been removed from the markets in the United States and Canada.

### *Buspirone*

Buspirone has a complex mechanism of action and rapid absorption. Side effects include sedation, GI distress, vomiting, and dizziness. The symptoms with overdose are exaggerations of the side effects noted with therapeutic dosing. The drug is generally well tolerated in overdose, and treatment is primarily supportive. Due to effects on the serotonergic system, serotonin syndrome has been reported. Seizures, hypotension, priapism, and dystonia are rare complications.

### *Carisoprodol and Meprobamate*

Carisoprodol and its active metabolite, meprobamate, are used as central-acting muscle relaxants and anxiolytics, respectively. In overdoses, both may cause sedation, coma, and cardiopulmonary depression. Carisoprodol, but not meprobamate, can cause myoclonus, which may be a clue in an unknown overdose. Meprobamate has been associated with pharmacobezoars, and may be a cause of prolonged toxicity.

### *Chloral Hydrate*

Chloral hydrate is the oldest sedative-hypnotic available today. At therapeutic doses, chloral hydrate produces mental status depression without loss of airway and respiratory reflexes. Vomiting and paradoxical excitation can occur in a small percentage of children. In overdose, coma and respiratory depression can occur. Chloral hydrate is a myocardial sensitizer and cardiac arrhythmias, decreased cardiac contractility, and asystole have been reported. When combined with alcohol, a potent, “knock-down” cocktail known as a “Mickey Finn” is created. A withdrawal syndrome similar to ethanol has been described.

Chloral hydrate may produce a characteristic pear-like odor. Abdominal radiographs may aid in the diagnosis of pharmacobezoars, as chloral

hydrate is radiopaque. Respiratory depression and coma are treated supportively. **Treat ventricular arrhythmias with IV  $\beta$ -blockers.**

### *$\gamma$ -Hydroxybutyrate*

$\gamma$ -Hydroxybutyrate (GHB) has been marketed as a muscle builder, fat burner, antidepressant, anxiolytic, hypnotic, and cholesterol-lowering medication. The only approved use in the United States is for the treatment of narcolepsy. GHB has a narrow therapeutic window and may produce a range of toxicity from mild sedation to coma. Seizures, bradycardia, hypothermia, and cardiac depression may occur. Rapid sedation with abrupt recovery 6 to 12 hours later is a common feature with GHB. Due to its amnestic effects and rapid sedation, GHB has been illicitly used for drug-facilitated sexual assault. Two compounds, 1,4 butanediol and  $\gamma$ -butyrolactone are GHB precursors and have been abused to produce similar effects. Treatment is primarily supportive with focus on airway management. Rapid awaking and self-extubation may be a clue to GHB intoxication. Toxicologic detection of GHB is difficult owing to its very short half-life and rapid elimination. Withdrawal from GHB mimics alcohol withdrawal and may be severe, lasting from 3 days to 2 weeks.

### *Melatonin*

The endogenous hormone melatonin is secreted by the pineal gland and is believed to help regulate the sleep-wake cycle. Melatonin can be purchased without prescription. Side effects following therapeutic dosing include headache, dizziness, fatigue, and irritability. Overdose data are limited, and signs and symptoms exaggerate the side effects from therapeutic doses.

### *Ramelteon*

Ramelteon is a relatively new medication used to treat insomnia. It binds to melatonin receptors in the brain. Absorption following oral dosing is rapid. In overdose, sedation is common and treatment is supportive. Abuse and withdrawal have not been reported.



### *Zolpidem, Zaleplon, and Zopiclone*

Zolpidem, zaleplon, and zopiclone have gained increased popularity for the treatment of insomnia. Though initially thought to produce little or no psychomotor impairment, addiction or withdrawal, experience has proved otherwise. Side effects in therapeutic doses include nausea and somnolence. Vivid dreams, sleep-walking, and driving have been reported with zolpidem. Fatalities following zolpidem overdoses have been reported, but are usually associated with mixed ingestions. There are limited data to guide management in overdoses of zaleplon and zopiclone.

### **Emergency Department Care and Disposition**

1. In general, management for the nonbenzodiazepines is supportive (see the preceding section on benzodiazepines for treatment priorities). Flumazenil is ineffective for these medications.
2. For ventricular arrhythmias in the setting of chloral hydrate intoxication, **IV  $\beta$ -blockers** (e.g. propranolol, 1 milligram IV or 0.01 to 0.1 milligram/kilogram IV in children) are first-line agents.
3. Disposition is largely guided by the degree of symptoms. Have a low threshold for admission of any patient with altered mental status, abnormal vital signs, or arrhythmias. Consult psychiatric services when appropriate.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 176, “Barbiturates,” by Chip Gresham and Frank LoVecchio; Chapter 177, “Benzodiazepines,” by Dan Quan; and Chapter 178, “Nonbenzodiazepines,” by Michael Levine and Dan Quan.

# **CHAPTER 104**

## **Alcohols**

Michael P. Kefer

All alcohols are potentially toxic and cause clinical inebriation and an anion gap metabolic acidosis. Ethanol and isopropanol are the most commonly ingested alcohols and cause direct toxicity, while methanol and ethylene glycol cause toxicity as a result of conversion to toxic metabolites.

## **ETHANOL**

Although acute ethanol intoxication may cause death directly from respiratory depression, morbidity and mortality are usually related to trauma from impaired cognitive function. On an average, nondrinkers eliminate ethanol from the bloodstream at a rate of 15 to 20 milligrams/dL/h and chronic drinkers at about 30 milligrams/dL/h.

### **Clinical Features**

Signs and symptoms of ethanol intoxication include slurred speech, disinhibited behavior, central nervous system (CNS) depression, and altered coordination. Manifestations of serious head injury or hypoglycemia may be identical to, or clouded by, ethanol intoxication. Nystagmus and a characteristic odor of ethanol may be observed.

### **Diagnosis and Differential**

Check a bedside glucose in all patients with altered mental status. Serum ethanol levels will confirm ethanol intoxication but are not required for mild to moderate intoxication. Consider comorbid disease or injury in the

inebriated patient and obtain additional labs as indicated: electrolytes may demonstrate an anion gap acidosis; liver enzymes may reveal hepatic damage; a urine toxicology screen may reveal coingestion of other drugs of abuse. Obtain imaging as indicated by external signs of trauma in the inebriated patient.

## **Emergency Department Care and Disposition**

1. The mainstay of treatment is observation. A careful physical examination should be performed to evaluate for complicating injury or illness.
2. Treat hypoglycemia with IV dextrose. **Thiamine** 100 milligrams IV or IM may be given concurrently if Wernicke encephalopathy is suspected.
3. Consider secondary causes of deterioration or lack of improvement during observation and manage accordingly.
4. Discharge the patient once sober enough to pose no threat to self or others.

## **ISOPROPANOL**

Isopropanol is commonly found in rubbing alcohol, solvents, skin and hair products, paint thinners, and antifreeze. Acetone is the principle toxic metabolite.

### **Clinical Features**

Clinically, isopropanol intoxication is similar to that of ethanol but lasts longer with deeper CNS depression. The smell of rubbing alcohol or the fruity odor of ketones may be noted on the patient's breath. Severe poisoning presents as coma, respiratory depression, and hypotension. Hemorrhagic gastritis is common and causes nausea, vomiting, abdominal pain, and upper gastrointestinal bleeding.

### **Diagnosis and Differential**

Check a bedside glucose in all patients with altered mental status. Classic isopropanol toxicity is associated with an elevated osmolal gap, ketonemia and ketonuria, without acidosis. In the setting of upper GI bleeding, coagulation studies, a CBC, and a type and screen should be obtained. When available, a serum isopropanol and acetone level confirm the diagnosis.

## **Emergency Department Care and Disposition**

1. Treat hypotension with aggressive infusion of IV crystalloids though persistent hypotension may require vasopressors. Treat significant bleeding from hemorrhagic gastritis with transfusion of packed red blood cells and plasma as indicated.
2. Do **not** administer metabolic blockade with Fomepizole or ethanol since acetone, the metabolite of isopropanol, is no more toxic than the parent compound.
3. Hemodialysis is indicated for refractory hypotension or an isopropanol level >400 milligrams/dL. Hemodialysis removes both isopropanol and acetone.
4. Patients with prolonged CNS depression require admission. Those who are asymptomatic after 6 to 8 hours of observation can be discharged or referred for psychiatric evaluation if indicated.
5. Charcoal does not bind alcohols, and is useful only if there is coingestion of an absorbable substance.

## **METHANOL AND ETHYLENE GLYCOL**

Methanol is a common solvent in paint products, windshield washing fluid, and antifreeze. Ethylene glycol is found in coolants, polishes, and detergents. Toxicity from these alcohols results from the formation of toxic metabolites, which produce a significant anion gap metabolic acidosis. Methanol leads to the formation of toxic formaldehyde and formic acid,

while ethylene glycol is metabolized into the toxic compounds glycolic and glyoxylic acid.

## **Clinical Features**

Symptoms of methanol toxicity may not appear for 12 to 24 hours after ingestion until toxic metabolites accumulate. Time to symptom onset may be longer if ethanol is consumed, as ethanol inhibits methanol metabolism. Signs and symptoms include CNS depression, visual disturbances (classically, a complaint of looking at a snowstorm), abdominal pain, nausea, and vomiting. The gastrointestinal symptoms may be due to mucosal irritation or pancreatitis. Funduscopic examination may reveal retinal edema or hyperemia of the optic disk.

Ethylene glycol poisoning often exhibits three distinct clinical phases after ingestion. First, within 12 hours, CNS effects predominate: the patient appears intoxicated without the odor of ethanol on the breath. Second, 12 to 24 hours after ingestion, cardiopulmonary effects predominate: elevated heart rate, respiratory rate, and blood pressure are common. Congestive heart failure, respiratory distress syndrome, and circulatory collapse may develop. Third, 24 to 72 hours after ingestion, renal effects predominate which are characterized by flank pain, costovertebral angle tenderness, and acute tubular necrosis with acute renal failure. Hypocalcemia may result from precipitation of calcium oxalate into tissues leading to tetany and typical ECG changes.

## **Diagnosis and Differential**

The diagnosis is based on clinical presentation and laboratory findings of an anion gap metabolic acidosis (which may take hours to develop) with elevated levels of methanol or ethylene glycol. An elevated osmolal gap is present and useful when immediate methanol or ethylene glycol testing is not available. Basic laboratory investigations include a bedside glucose, CBC, BMP, arterial blood gas, urinalysis, and methanol or ethylene glycol level. Ethylene glycol poisoning differs from methanol poisoning in that

visual disturbances and fundoscopic abnormalities are absent and calcium oxalate crystals are present in the urine.

The differential diagnosis includes other causes of an anion gap metabolic acidosis such as salicylate or isoniazid toxicity, diabetic ketoacidosis, alcoholic ketoacidosis, uremia, and lactic acidosis.

## Emergency Department Care and Disposition

Treatment is based on metabolic blockade and removing toxic metabolites from the body. Both fomepizole and ethanol have a greater affinity for alcohol dehydrogenase than methanol and ethylene glycol. Indications for metabolic blockade are listed in [Table 104-1](#).

1. Documented plasma methanol or ethylene glycol concentration of >20 milligrams/dL
2. If methanol or ethylene glycol level not immediately available: A. Documented or suspected significant methanol or ethylene glycol ingestion with ethanol level lower than approximately 100 milligrams/dL* B. Coma or altered mental status in patient with unclear history and: (1) Unexplained serum osmolar gap of >10 mOsm/L <i>or</i> (2) Unexplained metabolic acidosis <i>and</i> ethanol level of <100 milligrams/dL*

\*If serum ethanol level is >100 milligrams/dL, patient will be protected from the formation of toxic metabolites by coingestion of ethanol and specific metabolic blockade treatment can be delayed until toxic alcohol level is available. However, if ethanol level is likely to fall to <100 milligrams/dL, metabolic blockade treatment should be initiated.

**TABLE 104-1** Indications for Metabolic Blockade with Fomepizole or Ethanol

1. Administer **fomepizole** 15 milligrams/kilogram IV load followed by 10 mg/ kilogram every 12 hours. Fomepizole is a potent inhibitor of alcohol dehydrogenase with greater affinity and fewer side effects than ethanol. If fomepizole is not available, or the patient is allergic, use **ethanol** 800 milligrams/kilogram IV load, followed by a continuous infusion of 100 milligrams/kilogram/h in the average drinker and 150 milligrams/kilogram/h in the heavy drinker. Adjust the infusion accordingly to maintain a blood ethanol level at 100 to 150 milligrams/dL. If resources are limited, oral therapy with commercial **80 proof liquor** can be initiated. A load of 3 to 4 oz with maintenance of 1 to 2 oz/h is a typical dose for a 70 kilograms patient.

2. Monitor serum **glucose** during treatment with ethanol as hypoglycemia may be induced, especially in children. Treat hypoglycemia with 1 mL/kg 50% dextrose in water in adults and 4 mL/kg 10% dextrose in water in children.
3. **Dialysis** eliminates both methanol and ethylene glycol and their toxic metabolites. Indications are listed in [Table 104-2](#). Fomepizole or ethanol treatment do not alter the indications for dialysis; however, both fomepizole and ethanol are dialyzed and, therefore, increase the dosing interval of fomepizole to every 4 hours. Double the infusion rate of ethanol during dialysis and adjust accordingly to maintain the level at 100 to 150 milligrams/dL.

Refractory metabolic acidosis: pH <7.25 with anion gap >30 mEq/L and/or base deficit less than -15  
 Visual abnormalities\*  
 Renal insufficiency  
 Deteriorating vital signs despite aggressive supportive care  
 Electrolyte abnormalities refractory to conventional therapy  
 Serum methanol or ethylene glycol level of >50 milligrams/dL<sup>†</sup>

\*Applies only to methanol; visual abnormalities may not resolve immediately, so their persistence in the absence of other indications once hemodialysis is started is *not* an indication for continued hemodialysis.

<sup>†</sup>Although previously considered an indication for hemodialysis, there are reports of patients with levels of > 50 milligrams/dL successfully treated with fomepizole with or without bicarbonate and no hemodialysis.

**TABLE 104-2** Indications for Urgent Hemodialysis After Methanol or Ethylene Glycol Ingestion

4. Continue dialysis, fomepizole, or ethanol treatment until the methanol or ethylene glycol level is <20 milligrams/dL and the metabolic acidosis has resolved.
5. In methanol poisoning, administer **folate** 50 milligrams IV. In ethylene glycol poisoning, administer **pyridoxine** 100 milligrams IV and **thiamine** 100 milligrams IV.
6. Administer sodium bicarbonate 1 to 2 mEq/kg and titrated to maintain a normal pH in methanol toxicity to increase renal excretion of formic acid.
7. Treat documented and symptomatic hypocalcemia in ethylene glycol toxicity with calcium gluconate or calcium chloride.
8. Consult a medical toxicologist or regional poison control center to aid in the management of symptomatic methanol or ethylene glycol ingestion.

9. Patients with suspected ethylene glycol ingestion who are asymptomatic after 6 hours with no ethanol detected and no osmolar gap or metabolic acidosis may be safely discharged. Since methanol toxicity and coingestion of ethanol may result in delayed symptoms, these patients should be observed for a minimum of 12 hours. Patients with significant signs and symptoms should be admitted to an intensive care unit.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 179, "Alcohols," by Jennifer C. Smith and Dan Quan.



# CHAPTER 105

## Drugs of Abuse

Jeanmarie Perrone

### OPIOIDS

The term *opioid* refers to any drug that is active at the opioid receptor; while *opiates* refers to naturally occurring derivatives of the opium plant, morphine, and codeine. *Narcotic* is a legal term and generically refers to any drug that causes sedation. Emergency physicians commonly utilize opioids as analgesics and must be familiar with the range and route of opioid dosing, as well as the appropriate dose and indications for the antidote naloxone to reverse excess opioid effects.

#### Clinical Features

Opioid overdose produces a clinical toxidrome: pinpoint pupils, respiratory depression, and lethargy. Although each opioid may produce slightly varied manifestations of this toxidrome depending on the drug, dose, and tolerance, the degree of respiratory depression is the primary effect requiring emergency intervention. Heroin overdose may be associated with acute lung injury and ARDS. Histamine release from opioids can cause urticaria and bronchospasm, and other clinical effects include ileus and urinary retention. Opioid withdrawal is manifest by nausea, vomiting, diarrhea, dysphoria, piloerection, lacrimation and gooseflesh.

#### Diagnosis and Differential

The diagnosis is clinical. Nonopioid sympatholytics, such as clonidine, appear to act near the opioid receptor and produce varying degrees of

miosis, altered mentation, and respiratory depression, and mimic opioid intoxication. Because response to naloxone with these agents is less reliable, consider clonidine intoxication in patients who appear opioid poisoned but do not respond to naloxone. Other possible causes of a decreased response to naloxone include mixed opioid agonist/antagonists, such as buprenorphine, and super potent opioids, such as fentanyl derivatives.

## **Emergency Department Care and Disposition**

1. Naloxone is the primary treatment for respiratory depression. Administer 2 milligrams IV, SC, or IM initially for apnea, 0.4 milligram for opioid-dependent patients with respiratory depression, and 0.05 milligram to opioid-dependent patients to avoid precipitating withdrawal. The pediatric dose is 0.01 milligram/kilogram.
2. In large overdoses, consider an infusion of naloxone: two-thirds of the dose required to initially “wake up” the patient per hour.
3. Consider endotracheal intubation in patients who respond poorly to naloxone and those with acute lung injury from overdose.
4. Patients with short-acting opioids, such as heroin, who are awake and asymptomatic 2 to 3 hours after the last naloxone dose can be discharged. Symptomatic patients with exposure to long-acting opioids (sustained release morphine or oxycodone) require prolonged observation and admission.

## **COCAINE, METHAMPHETAMINE, AND OTHER STIMULANTS**

Cocaine and methamphetamine produce similar clinical manifestations, but have regional differences in prevalence.

### **Clinical Features**

Cocaine and methamphetamine induce euphoria and produce complications secondary to the release of catecholamines. Onset of effect via intranasal, inhalational (crack use), and intravenous use is rapid. Repeated drug administration leads to prolonged effects and increased toxicity. Symptoms of sympathomimetic overdose include hypertension, tachycardia, diaphoresis, and agitation. Complications include dysrhythmias, myocardial ischemia, aortic rupture, aortic and coronary artery dissection, seizures, intracranial hemorrhage, hyperthermia, rhabdomyolysis, and acute renal failure, which can be life threatening.

“Cocaine chest pain” is a common ED complaint and may manifest with electrocardiographic changes and hemodynamic complications or with mild tachycardia and chest discomfort. Cardiovascular complications of cocaine may occur even in those without coronary artery disease. Cocaine abuse during pregnancy increases risk for spontaneous abortion, abruptio placentae, fetal prematurity, and intrauterine growth retardation. Crack cocaine use has been associated with bronchospasm, pneumonitis, pulmonary hemorrhage, pulmonary edema, and barotrauma. “Body stuffers” (hasty ingestion of drugs to avoid police) and “body packers” (ingestion of large amounts of tightly packed pure drug for importation) may be asymptomatic or demonstrate signs of severe cocaine toxicity if a bag ruptures. Intestinal ischemia, bowel necrosis, ischemic colitis, gastrointestinal bleeding, and bowel perforation may result.

Mortality from methamphetamine toxicity is most commonly the result of hyperthermia, dysrhythmias, seizures, and hypertension that results in intracranial infarction or hemorrhage and encephalopathy. Stimulants, such as ephedrine and methylphenidate, produce toxic effects similar to those of cocaine and amphetamines. Ephedrine has been linked to significant cardiovascular and neurologic toxicities, psychosis, severe hypertension, and death.

## **Diagnosis and Differential**

Diagnosis of cocaine, amphetamine, or stimulant intoxication is usually clinical. Urine drug screening for cocaine is reliable and can detect exposure

within 72 hours. Urine screens for amphetamines are less specific and have high false negative and false positive results.

Additional laboratory evaluation for intoxicated patients includes a complete metabolic panel to assess acid/base status and creatine kinase (CK) to assess for rhabdomyolysis. The evaluation of altered mental status may include a head CT to exclude intracranial hemorrhage. Consider ECG, chest radiograph, and cardiac enzymes in cocaine- or amphetamine-intoxicated patients presenting with chest pain.

Include traumatic injury and hypoglycemia in the differential diagnosis. Concomitant use of substances such as alcohol or opioids may significantly alter the presentation.

## Emergency Department Care and Disposition

The treatment principals for sympathomimetics are outlined in [Table 105-1](#).

Vital sign monitoring, especially temperature
Benzodiazepines for sedation
Aggressive cooling for hyperthermia (ice immersion)
IV fluids for rhabdomyolysis
Anticonvulsants (benzodiazepines, phenobarbital) for seizures
Phentolamine for uncontrolled hypertension

**TABLE 105-1** Management of Sympathomimetic Toxicity

1. Benzodiazepines are the mainstay of treatment for cardiovascular and CNS effects. Administer **lorazepam** 2 milligrams IV (0.1 milligram/kilogram) for agitation, hypertension, and tachycardia and titrate to effect. Avoid antipsychotic medications, which may precipitate seizures, hyperthermia, and dysrhythmias.
2. Treat seizures with benzodiazepines. **Phenobarbital** (15 to 20 milligrams/kilogram) and neuromuscular blockade with continuous EEG monitoring may be necessary for status epilepticus.
3. Treat cardiac ischemia or infarction with aspirin, nitrates, morphine, and benzodiazepines.  $\beta$ -blockers are contraindicated due to unopposed  $\alpha$ -receptor stimulation. Fibrinolytic therapy should be used with great caution because of the risk of cocaine-associated intracranial hemorrhage.

4. Treat cocaine-induced wide complex tachydysrhythmia and QRS interval prolongation with sodium bicarbonate 1 to 2 mEq/kg titrated to a serum pH of 7.45 to 7.5. Acidification of the urine for amphetamine intoxication is contraindicated.
5. Treat hypertension unresponsive to benzodiazepines with **nitroprusside (0.3 microgram/kilogram/min IV)** or **phentolamine (2.5 to 5.0 milligrams IV)**.
6. Treat asymptomatic “body packers” with whole-bowel irrigation using **polyethylene glycol**. Symptomatic patients with presumed rupture of ingested packets are treated for acute toxicity as above and immediate surgical consultation for possible laparotomy.
7. Patient disposition depends on initial presentation, response to treatment, stimulant involved, and expected duration of effect. Amphetamines have a longer duration of effect than cocaine does; therefore, intoxication may require longer periods of observation or hospital admission. Admit patients with rhabdomyolysis, hyperthermia, or ECG changes consistent with ischemia to intensive care.

## HALLUCINOGENS

### Clinical Features

[Table 105-2](#) summarizes the classification, features, complications, and specific treatments of commonly abused hallucinogens. The hallucinogens with the most significant potential for morbidity include phencyclidine because of the potential for concomitant trauma, and ecstasy (MDMA), which can cause hyperthermia, seizures, and hyponatremia.

Drug	Typical Dose	Duration of Action	Clinical Features	Complications	Specific Treatment
Lysergic acid diethylamide (LSD)	20-80 micrograms	6-12 h	Mydriasis Tachycardia Anxiety Muscle tension	Coma Hyperthermia Cocciopathy Persistent psychosis Hallucinogen persisting perception disorder	Supportive Benzodiazepines Haloperidol
Psilocybin	5-100 mushrooms 4-6 milligrams of psilocybin	4-6 h	Mydriasis Tachycardia Muscle tension Nausea and vomiting	Seizures (rare) Hyperthermia (rare)	Supportive Benzodiazepines
Mescaline	5-12 "buttons" 300-500 milligrams of mescaline	6-12 h	Mydriasis Abdominal pain Nausea and vomiting Dizziness Nystagmus Ataxia	Rare	Supportive Benzodiazepines

Methylenedioxymethamphetamine (MDMA, "Ecstasy")	50-200 milligrams	4-6 h	Mydriasis Bruising Jaw tension Agitation Dry mouth Nausea	Hypotatremia Hypertension Seizures Hyperthermia Arrhythmias Rhabdomyolysis	Benzodiazepines Hydration Active cooling Serotonin antagonists
Phencyclidine (PCP, "angel dust")	1-9 milligrams	4-6 h	Small or mid-sized pupils Nystagmus Muscle rigidity Hypersalivation Agitation Catalepsy	Coma Seizures Hyperthermia Rhabdomyolysis Hypertension Hypoglycemia	Benzodiazepines Hydration Active cooling
Marijuana (cannabis)	5-15 milligrams of tetrahydrocannabinol	2-4 h	Tachycardia Conjunctival injection	Acute psychosis (rare) Panic reactions (rare)	Supportive Benzodiazepines

**TABLE 105-2** Commonly Abused Hallucinogens

## Diagnosis and Differential

Diagnosis is primarily clinical. Routine drug screens will not detect LSD, psilocybin, or mescaline. Urine tests for phencyclidine (PCP) are unreliable. Some amphetamine screens will detect MDMA. Urine tests for marijuana are unreliable indicators of acute use because patients may be positive for days to weeks after their last use. Check glucose, electrolytes, renal function, CK, and urinalysis to evaluate for hyponatremia, rhabdomyolysis, and renal failure.

Exclude other causes of altered mental status, including traumatic injuries, hypoglycemia, and infection in patients with hyperthermia. The differential diagnosis of hallucinogen intoxication includes alcohol and benzodiazepine withdrawal, hypoglycemia, anticholinergic poisoning, thyrotoxicosis, CNS infections, structural CNS lesions, and acute psychosis.

## **Emergency Department Care and Disposition**

Most hallucinogen intoxications are managed by monitoring, providing a calm environment, and the use of benzodiazepines for agitation and sympathomimetic symptoms. Antipsychotic medications should be used with caution as these may lower seizure threshold.  $\beta$ -blockers should not be used to treat tachycardia or hypertension as these can lead to unopposed  $\alpha$ -receptor stimulation.

1. Treat agitation, seizures, tachycardia, and hypertension with **lorazepam** 1 to 2 milligram IV or PO (0.1 milligram/kilogram).
2. Consider **nitroprusside** or **phentolamine** for severe hypertension refractory to benzodiazepines.
3. Treat symptomatic hyponatremia with 3% saline.
4. Treat rhabdomyolysis with aggressive isotonic IV fluid administration.
5. Most patients with hallucinogen intoxication can be safely discharged from the ED after a period of observation. Admit patients with persistent altered mental status or serious medical complications, such as severe hyperthermia, hypertension, seizures, and rhabdomyolysis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 180, “Opioids” by Suzanne Doyon, Chapter 181, “Cocaine Methamphetamine, and other Amphetamines” by Jane M. Prosser and Jeanmarie Perrone, and Chapter 182, “Hallucinogens” by Katherine Prybys and Karen N. Hansen.



# CHAPTER 106

## Analgesics

Joshua N. Nogar

Over-the-counter analgesics, such as salicylates (ASA) and acetaminophen, can result in fatal overdose, but early identification of toxicity and initiation of appropriate treatment can significantly reduce mortality from these exposures. Nonsteroidal anti-inflammatory drug (NSAID) overdoses are rarely fatal and typically require only supportive care.

### ASPIRIN AND SALICYLATES

#### Clinical Features

The features of aspirin (ASA) toxicity are summarized in [Table 106-1](#). Clinical symptoms of acute toxicity include hyperthermia, tachypnea, and altered mental status. Chronic or “therapeutic” (repeated dose) poisonings are generally more serious and associated with higher mortality than acute overdoses, and are typically encountered in elderly patients with multiple medical problems. Chronic toxicity develops at lower drug levels compared to acute overdoses. The duration of symptoms is often prolonged and there may be a delay in diagnosis because the clinical picture is similar to that of infection. Consider chronic salicylism in any patient with unexplained non-focal neurologic and behavioral abnormalities, especially with coexisting acid-base disturbance, tachypnea, dyspnea, or noncardiogenic pulmonary edema. Patients taking carbonic anhydrase inhibitors to treat glaucoma are at increased risk for chronic salicylism.

	Mild	Moderate	Severe
Acute ingestion (dose)	<150 milligrams/kilogram	150 to 300 milligrams/kilogram	>300 milligrams/kilogram
End-organ toxicity	Tinnitus Hearing loss Dizziness Nausea/vomiting	Tachypnea Hyperpyrexia Diaphoresis Ataxia Anxiety	Abnormal mental status Seizures Acute lung injury Renal failure Cardiac arrhythmias Shock

**TABLE 106-1** Severity Grading of Salicylate Toxicity in Adults

In children, acute ASA overdoses generally present within hours of ingestion. Children younger than 4 years of age tend to develop early metabolic acidosis (pH < 7.38), whereas children older than 4 years usually manifest a mixed acid-base disturbance as seen in adults.

## Diagnosis and Differential

ASA toxicity is a *clinical diagnosis* made in conjunction with the patient's acid-base status. Respiratory alkalosis with an anion-gap metabolic acidosis, and hypokalemia are the classic features of this poisoning. ASA blood concentrations correlate poorly with toxicity, and relying on drug levels as a measure of toxicity is the most common pitfall in the management of ASA overdose.

Check bedside glucose determination in all patients with altered mental status. Additional laboratory studies include electrolytes, blood urea nitrogen (BUN), creatinine, complete blood count (CBC), prothrombin time (PT), ASA level, acetaminophen level (to exclude coingestion), and venous blood gas. Hypoglycemia or hyperglycemia may be seen with severe or chronic toxicity.

The differential diagnosis of ASA toxicity includes diabetic ketoacidosis, sepsis, meningitis, acute iron poisoning, caffeine overdose, theophylline toxicity, and Reye syndrome.

## Emergency Department Care and Disposition

1. Institute cardiac monitoring and support the ABCs. Establish intravenous (IV) or intraosseous (IO) access early. Careful airway management is critical in ASA-poisoned patients: a sudden drop in serum pH due to respiratory failure can precipitously worsen ASA toxicity, and careful ventilation guided by acid-base status is essential in the intubated patient. Respiratory acidosis frequently occurs shortly after a mechanical ventilator is set to a "normal" rate and volume parameters, and is typically a premorbid event.
2. Administer **activated charcoal** 1 gram/kilogram PO. **Whole-bowel irrigation** may effectively decontaminate the GI tract in the setting of large overdoses, enteric-coated, or sustained-release preparations.
3. Administer **IV normal saline (NS)** to patients with evidence of volume depletion. During initial resuscitation, monitor urine pH, ASA level, electrolytes, glucose, and acid-base status hourly. Add dextrose to parenteral fluids after initial NS resuscitation. Consider 10% dextrose in the setting of hypoglycemia or neurologic

symptoms. Add potassium 40 mEq/L after establishing adequate urine output (1 to 2 mL/kg/h), if not contraindicated by initial electrolytes and renal function.

4. **Alkalinize** the serum and urine to enhance ASA protein binding and urinary elimination: administer a bolus of 1 to 2 mEq/kg of **sodium bicarbonate**, then add 150 mEq (3 ampules) of sodium bicarbonate to 1 L 5% dextrose in water and infuse at 1.5 to 2.0 times the patient's maintenance rate; adjust the infusion to maintain urine  $\text{pH} > 7.5$  if possible. Bicarbonate may worsen hypokalemia and precipitate arrhythmias.
5. Consider **hemodialysis** for all cases with ASA levels in excess of 100 milligrams/dL. Indications for hemodialysis may be significantly lower in the setting of chronic toxicity (60 to 80 milligrams/dL). Also consider hemodialysis for clinical deterioration despite supportive care and alkalinization, renal insufficiency or failure, severe acid-base disturbance, altered mental status, or adult respiratory distress syndrome. Check serial ASA levels every 2 hours until they begin to fall, then every 4 to 6 hours until the level is nontoxic.
6. Enteric-coated and sustained-release preparations result in delayed peak serum levels (0 to 60 hours postingestion) and their ingestion requires admission for at least 24 hours to ensure declining serial ASA levels and improving clinical status.
7. Discharge a patient from the ED if there is progressive clinical improvement, no significant acid-base abnormality, and a decline in serial ASA levels toward the therapeutic range. In deliberate overdoses, obtain a psychiatric consultation before discharge.

## ACETAMINOPHEN

### Clinical Features

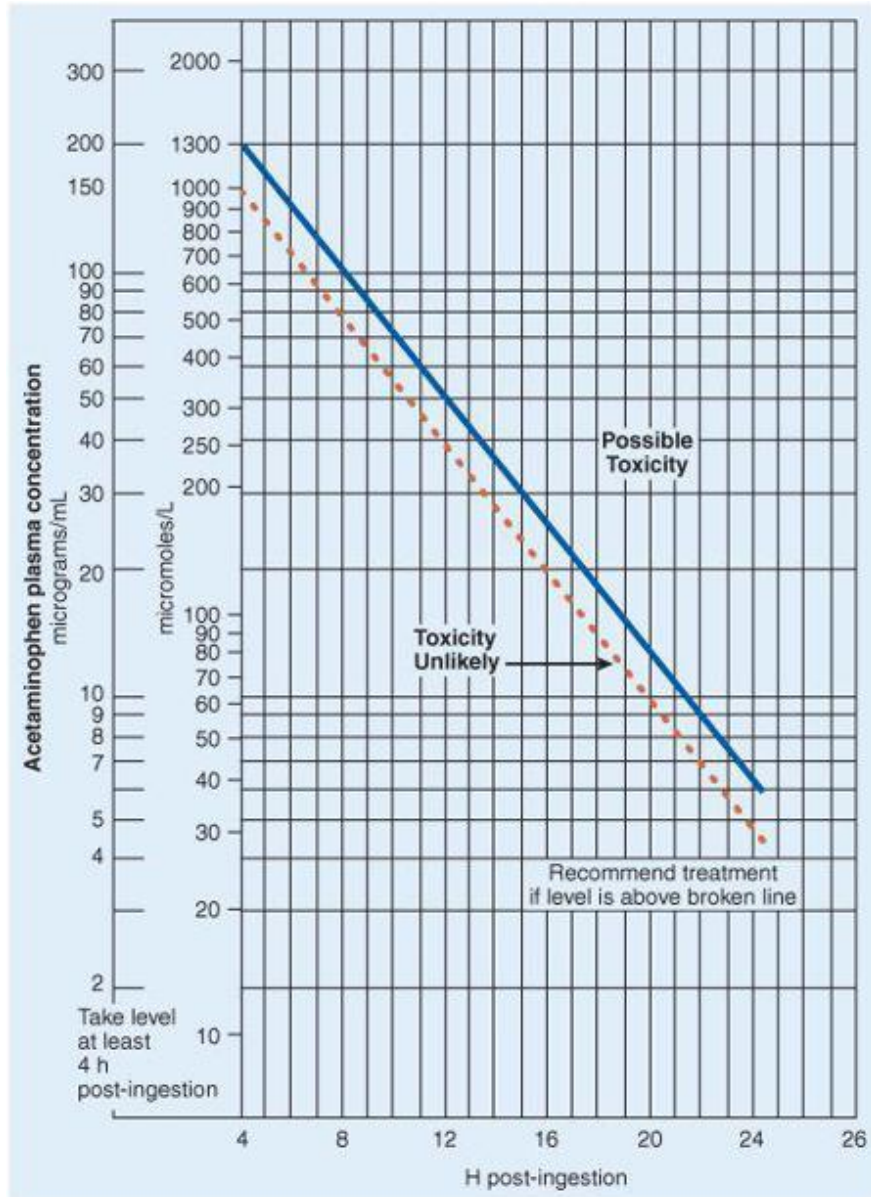
Acute acetaminophen toxicity can present in 4 classic stages outlined in [Table 106-2](#). Massive acetaminophen ingestions (4-hour acetaminophen level  $> 800$  micrograms/mL) may cause coma or agitation and lactic acidosis early, as opposed to delayed symptom onset seen in smaller overdoses.

	Stage 1	Stage 2	Stage 3	Stage 4
Timing	First 24 h	Days 2 to 3	Days 3 to 4	After day 5
Clinical manifestations	Anorexia Nausea  Vomiting Malaise	Improvement in anorexia, nausea, and vomiting  Abdominal pain Hepatic tenderness	Recurrence of anorexia, nausea, and vomiting  Encephalopathy Anuria Jaundice	Clinical improvement and recovery (7 to 8 days) <i>or</i> Deterioration to multiorgan failure and death
Laboratory abnormalities	Hypokalemia	Elevated serum transaminases Elevated bilirubin and prolonged prothrombin time if severe	Hepatic failure  Metabolic acidosis Coagulopathy Renal failure Pancreatitis	Improvement and resolution <i>or</i> Continued deterioration

**TABLE 106-2** Clinical Stages of Acute Acetaminophen Toxicity

## Diagnosis and Differential

Toxic exposure to acetaminophen is likely when an adult ingests > 10 grams or 200 milligrams/kilogram in a single ingestion or over a 24-hour period, or > 6 grams or 150 milligrams/kilogram/day for 2 consecutive days. Confirm toxicity with a serum acetaminophen concentration and determining the time of ingestion. Plot the serum acetaminophen level on the Rumack-Matthew nomogram ([Fig. 106-1](#)); this nomogram applies only to the setting of a single acute exposure and a window between 4 hours and 24 hours postingestion. Obtain additional laboratory studies, including electrolytes, glucose, BUN, creatinine, transaminases, CBC, and PT, ASA, urine toxicology screen, and ECG as clinically indicated (eg, potential coingestion in the suicidal patient).



**FIGURE 106-1.** Rumack-Matthew nomogram. (Reproduced with permission from Rumack BH, Matthew H. Acetaminophen poisoning and toxicity. *Pediatrics*. 1975 Jun;55(6):871-6.)

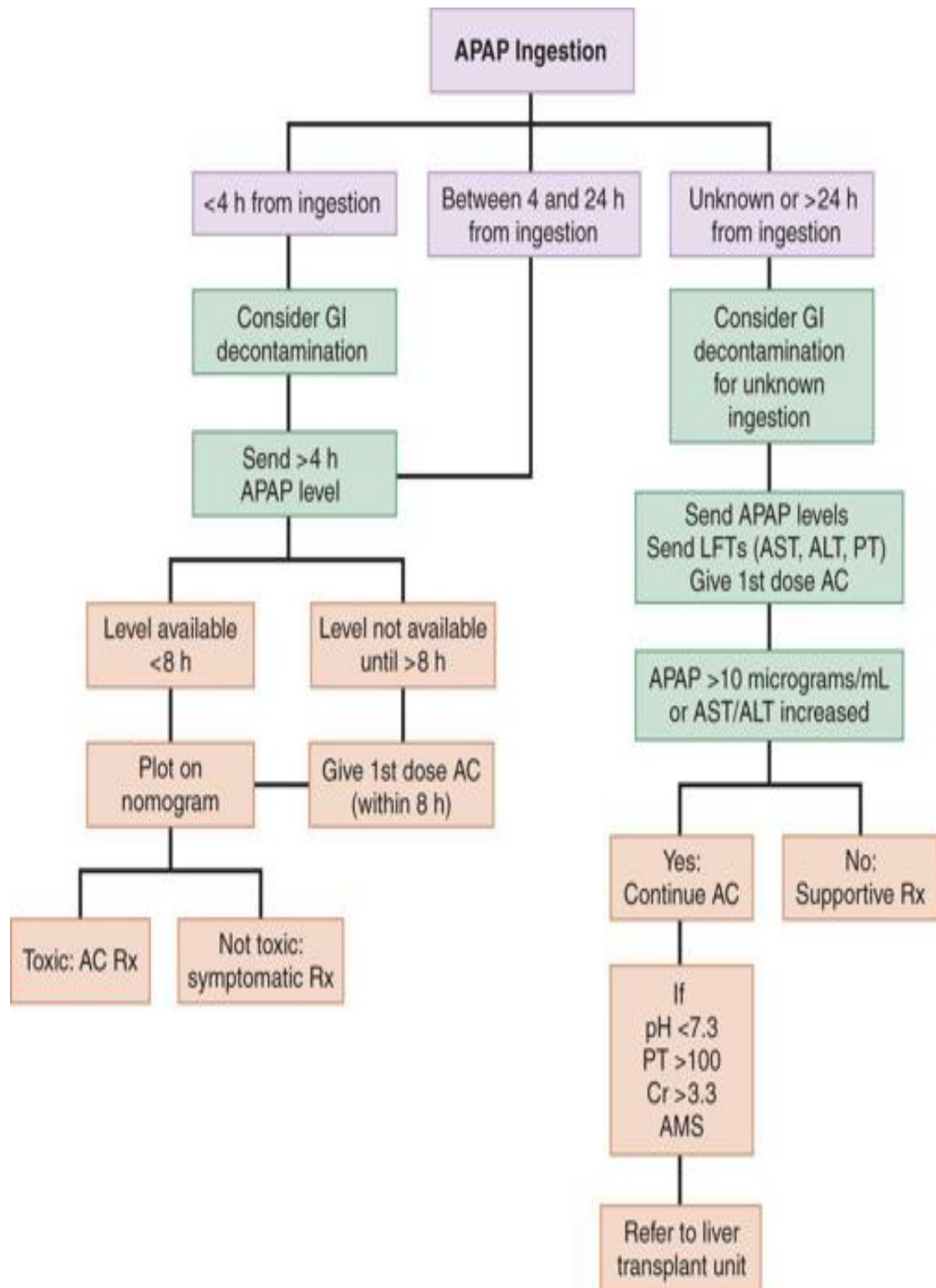
## Emergency Department Care and Disposition

1. **N-acetylcysteine (NAC)** is the antidote for acetaminophen poisoning. Dosing of NAC is outlined in [Table 106-3](#), and the treatment algorithm is depicted in [Fig.](#)

106-2.

	Oral	IV Adult	IV Pediatric (<40 kg)
Preparation	Available as 10% and 20% solutions Dilute to 9% solution for oral administration	Available as 20% solution	Available as 20% solution Dilute to 2% solution by mixing 50 mL in 450 mL 9% dextrose in water
Loading dose	140 milligrams/kilogram	150 milligrams/kilogram in 200 mL 9% dextrose in water infused over 15 to 60 min	150 milligrams/kilogram (7.5 mL/kg) infused over 15 to 60 min
Maintenance dose	70 milligrams/kilogram every 4 h for 17 doses	50 milligrams/kilogram in 500 mL 9% dextrose in water infused over 4 h followed by 100 milligrams/kilogram in 1000 mL 9% dextrose in water infused over 16 h	50 milligrams/kilogram (2.5 mL/kg) infused over 4 h followed by 100 milligrams/kilogram (5 mL/kg) infused over 16 h
Duration of therapy	72 h	20 h	20 h
Comments	Dilute with powdered drink mix, juice, or soda Serve chilled Drink through a straw to reduce dyspeptic smell	Monitor for drug-related adverse effects and anaphylactoid reactions	Monitor for drug-related adverse effects and anaphylactoid reactions 500 mL of the 2% solution prepared as described above is enough to treat a 33-kilogram child for the full 20-h course

**TABLE 106-3** Acetylcysteine Dosing Regimens



**FIGURE 106-2.** Treatment guidelines for acetaminophen (APAP) ingestion. All times noted are postingestion. *Key:* AC = acetylcysteine; ALT = alanine

aminotransferase; AMS = altered mental status; AST = aspartate aminotransferase; Cr = creatinine; LFTs = liver function tests; PT = prothrombin time; Rx = treatment.

2. If acetaminophen is still detectable after the dosing regimens described in [Table 106-3](#) are complete, continue NAC until acetaminophen is undetectable in serum.
3. Treat abnormalities related to fulminant hepatic failure by correcting coagulopathy and acidosis, managing cerebral edema, and supporting multiorgan failure, and refer early to a transplant center.
4. Patients with nontoxic acetaminophen levels based on the Rumack-Matthew nomogram may be medically cleared from the ED if there is no evidence of other drug ingestion. Admit all patients receiving NAC therapy.

## NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

### Clinical Features

Clinical features of NSAID toxicity after acute overdose are outlined in [Table 106-4](#), though toxicity is more commonly associated with chronic therapeutic use of NSAIDs than acute ingestion.

Initial symptoms within 4 h of ingestion	Abdominal pain, nausea, vomiting
Central nervous system	Headache, nystagmus, diplopia, altered mental status, coma, muscle twitching, and seizures (mefenamic acid)
Cardiovascular	Hypotension, shock, bradydysrhythmia
Electrolyte	Hyperkalemia, hypocalcemia, hypomagnesemia
GI and hepatic	Continued abdominal pain, nausea, vomiting, hepatic injury, pancreatitis (rare)
Renal	Renal insufficiency

**TABLE 106-4** NSAID Toxicity After an Acute Overdose

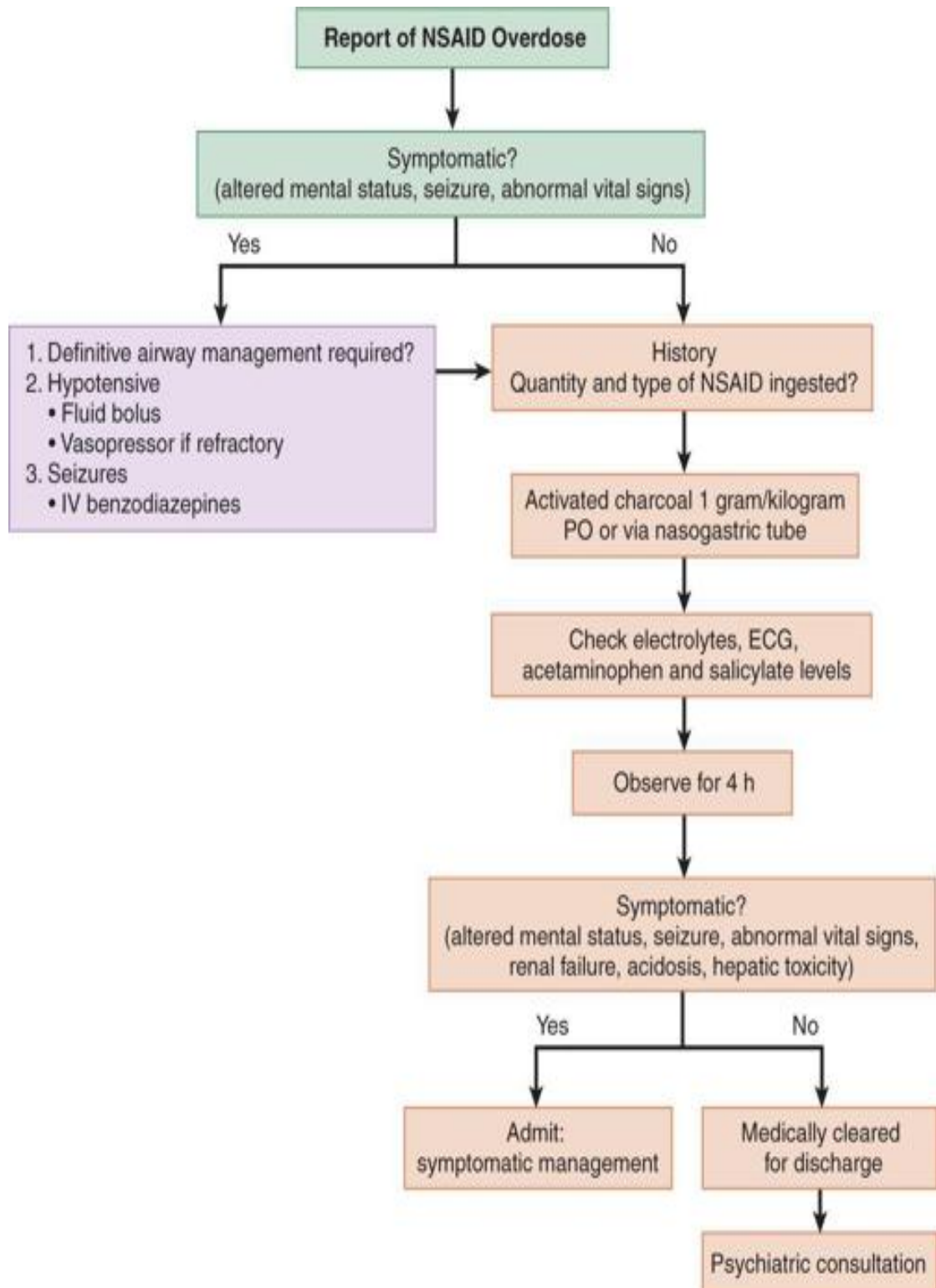
### Diagnosis and Differential

The manifestations of NSAID toxicity are nonspecific. NSAID levels are not readily available or clinically useful in assessing toxicity. Laboratory evaluation should include electrolytes, glucose, renal and hepatic function tests, and acetaminophen level (if indicated). Bedside glucose determination is indicated for altered mental status or seizures.



## **Emergency Department Care and Disposition**

A general algorithm for acute NSAID overdoses is outlined in [Fig. 106-3](#).



**FIGURE 106-3.** Approach to treatment of acute NSAID overdose.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 183, "Aspirin and Salicylates," by Luke Yip; Chapter 184, "Acetaminophen," by Oliver L. Hung and Lewis S. Nelson; and Chapter 185, "Nonsteroidal Anti-inflammatory Drugs," by Joseph G. Rella and Wallace A. Carter.

# **CHAPTER 107**

## **Xanthines and Nicotine**

Jean C.Y. Lo

Theophylline, theobromine, and caffeine are methylxanthines. Theophylline was once widely used for the management of asthma and chronic obstructive pulmonary disease. Theobromine is found in chocolate and cocoa as well as numerous “energy drinks.” Caffeine is the most commonly used psychoactive drug in the world and can be legally purchased by children. It is used medically in the management of apnea of prematurity, as an analgesic adjunct, and in some over-the-counter weight loss preparations.

Nicotine is rapidly absorbed through the lungs, mucous membranes, intestinal tract, and skin. Once absorbed, it binds to nicotinic receptors throughout the body including the central nervous system, autonomic system, and neuromuscular junction.

### **CLINICAL FEATURES**

Methylxanthine toxicity can cause life-threatening cardiac, neurologic, and metabolic abnormalities. Even therapeutic concentrations of theophylline can cause significant side effects in some individuals. Elderly patients with concomitant medical problems are more susceptible to life-threatening toxicity with chronic use than are younger patients with acute overdose.

Cardiac side effects include sinus tachycardia, premature atrial contractions, atrial flutter, and atrial fibrillation. Ventricular arrhythmias are more common with chronic toxicity, in the elderly, and those with underlying cardiac dysfunction. Hypotension may also occur.

Neurologic toxicity includes agitation, headache, irritability, sleeplessness, tremors, hallucinations, and seizures. Methylxanthine-induced seizures can be severe and refractory to treatment.

Metabolic side effects include hypokalemia, hyperglycemia, and metabolic acidosis. Rhabdomyolysis has been reported with theophylline and caffeine overdose. Gastrointestinal effects commonly include nausea and vomiting.

Nicotine toxicity affects the GI, neurologic, cardiovascular, and respiratory systems. Nausea, vomiting, bradycardia, arrhythmias, hypoventilation, coma, and seizures can occur. In severe poisoning, nicotine can result in paralysis and respiratory arrest. [Table 107-1](#) lists the clinical effects of nicotinic receptor stimulation.

Organ System	Signs and Symptoms of Nicotine Toxicity*	
	Immediate (<1 h)	Delayed (>1 h)
GI	Hypersalivation Nausea Vomiting	Diarrhea
Cardiovascular	Tachycardia Hypertension	Arrhythmias Bradycardia Hypotension
Neurologic	Tremor Headache Ataxia	Hypotonia Seizure Coma
Respiratory	Bronchorrhea	Hypoventilation Apnea

\*Onset of toxicity is varied and can be delayed for hours following dermal exposure.

**TABLE 107-1** Clinical Effects of Nicotine Toxicity

## DIAGNOSIS AND DIFFERENTIAL

Therapeutic serum theophylline levels of 10 to 15 micrograms/mL can produce toxic effects and the severity of symptoms may not correlate with serum levels, especially in the setting of chronic use. Life-threatening side effects can occur with little warning and before lesser symptoms manifest. Smoking cessation, cirrhosis, and numerous medications, such as cimetidine and erythromycin, increase the half-life of theophylline and may precipitate toxicity. Laboratory evaluation for theophylline toxicity includes serum theophylline level, and electrolytes and an ECG should be obtained

in cases of all methylxanthine toxicity. The differential diagnosis includes other stimulant drug overdose (eg, amphetamines, cocaine) and electrolyte abnormalities.

Diagnosis of acute nicotine toxicity is largely based on history and physical examination. Qualitative urine toxicological screen is of little value. Poisoning by pesticides such as organophosphates and carbamates can lead initially to nicotinic receptor stimulation that can mimic nicotine poisoning.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

### **Methylxanthine Poisoning Treatment**

Treatment of **methylxanthine poisoning** consists of stabilization, gastric decontamination and elimination, treatment of life-threatening toxic effects, and, in severe cases, hemoperfusion or dialysis.

1. [Table 107-2](#) lists appropriate methods of GI decontamination and elimination in methylxanthine toxicity.

GI Decontamination Technique	Indication*	Dosing
Activated charcoal (single dose)	Acute ingestion	< 12 y old: 0.5 to 1.0 gram/kilogram PO > 12 y old: 25 to 100 grams PO
Multidose activated charcoal (requires close observation)	Acute ingestion	Normal activated charcoal loading dose, followed by: 0.25 to 0.5 grams/kilogram PO every 2 to 4 h for 12 h (frequency and duration may vary)
Whole-bowel irrigation using polyethylene glycol 3350 with added electrolyte lavage solution	Acute ingestion of sustained-release preparations	9 mo to 6 y: 25 mL/kg/h 6 to 12 y old: 1000 mL/h > 12 y old: 1500 to 2000 mL/h Duration: 4 to 6 h or until clear rectal effluent
Ipecac syrup	Not indicated	
Gastric lavage	Not indicated	
Cathartics	Not indicated	

\*Consider contraindications; for details see <http://www.clintox.org/positionstatements.cfm>.

**TABLE 107-2** GI Decontamination for Methylxanthine Toxicity

- Place all patients on monitors and establish intravenous (IV) or intraosseous (IO) access.
- Treat nausea and vomiting with **ondansetron** 4 milligrams IV or PO (0.1 to 0.15 milligram/kilogram). Consider **ranitidine** for gastric hypersecretion but *avoid cimetidine*, which can prolong the half-life of theophylline.
- Treat seizures with **lorazepam** 1 to 2 milligrams IV (0.1 milligram/kilogram). Give **phenobarbital** 10 to 20 milligrams/kilogram IV if benzodiazepines are ineffective. Anticipate respiratory depression and the need for ventilator assistance. Phenytoin is *contraindicated* in theophylline toxicity.
- Administer IV isotonic crystalloid for hypotension. Consider cardio-selective  $\beta$ -blockers such as **esmolol** or **metoprolol** in patients with hypotension unresponsive to IV fluids or conventional vasopressors.
- Treat cardiac arrhythmias with cardio-selective  $\beta$ -blockers such as **metoprolol** and **esmolol**. Consider a calcium channel blocker such as **diltiazem** for atrial fibrillation. Identify and treat electrolyte abnormalities such as hypokalemia, which may contribute to arrhythmias.
- Consider **hemodialysis** or **hemoperfusion** in a symptomatic patient with a serum theophylline level > 90 micrograms/mL after acute ingestion, or

> 40 micrograms/mL in the setting of chronic toxicity and in patients with life-threatening seizures or arrhythmias.

8. Admit patients with seizures or ventricular dysrhythmias to intensive care. Patients with mild symptoms and theophylline levels below 25 micrograms/mL do not require specific treatment or admission, but their medication dosing should be decreased or discontinued. Patients with levels > 30 micrograms/mL should be treated with oral activated charcoal and monitored for toxic side effects.

## **Nicotine Toxicity Treatment**

1. Place patients on a cardiac monitor and establish intravenous or intraosseous access.
2. Consider dermal decontamination with soap and water for skin exposure (eg, transdermal patches). Activated charcoal and enhanced elimination are not recommended. Urine acidification is contraindicated.
3. Treat nausea and vomiting with **ondasetron** 4 milligrams IV or PO (0.1 to 0.15 milligram/kilogram).
4. Treat seizures with **lorazepam** 1 to 2 milligrams IV (0.1 milligram/kilogram).
5. Administer isotonic crystalloid for hypotension.
6. Anticipate neuromuscular weakness or respiratory depression in severe toxicity and be prepared for endotracheal intubation and mechanical ventilation.
7. Patients who remain asymptomatic at least 3 hours after ingestion of nicotine-containing products, except after intact transdermal patch ingestion, can be discharged. Patients who ingest transdermal patches should be monitored for at least 6 hours.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 186, “Methylxanthines and Nicotine,” by Chip Gresham and Daniel E. Brooks.



# CHAPTER 108

## Cardiac Medications

D. Adam Algren

### DIGITALIS GLYCOSIDES

Digoxin is used to treat supraventricular tachyarrhythmias and congestive heart failure. Other cardiac glycosides are found in plants such as foxglove, oleander, and lily of the valley.

#### Clinical Features

Toxicity can occur following acute ingestion or develop during chronic therapy ([Table 108-1](#)). *Acute toxicity* typically presents with nausea, vomiting, or abdominal pain. Characteristic cardiac effects include bradyarrhythmias and/or supraventricular tachycardia with atrioventricular block. Severe toxicity can result in ventricular arrhythmias. *Chronic toxicity* is more common in the elderly and often occurs as a result of concomitant illness (heart disease, renal/liver failure, hypothyroidism, chronic obstructive pulmonary disease), electrolyte abnormality (hypokalemia, hypomagnesemia), or drug interactions (quinidine, amiodarone, spironolactone, calcium channel blockers, macrolide antibiotics). Neuropsychiatric symptoms are more common with chronic toxicity, though cardiac effects are similar to those seen with acute toxicity.

Acute toxicity	
Clinical history	Intentional or accidental ingestion
GI effects	Nausea and vomiting
Central nervous system effects	Headache, dizziness, confusion, coma
Cardiac effects	Bradycardias or supraventricular tachycardias with atrioventricular block
Electrolyte abnormalities	Hyperkalemia
Digoxin level	Marked elevation (if obtained within 6 h)
Chronic toxicity	
Clinical history	Typically in elderly cardiac patients taking diuretics; may have renal insufficiency
GI effects	Nausea, vomiting, diarrhea, abdominal pain
Central nervous system effects	Fatigue, weakness, confusion, delirium, coma
Cardiac effects	Almost any ventricular or supraventricular dysrhythmia can occur; ventricular dysrhythmias are common
Electrolyte abnormalities	Normal or decreased serum potassium, hypomagnesemia
Digoxin level	Minimally elevated or within "therapeutic" range

**TABLE 108-1** Clinical Presentation of Digitalis Glycoside Toxicity

## Diagnosis and Differential

Hyperkalemia is often seen in acute poisoning, but may be absent in chronic toxicity. Serum digoxin levels are neither sensitive nor specific for toxicity. However, those patients with higher levels (> 2 milligrams/mL) are more likely to experience toxicity. Almost any arrhythmia can occur with toxicity; however, the most common finding is premature ventricular beats. The differential diagnosis includes sinus node disease or toxicity from calcium channel blockers,  $\beta$ -blockers, class IA antidysrhythmics, clonidine, organophosphates, or other cardiotoxic plants.

## Emergency Department Care and Disposition

All patients require continuous cardiac monitoring, intravenous (IV) access, and frequent reevaluation ([Table 108-2](#)).

<p>Asymptomatic patients</p> <ul style="list-style-type: none"> <li>Obtain accurate history</li> <li>Continuous cardiac monitoring</li> <li>IV access</li> <li>GI decontamination: activated charcoal, 1 gram/kilogram PO</li> <li>Frequent reevaluation</li> <li>Calculate digoxin-specific Fab antibody fragments dose in anticipation of potential need: may bring to drug bedside, depending on ready availability</li> </ul>
<p>Symptomatic patients</p> <ul style="list-style-type: none"> <li>Obtain accurate history</li> <li>IV access</li> <li>Continuous cardiac monitoring</li> <li>GI decontamination: activated charcoal, 1 gram/kilogram PO, then 0.5 gram/kilogram every 4 to 6 h</li> <li>Bradyarrhythmias <ul style="list-style-type: none"> <li>Atropine: 0.5 to 2.0 milligrams IV (0.02 milligrams/kilogram minimum dose 0.1 milligram)</li> <li>Pacer: external or transvenous</li> <li>Digoxin-specific Fab antibody fragments: IV infusion</li> </ul> </li> <li>Ventricular dysrhythmias <ul style="list-style-type: none"> <li>Digoxin-specific Fab antibody fragments: IV infusion or bolus</li> <li>Magnesium sulfate: 2 to 4 grams IV (30-50 milligrams/kilogram)</li> <li>Lidocaine: 1 milligram/kilogram</li> <li>Fosphenytoin: 15 milligrams PE/kg, infuse at 150 milligrams PE/min</li> <li>Electrocardioversion: 10 to 25 J (last resort)</li> </ul> </li> </ul>
<p>Cardiac arrest</p> <ul style="list-style-type: none"> <li>CPR with current ACLS or PALS protocols</li> <li>Digoxin-specific Fab antibody fragments: IV bolus (5 to 10 vials if amount ingested is unknown)</li> </ul> <p>Hyperkalemia</p> <ul style="list-style-type: none"> <li>Avoid calcium chloride or calcium gluconate*</li> <li>Glucose-insulin</li> <li>Sodium bicarbonate</li> <li>Digoxin-specific Fab antibody fragments: IV infusion or bolus</li> <li>Potassium binding resin</li> <li>Hemodialysis</li> </ul> <p>Hypomagnesemia</p> <ul style="list-style-type: none"> <li>Evaluate renal status prior to replacement</li> <li>Magnesium sulfate: 2 to 4 grams (30-50 milligrams/kilogram) IV</li> </ul>

Key: Fab = antigen-binding fragment; PE = phenytoin equivalents.

\*Recommendation based on older literature.

### TABLE 108-2 Treatment of Digitalis Glycoside Poisoning

1. Administer **activated charcoal**, 1 gram/kilogram in cases of acute ingestion.
2. Use **atropine** 0.5 to 2.0 milligrams (0.02 milligram/kilogram, minimum dose 0.1 milligram) IV to treat bradydysrhythmias.
3. Administer **digoxin-specific Fab** for ventricular dysrhythmias, hemodynamically significant bradydysrhythmias, and hyperkalemia greater than 5.5 mEq/L. Dosing of digoxin-specific Fab is calculated according to [Table 108-3](#).

<p>Calculate total-body load</p> <p>Based on history of amount ingested: total-body load = amount ingested (milligrams) × 0.80 (bioavailability)</p> <p>Based on serum digoxin concentration: total-body load = [serum digoxin level (ng/mL) × 5.6 L/kg × patient's weight (kg)]/1000</p>
<p>Calculate number of vials of digoxin-specific Fab antibody fragments needed to neutralize the calculated total-body load</p> <p>An equimolar dose is required for neutralization—one vial contains 38 or 40 milligrams of digoxin-specific Fab antibody fragments that will bind approximately 0.5 milligram of digoxin*</p> <p>Number of vials = total-body load/0.5</p>
<p>A simple and accurate variation using serum digoxin level</p> <p>Number of vials = [serum digoxin level (ng/mL) × patient's weight (kg)]/100</p>

Key: Fab = antigen-binding fragment.

\*The digoxin-specific Fab antibody fragments commercially available in the United States contain 38 or 40 milligrams per vial, depending in the manufacturer, but both bind approximately 0.5 milligram of digoxin.

**TABLE 108-3** Calculation of Digoxin-Specific Fab Antibody Fragment Dose

4. Treat ventricular dysrhythmias with **phenytoin** 15 milligrams/kilograms infused no faster than 25 milligrams/min; **lidocaine** 1 milligram/kilogram; or **magnesium sulfate** 2 to 4 grams (25 to 50 milligrams/kilograms) IV. Electro-cardioversion may induce refractory ventricular dysrhythmias and should be considered only as a last resort. The initial setting should be 10 to 25 J.
5. Treat hyperkalemia with dextrose followed by insulin; other options are sodium bicarbonate, potassium-binding resin, or hemodialysis. Historically, IV calcium use has been discouraged because of reports of ventricular arrhythmias. However, recent evidence suggests that IV calcium use is likely safe.
6. Admit patients with signs of mild toxicity to a monitored setting and manage those with significant toxicity in an intensive care unit. Repeated digoxin levels following digoxin Fab are not accurate and should not be obtained. Discharge asymptomatic patients not treated with digoxin specific Fab following 6 hours of observation if they remain asymptomatic with normal serum potassium and digoxin levels at 6 hours.

## β-BLOCKERS

β-Blockers are used in the management of acute coronary syndromes, dys-rhythmias, hypertension, thyrotoxicosis, migraines, and glaucoma. In overdose, their negative inotropic and chronotropic effects result in progressive bradycardia and hypotension.

### Clinical Features

Toxicity usually develops within 6 hours of ingestion of an immediate release product. With sustained-release preparations toxicity is generally seen within 8 to 12 hours of ingestion. The cardiovascular system is the primary organ system affected; however, other noncardiac manifestations may occur ([Table 108-4](#)). Sotalol, unlike other  $\beta$ -blockers, is also a class III antidysrhythmic. As such, it may cause QT-interval prolongation and torsades de pointes.

<b>Cardiac</b>
Hypotension
Bradycardia
Conduction delays and blocks
Ventricular dysrhythmias*
Asystole
Decreased contractility
<b>Central nervous system</b>
Depressed mental status
Coma
Psychosis
Seizures
<b>Pulmonary</b>
Bronchospasm

\*Seen with sotalol.

**TABLE 108-4** Common Findings With  $\beta$ -Blocker Toxicity

## Diagnosis and Differential

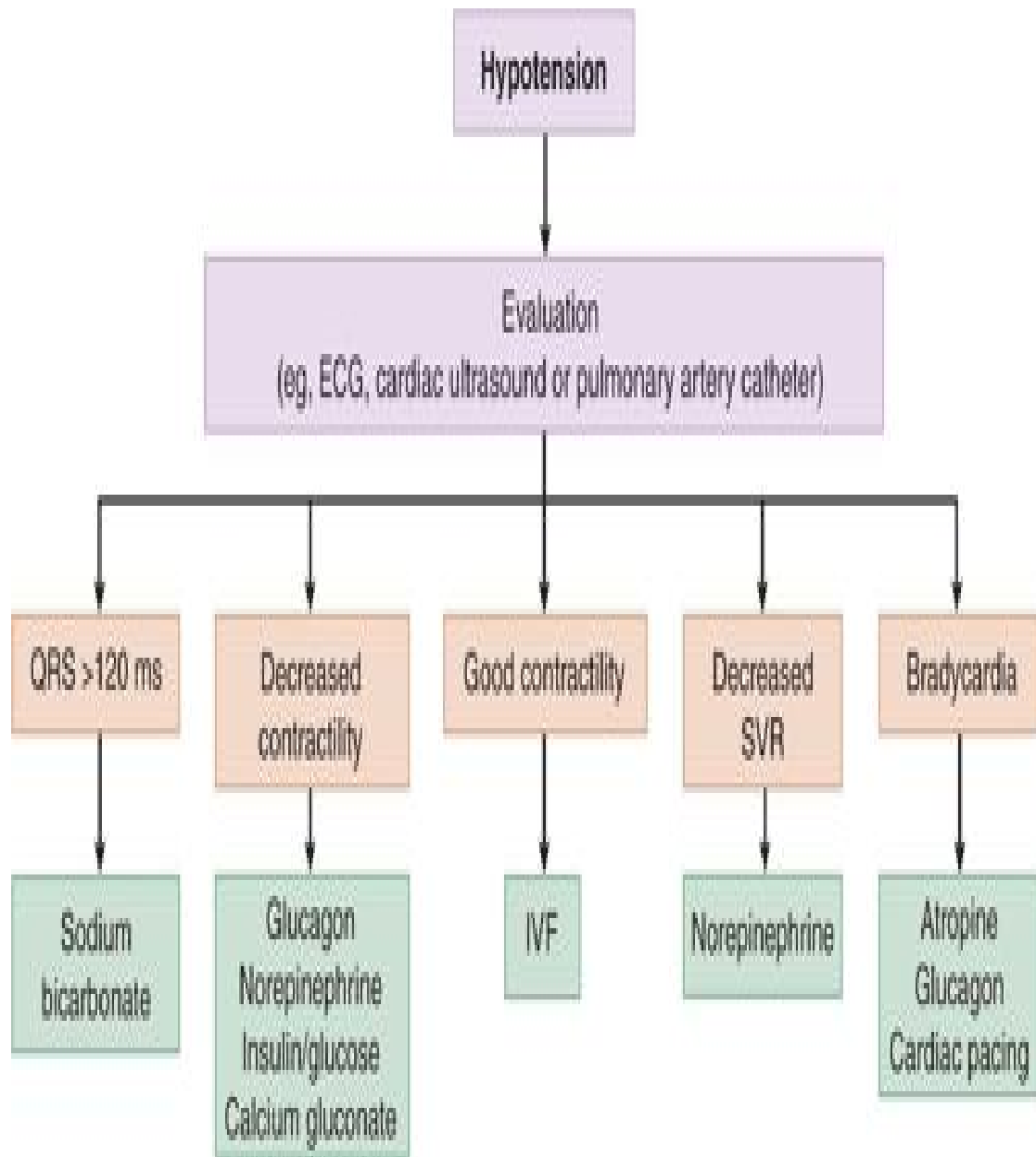
The diagnosis is made based on clinical findings. An ECG should be obtained in all cases. Laboratory studies are directed at identifying underlying medical conditions or complications. Specific drug levels are not commonly available and correlate poorly with clinical effects. [Table 108-5](#) lists other agents that result in bradycardia and hypotension.

Cause	Differentiating Features
Calcium channel blockers	Elevated lactate level and marked hyperglycemia
Naturally occurring cardiac glycosides (oleander, foxglove, lily of the valley, rhododendron, and toad-derived bufotoxin)	Ventricular ectopy May cross-react with digoxin immunoassay
Class IC antiarrhythmic drugs (propafenone)	Wide-complex bradycardia
Clonidine	Opioid-like manifestations: coma, miosis, decreased respirations
Cyanide	Profound metabolic acidosis and elevated lactate level
Digoxin (acute)	Hyperkalemia Elevated level on digoxin immunoassay
Organophosphates	Muscarinic toxidrome

**TABLE 108-5** Toxicologic Causes of Bradycardia and Hypotension

### Emergency Department Care and Disposition

The goal of therapy is to restore cardiac output by increasing heart rate and improving myocardial contractility ([Fig. 108-1](#)). Establish continuous cardiac monitoring and IV access. IV fluids may be administered for hypotension.



**FIGURE 108-1.** Management strategies in  $\beta$ -blocker toxicity. Cardiac function is evaluated using ECG and cardiac US and/or a pulmonary artery catheter. For patients with a wide QRS interval, consider sodium bicarbonate therapy. For patients with impaired myocardial contractility, consider glucagon, norepinephrine, insulin and glucose, and/or calcium therapy. For patients with preserved cardiac contractility, administer IV fluids. For patients with decreased systemic vascular resistance, consider norepinephrine. For patients with bradycardia, consider atropine, glucagon, and/or cardiac pacing. (See text for details.)

*Key:* IVF = IV fluids; SVR = systemic vascular resistance.

1. Administer **activated charcoal**, 1 gram/kilogram within 1 to 2 hours of ingestion if no contraindications exist. Gastric lavage may be beneficial if performed within 1 hour of ingestion. Consider whole-bowel irrigation for cases involving sustained-release preparations.
2. Give **atropine**, 0.5 to 1 milligram (0.02 milligram/kilogram, minimum dose 0.1 milligram) IV, for bradycardia.
3. **Glucagon** has inotropic and chronotropic effects and is the agent of choice for the treatment of toxicity. Administer as an IV bolus of 3 to 5 milligrams (0.05 milligram/kilogram). Follow with a continuous infusion of 1 to 10 milligrams/h. Nausea and vomiting are common side effects.
4. Use vasopressors, such as **norepinephrine** 2 to 30 micrograms/ kilograms/min, **epinephrine** 1 to 20 micrograms/kilograms/min, or **dopamine** 2.5 to 20 micrograms/kilograms/min, for refractory brady-cardia and hypotension.
5. Calcium may be of limited benefit in cases of refractory hypotension as either calcium gluconate or calcium chloride (10 mL of 10% [0.15 mL/kg] repeated 3 to 6 times as necessary). Although calcium chloride contains more elemental calcium than calcium gluconate, it is irritating to soft tissues and should be administered via central line.
6. **Hyperinsulinemia-euglycemia (HIE)** therapy can improve myocardial contractility. Bolus regular insulin IV (1 unit/kg) followed by a continuous infusion (0.5 to 1 unit/kg/h). Serum glucose should be supplemented and monitored frequently to avoid hypoglycemia. Monitor serum potassium for hypokalemia.
7. Cardiac pacing may be attempted but it is often unsuccessful. Aggressive measures include extracorporeal circulation or intraaortic balloon pump placement. Hemodialysis may be of benefit in cases involving acebutolol, atenolol, nadolol, or sotalol.
8. Use **lidocaine, magnesium sulfate, isoproterenol, and overdrive pacing** to treat **sotalol-induced** ventricular dysrhythmias.
9. Patients who develop bradycardia, hypotension, conduction disturbances, or altered mental status should be managed in an ICU. Admit patients who have ingested a sustained-release preparation or sotalol to a monitored setting due to concern for delayed toxicity. Those patients who remain asymptomatic 6 hours after ingestion of an immediate release agent can be medically cleared.

## **CALCIUM CHANNEL BLOCKERS**

Calcium channel blockers are used in the treatment of hypertension, vasospasm, and rate control of supraventricular tachydysrhythmias. Three widely used classes of



calcium channel blockers are the phenylalkylamines (verapamil), benzothiazepines (diltiazem), and dihydropyridines (nifedipine, etc).

## **Clinical Features**

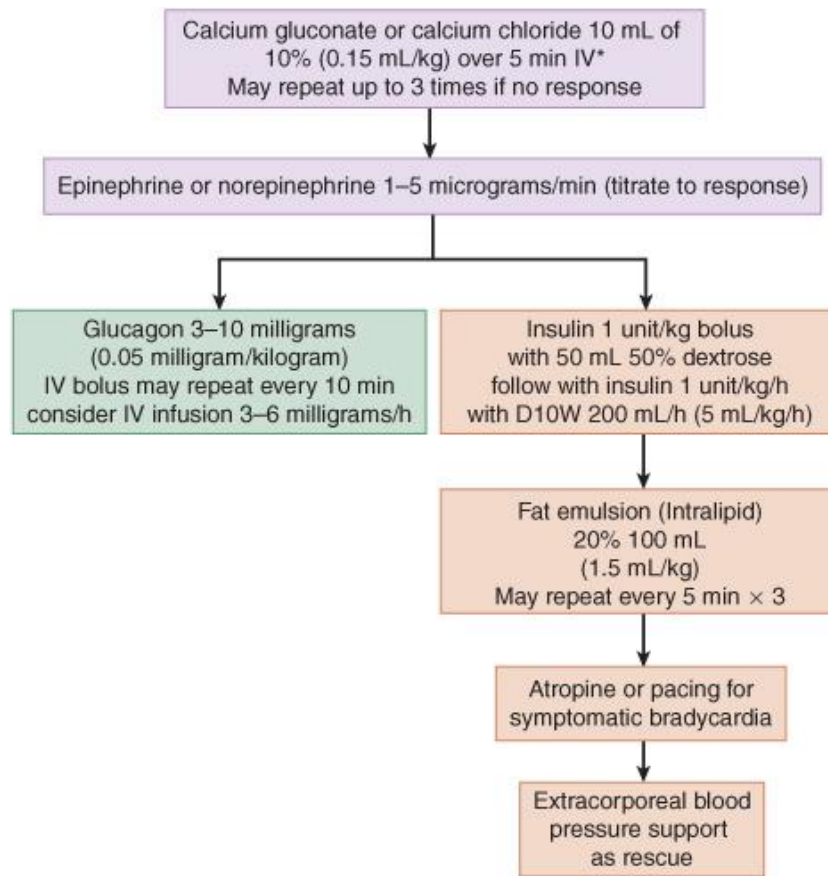
Toxicity usually develops within 6 hours of ingestion of an immediate release product. With sustained-release preparations toxicity can be delayed 12 to 24 hours. Toxicity primarily affects the cardiovascular system causing sinus bradycardia, atrioventricular block, and hypotension. Verapamil and diltiazem have a proportionally greater effect on the myocardium than the dihydropyridines. Dihydropyridine overdose can result in reflex tachycardia. With severe toxicity, all classes of calcium channel blockers can cause bradycardia, depressed myocardial contractility, and vasodilatation. Hyperglycemia, lactic acidosis, and noncardiogenic pulmonary edema may occur. Central nervous system effects are due to hypoperfusion and other etiologies should be sought if the blood pressure is normal.

## **Diagnosis and Differential**

The diagnosis is based on clinical findings. Laboratory studies help identify complications. Hyperglycemia is common and helps distinguish calcium channel blocker from  $\beta$ -blocker toxicity, which is associated with hypoglycemia. The differential diagnosis for bradycardia and hypotension is listed in [Table 108-5](#).

## **Emergency Department Care and Disposition**

Treatment is supportive, with an emphasis on increasing cardiac output and systemic vascular resistance ([Fig. 108-2](#)). Establish continuous cardiac monitoring and IV access. Administer IV fluids for hypotension.



**FIGURE 108-2.** Treatment algorithm for severe calcium channel blocker toxicity showing recommended progression in therapy at each step if no response occurs. All listed modalities have been shown to be potentially beneficial and in severe cases can be started simultaneously. D10W = 10% dextrose in water.

\* Calcium chloride provides 3 times as much elemental calcium as calcium gluconate; it should be used with caution in cases of digoxin toxicity.

1. Administer **activated charcoal**, 1 gram/kilogram within 1 to 2 hours of ingestion if no contraindications exist. Gastric lavage may be beneficial if performed within 1 hour of ingestion. Consider whole-bowel irrigation for cases involving sustained-release preparations.
2. Atropine 0.5 to 1 milligram (0.02 milligram/kilogram, minimum dose 0.1 milligram) and calcium may be of limited benefit in cases of severe toxicity; give calcium gluconate or calcium chloride [10 mL of 10% (0.15 mL/kg) repeated 3 to 6 times as necessary]. Although calcium chloride contains more elemental calcium than calcium gluconate it is irritating to soft tissues and should be administered via central line.

3. Use **norepinephrine** 2 to 30 micrograms/kg/min, **epinephrine** 1 to 20 micrograms/kilograms/min, **dopamine** 2.5 to 20 micrograms/kilograms/min, for refractory bradycardia and hypotension.
4. **Hyperinsulinemia-euglycemia (HIE)** therapy can improve myocardial contractility and blood pressure ([Table 108-6](#)). Bolus regular insulin (1 unit/kg IV) followed by continuous infusion (0.5 to 1 unit/kg/h). Monitor serum glucose and potassium frequently.

Administer 50 mL of 50% dextrose (0.5 gram/mL) in water IV.  
Administer regular insulin 1 unit/kg IV bolus.  
Begin regular insulin infusion at 0.5 to 1.0 unit/kg/h along with dextrose 10% (0.1 gram/mL) in water at 200 mL/h (adult) or 5 mL/kg/h (pediatric).  
Monitor serum glucose every 20 min. Titrate dextrose infusion rate to maintain serum glucose level between 150 and 300 milligrams/dL.  
Once infusion rates have been stable for 60 min, glucose monitoring may be decreased to hourly.  
Monitor serum potassium level and start IV potassium infusion if serum potassium level is <3.5 milliequivalents/L.

**TABLE 108-6** Protocol for Hyperinsulinemia-Euglycemia Therapy in Severe Calcium Channel Blocker Overdose

5. **Glucagon** is variably successful in the treatment of calcium channel blocker toxicity. Administer as an IV bolus of 3 to 5 milligrams (0.05 milligram/ kilogram) followed by continuous infusion of 1 to 10 milligrams/h.
6. IV fat emulsion (20% solution) has shown promising success in the treatment of severe toxicity. Administer as a bolus of 1.5 mL/kg IV followed by a continuous infusion of 0.25 mL/kg/min.
7. Patients who develop bradycardia, hypotension, or conduction disturbances should be managed in an ICU. Patients who remain asymptomatic 6 hours after ingestion of an immediate release agent can be medically cleared. Admit patients who have ingested a sustained-release preparation or sotalolol to a monitored setting due to concern for delayed toxicity.

## ANTIHYPERTENSIVES

Commonly available antihypertensive agents include diuretics, clonidine, angiotensin converting enzyme inhibitors, and angiotensin II receptor antagonists ([Table 108-7](#)). Acute overdose of diuretics are not expected to result in life-threatening clinical toxicity.

Class	Drug	Mechanism of Action	Clinical Presentation of Toxicity	Comments
Diuretics	Acetazolamide	Inhibition of proximal tubule sodium-hydrogen exchange	Hypocalemia Nonanion gap metabolic acidosis	
	Chlorthalidone	Inhibition of distal tubule sodium chloride absorption	Hypocalemia	
	Chlorthalidone		Hypokalemia	
	Hydrochlorothiazide			
	Indapamide			
	Metolazone			
	Bumetanide	Inhibition of sodium-potassium-chloride symporter in renal loop of Henle	Hypocalemia	
	Furosemide		Hypocalemia Hypokalemia Hypomagnesemia	
	Amiloride	Inhibition of sodium absorption and potassium elimination in renal distal collecting duct	Hypocalemia	
	Tamoxifen		Hypokalemia	
	Eplerenone	Mineralocorticoid antagonist	Hypocalemia	
	Spirolactone		Hypokalemia	

Sympatholytics	Doxazosin	$\alpha_1$ -adrenergic receptor antagonist	Hypotension	Phenylephrine may be used for refractory hypotension.
	Prazosin			
	Tamsulosin			
	Terazosin			
	Clonidine	$\alpha_2$ -adrenergic receptor agonist	Hypotension	Hypotension may respond to high-dose nifedipine. Phenylephrine may be used for refractory hypotension.
	Guafenesin	Imidazoline receptor agonist	Bradycardia	
	Guafacine	$\mu$ -receptor opioid agonist	Neurologic depression	
	Ormetopidine			
	Tetrahydrozoline			
	Guafacine	Decreased norepinephrine release	Hypotension	
	Methyldopa		Bradycardia	
	Reserpine		Hemolytic anemia (idiosyncratic reaction to methyldopa)	

ACE inhibitors	Benazepril Captopril Enalapril Fosinopril Moexapril Perindopril Quinapril Terdolapril	Inhibition of angiotensin-converting enzyme Inhibition of bradykininase	Hypotension Hyperkalemia Angioedema (idiosyncratic) Cough (idiosyncratic)	Hypotension may respond to norepinephrine. Corticosteroids and diphenhydramine may be administered for angioedema.
Angiotensin receptor blockers	Candesartan Eprosartan Irasartan Losartan Telmisartan Valsartan	Angiotensin II receptor antagonist	Hypotension Hyperkalemia Angioedema (less common than with ACE inhibitors)	Corticosteroids and diphenhydramine may be administered for angioedema.

Vasodilators	Hydralazine Minoxidil	Arterial vasodilation	Hypotension Tachycardia Increased myocardial oxygen demand Lupuslike syndrome (idiosyncratic reaction to hydralazine)	
	Sodium nitroprusside	Arterial and venous vasodilation (via nitric oxide release)	Hypotension Tachycardia Tachycardia Thiocyanate toxicity (after prolonged infusion) Cyanide toxicity (very rare)	Thiosulfate should be administered if cyanide toxicity is considered.

Key: ACE = angiotensin-converting enzyme

**TABLE 108-7** Summary of Antihypertensive Drugs

## Clinical Features

**Thiazides and loop diuretics** (hydrochlorothiazide, furosemide) can cause mild hypotension, tachycardia, and hypokalemia.

**Potassium-sparing diuretics** (spironolactone, triamterene, and amiloride) can cause hyperkalemia.

## Emergency Department Care and Disposition

Management is supportive including correction of electrolyte abnormalities. Establish continuous cardiac monitoring and IV access in all patients.

1. Administer IV normal saline to correct hypovolemia.
2. Start **dopamine** 2.5 to 20 micrograms/kilogram/min for hypotension refractory to volume resuscitation.
3. Correct potassium abnormalities using standard measures. Patients with severe hyperkalemia from potassium-sparing diuretics may require dialysis.

4. Most patients can be medically cleared after a 4 to 6 hours observation period. Patients with hypotension or electrolyte abnormalities require admission.

## CLONIDINE

Clonidine is a centrally acting  $\alpha$  agonist used for the management of hyper-tension and opiate withdrawal.

### Clinical Features

Clonidine toxicity causes hypotension and bradycardia as well as CNS and respiratory depression.

### Emergency Department Care and Disposition

Management is supportive though respiratory depression and apnea, most commonly seen in children, may require endotracheal intubation. Establish continuous cardiac monitoring and IV access in all patients.

1. Administer IV normal saline for hypotension.
2. Use **dopamine** 2.5 to 20 micrograms/kilogram/min or **norepinephrine** 2 to 30 micrograms/kilogram/min for hypotension refractory to fluid resuscitation.
3. Give **atropine** 0.5 to 1 milligram (0.02 milligram/kilogram, minimum dose 0.1 milligram) for symptomatic bradycardia.
4. **Naloxone** may be effective for cases of refractory hypotension or altered mental status, but often requires high doses (up to 10 milligrams IV).
5. Patients who remain asymptomatic after a 4-hour observation period can be medically cleared. Admit symptomatic patients to a monitored setting.

## ANGIOTENSIN-CONVERTING INHIBITORS

## ENZYME

Agents include captopril, enalapril, and lisinopril. The primary toxicity in overdose is hypotension which may be treated with IV normal saline and vasopressors. Naloxone has been reported to reverse ACEI-induced hypotension. Patients require monitoring for at least 6 hours.



## ANGIOTENSIN II RECEPTOR ANTAGONISTS

Agents include losartan, valsartan, and candesartan. Experience with toxicity is limited, but hypotension and tachycardia are the most common toxic effects. Hyperkalemia has also been reported. Therapy is supportive and includes IV fluid administration, correction of electrolyte disturbances, and cardiac monitoring for at least 6 hours.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 187, "Digitalis Glycosides," by Jennifer S. Boyle and Mark A Kirk; Chapter 188, "β-Blockers," by Jennifer L. Englund and William P. Kerns II; Chapter 189, "Calcium Channel Blockers," by Alicia B. Minns and Christian Tomaszewski; and Chapter 190, "Antihypertensive Agents," by Andrew Stolbach and Arjun Chanmugam.

# CHAPTER 109

## Anticonvulsants

Alicia Minns

### PHENYTOIN

Intentional phenytoin overdose rarely leads to death, provided adequate supportive care is administered. Most phenytoin-related deaths have been caused by rapid IV administration and hypersensitivity reactions.

#### Clinical Features

The toxic effects of **phenytoin** depend on the duration of exposure, dosage taken, and route of administration. Life-threatening effects such as hypotension, bradycardia, and asystole are seen with IV administration and are secondary to the diluent, propylene glycol. Morbidity can be avoided by slowing the rate of administration. **Fosphenytoin** is well tolerated intramuscularly or intravenously; adverse and toxic effects are the same as those of phenytoin, except the toxic effects of propylene glycol are not present.

Clinical manifestations in overdose are typically dose related and are listed in [Table 109-1](#). The primary clinical features of overdose are related to acute CNS effects while cardiovascular toxicity is almost entirely seen in cases of IV administration. Skin and soft tissue injury may be seen with IM injection of phenytoin or after extravasation from IV infusion, but are rarely seen with fosphenytoin. Therapeutic use has been associated with hypersensitivity reactions and gingival hyperplasia. Phenytoin is teratogenic.

Central nervous system effects	Dizziness, tremor (intention), visual disturbance, horizontal and vertical nystagmus, diplopia, miosis or mydriasis, ophthalmoplegia, abnormal gait (bradykinesia, truncal ataxia), choreoathetoid movements, vomiting, dysphagia, irritability, agitation, confusion, hallucinations, fatigue, coma, encephalopathy, pseudo-degenerative disease, dysarthria, meningeal irritation with pleocytosis, seizures (rare)
Peripheral nervous system effects	Peripheral neuropathy, urinary incontinence
Hypersensitivity reactions (anticonvulsant hypersensitivity syndrome)	Eosinophilia, rash, pseudolymphoma (diffuse lymphadenopathy), systemic lupus erythematosus, pancytopenia, hepatitis, pneumonitis
GI effects	Nausea and vomiting, hepatotoxicity
Dermatologic effects	Hirsutism, acne, rashes (including Stevens-Johnson syndrome)
Other effects	Fetal hydantoin syndrome, gingival hyperplasia, coarsening of facial features, hemorrhagic disease of the newborn, hyperglycemia, hypocalcemia
Parenteral toxicity	May cause hypotension, bradycardia, conduction disturbances, myocardial depression, ventricular fibrillation, asystole, and tissue necrosis from infiltration

**TABLE 109-1** Clinical Features of Phenytoin Toxicity

## Diagnosis and Differential

Diagnosis is made by history and serum drug levels. The therapeutic range is 10 to 20 micrograms/mL and toxicity generally correlates with increasing plasma levels ([Table 109-2](#)). Absorption is erratic, and serial levels should be obtained. Electrocardiographic changes in toxicity include increased PR interval, widened QRS interval, and altered ST-wave and T-wave segments.

Plasma Level (micrograms/mL)	Side Effects
<10	Usually none
10 to 20	Occasional mild nystagmus
20 to 30	Nystagmus
30 to 40	Ataxia, slurred speech, nausea and vomiting
40 to 50	Lethargy, confusion
>50	Coma, seizures

**TABLE 109-2** Correlation of Plasma Phenytoin Level and Side Effects

Almost any CNS-active drug can mimic phenytoin toxicity. Disease states that resemble phenytoin toxicity include hypoglycemia, Wernicke encephalopathy, and posterior fossa hemorrhage or tumor.

## **Emergency Department Care and Disposition**

1. Place patients on monitors and obtain IV or IO access.
2. Treat acute oral overdose with multi-dose of oral **activated charcoal** (1 gram/kilogram) every 4 hours for the first 24 hours. Correct acidosis to decrease free serum phenytoin. Hemodialysis and hemoperfusion are of no benefit.
3. Treat hypotension from IV administration of phenytoin with IV isotonic crystalloid and discontinuation of the infusion.
4. Treat bradydysrhythmias with **atropine or cardiac pacing**.
5. Treat seizures with a **benzodiazepine or phenobarbital**.
6. Obtain orthopedic or plastic surgery consultation for significant soft tissue injury.
7. Admit patients with serious complications (eg, seizures, coma, altered mental status, and ataxia). Observe patients with mild symptoms until serum levels are declining; cardiac monitoring after isolated oral ingestion is unnecessary. Discontinue phenytoin and recheck levels in 2 to 3 days.

## **CARBAMAZEPINE**

### **Clinical Features**

Carbamazepine's anticholinergic properties delay GI motility and can cause delayed clinical deterioration. Manifestations of acute toxicity include coma, respiratory failure, ataxia, nystagmus, miosis or mydriasis, ileus, bowel obstruction, hypertonicity, increased deep tendon reflexes, dys-tonic reactions, and an anticholinergic toxidrome. Seizures can occur at high

concentrations. Though rare, carbamazepine can widen the QRS interval and cause cardiac arrhythmias.

## **Diagnosis and Differential**

Serum carbamazepine concentrations do not correlate with the severity of poisoning, though concentrations > 40 micrograms/mL may be associated with an increased risk of complications, and those > 60 to 80 micrograms/mL may be fatal. A false positive tricyclic antidepressant result on urine drug screen can occur. Obtain an ECG to evaluate for QRS interval widening.

## **Emergency Department Care and Disposition**

1. Consider **activated charcoal** in the awake patient within 1 hour of ingestion.
2. Consider **hemodialysis or hemodiafiltration** for life-threatening overdose.
3. Treat QRS interval widening with sodium bicarbonate.
4. Patients can be discharged if asymptomatic, with declining serum levels (below 15 micrograms/mL), and a normal ECG.

# **VALPROATE**

## **Clinical Features**

In acute overdose, valproate causes central nervous system depression. Other findings include respiratory depression, hypotension, hypoglycemia, hypernatremia, hypophosphatemia, and anion gap metabolic acidosis that may persist for days. Liver toxicity, cerebral edema, hyperammonemia, pancreatitis, and thrombocytopenia have been reported after acute overdose. Hepatic failure (microvesicular steatosis) occurs in about 1 in 20,000

patients on long-term therapy, with children <3 years of age on multiple antiepileptics at greatest risk.

## **Diagnosis and Differential**

Obtain a serum valproate level. The therapeutic range is 50 to 100 micrograms/ mL; adverse effects increase at concentrations > 150 micrograms/mL and frank coma may occur with levels above 800 micrograms/mL. Check serum ammonia and bedside glucose in patients with altered level of consciousness. Consider liver function tests, electrolytes, and a CBC.

## **Emergency Department Care and Disposition**

1. Consider **multidose activated charcoal** after ingestion of enteric-coated, delayed-release preparations and measure serial concentrations due to delayed peak serum concentration. Consider whole-bowel irrigation in extended-release preparations.
2. **L-carnitine**, 50 milligrams/kilogram/d, may hasten recovery in patients with acute intoxication and increase survival from hepatotoxicity.
3. **Hemoperfusion** and **hemodiafiltration** can be used to treat severe overdose.
4. All symptomatic patients require admission, while asymptomatic patients with declining serum levels can be discharged.

## **SECOND-GENERATION ANTICONVULSANTS**

As a group, the second-generation anticonvulsants possess little toxicity in acute overdose.

- **Felbamate** may cause aplastic anemia and hepatic failure and can crystallize in the kidney, leading to acute renal failure in large overdose.
- **Gabapentin** may cause drowsiness, ataxia, nausea, and vomiting that generally resolve in about 10 hours.
- **Lacosamide** may cause dizziness, headache, nausea, and diplopia in therapeutic use.
- **Lamotrigine** has been associated with autoimmune reactions in therapeutic use and drowsiness, vomiting, ataxia, and dizziness in overdose. Seizures, coma, cardiac toxicity (QRS and QT-interval prolongation), and acute pancreatitis have been reported. Treatments include sodium bicarbonate and IV lipid.
- **Levetiracetam** may cause lethargy, coma, and respiratory depression.
- **Oxcarbazepine** may cause hyponatremia and a drug rash in therapeutic use.
- **Pregabalin** has been reported to cause somnolence and dizziness in long-term therapeutic use. Overdose may cause depressed level of consciousness.
- **Rufinamide** may cause headache, dizziness, fatigue and somnolence in long-term therapy.
- **Tiagabine** may cause the rapid neurologic toxicity, including lethargy, coma, seizures, myoclonus, muscular rigidity, and delirium.
- **Topiramate** may cause nephrolithiasis and glaucoma in therapeutic use. In overdose, somnolence, vertigo, agitation, mydriasis, and seizures have been reported. It can produce a metabolic acidosis, which can last up to 7 days due to the long half-life of the drug.
- **Zonisamide** may promote renal stone formation and cause a drug rash in therapeutic use.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 191, “Anticonvulsants” by Frank LoVecchio and Jennifer C. Smith.

# CHAPTER 110

## Iron

O. John Ma

### CLINICAL FEATURES

Iron toxicity from an intentional or accidental ingestion is a common poisoning. Based on clinical findings, iron poisoning can be divided into 5 stages.

The first stage develops within the first few hours of ingestion. Direct irritative effects of iron on the gastrointestinal (GI) tract produce abdominal pain, vomiting, and diarrhea. Vomiting is the clinical sign most consistently associated with acute iron toxicity. The absence of these symptoms within 6 hours of ingestion essentially excludes a diagnosis of significant iron toxicity.

During the second stage, which may continue for up to 24 hours following ingestion, the patient's GI symptoms may resolve, providing a false sense of security despite toxic amounts of iron absorption. While patients may be asymptomatic, they often appear ill, and may have abnormal vital signs reflecting hypovolemia and metabolic acidosis.

The third stage may appear early or develop hours after the second stage as shock and a metabolic acidosis evolve. Iron-induced coagulopathy may cause bleeding and worsen hypovolemia. Hepatic dysfunction, cardiomyopathy, and renal failure may also develop.

The fourth stage develops 2 to 5 days after ingestion and is characterized by elevation of aminotransferase levels and possible progression to hepatic failure.

The fifth stage, which occurs 4 to 6 weeks after ingestion, reflects the corrosive affects of iron on the pyloric mucosa and may cause gastric outlet obstruction.

### DIAGNOSIS AND DIFFERENTIAL

The diagnosis of iron poisoning is based on the clinical picture and the history provided by the patient, significant others, or EMS providers. When determining a patient's potential for toxicity, the total amount of elemental iron must be used in calculations. [Table 110-1](#) reviews the predicted clinical effects based on the amount of iron ingested.



Predicted Clinical Effects	Elemental Iron Dose*	Serum Iron Concentration <sup>†</sup>
Nontoxic or mild GI symptoms	<20 milligrams/kilogram	<300 micrograms/dL
Expected significant GI symptoms and potential for systemic toxicity	20 to 60 milligrams/kilogram	300 to 500 micrograms/dL
Moderate to severe systemic toxicity	>60 milligrams/kilogram	>500 micrograms/dL
Severe systemic toxicity and increased morbidity	—	>1000 micrograms/dL

\*Elemental iron dose by history.

<sup>†</sup>Serum iron concentration obtained within 4 to 6 hours of ingestion.

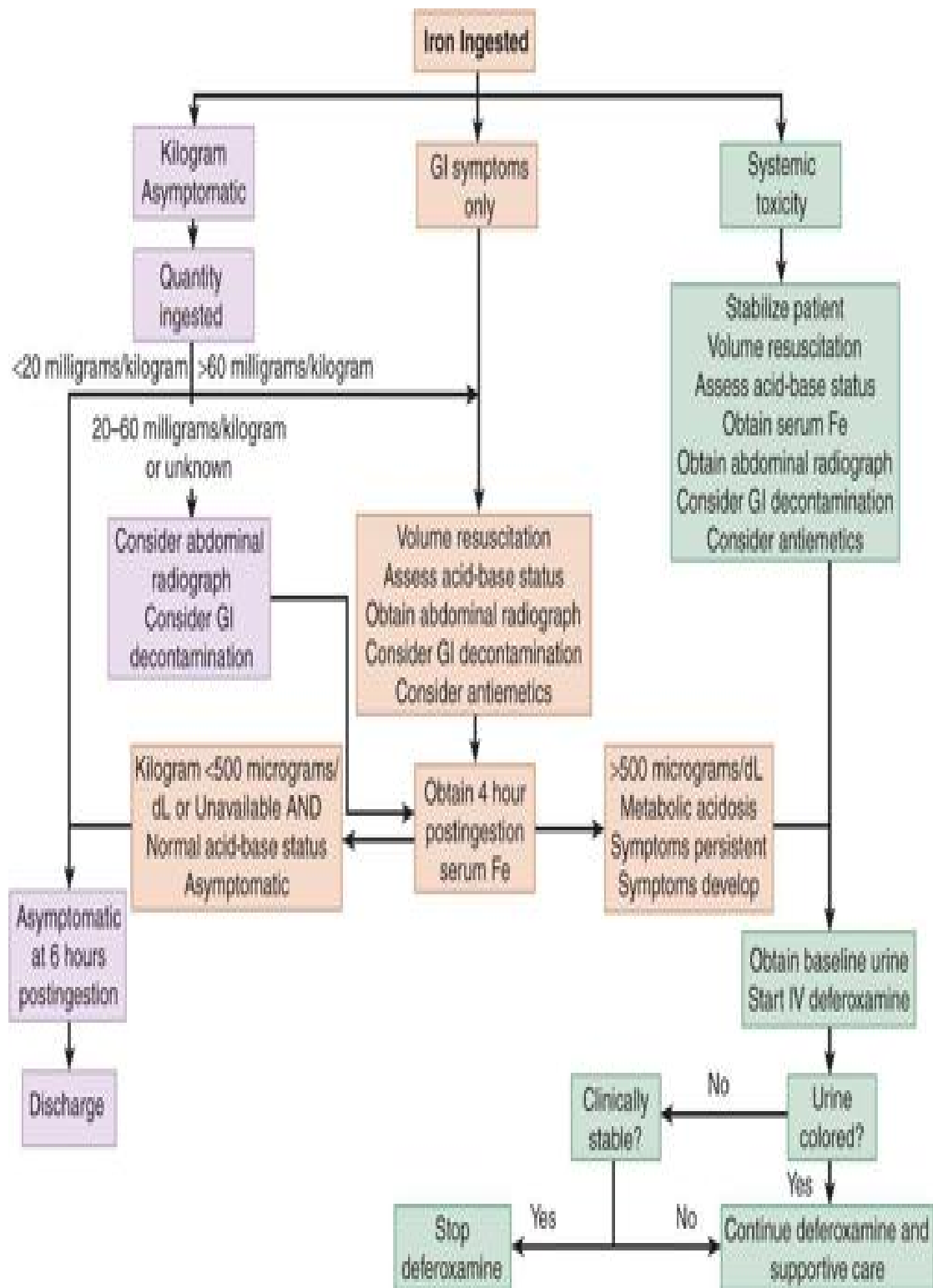
**TABLE 110-1** Predicted Toxicity of Iron Ingestion

Laboratory evaluation includes serum electrolytes, renal studies, serum glucose, coagulation studies, complete blood count, hepatic enzymes, and a serum iron level. It is crucial to note that a single serum iron level does not reflect what iron levels have been previously, what direction they are going, or the degree of iron toxicity in tissues; a single low serum level does not exclude the diagnosis of iron poisoning since there are variable times to peak level following ingestion of different iron preparations. Serum iron levels have limited use in directing management since toxicity is primarily intracellular rather than in the blood. The total iron binding capacity (TIBC) becomes falsely elevated in the presence of elevated serum iron levels or deferoxamine, and is of no clinical value.

Plain radiographs may reveal iron in the GI tract; however, many iron preparations are not radiopaque so normal radiographs do not exclude iron ingestion.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Patients who have remained asymptomatic for 6 hours after ingestion of iron, have not ingested a potentially toxic amount, and who have a normal physical examination do not require medical treatment for iron toxicity. Patients whose symptoms resolve after a short period of time, and who have normal vital signs, usually have mild toxicity and require only supportive care. This subset of patients still requires an observation period. [Figure 110-1](#) is an algorithm for the clinical management of patients after an acute iron ingestion.



**FIGURE 110-1.** Algorithm for clinical management of patients following iron ingestion.

Patients who are symptomatic or demonstrate signs of hemodynamic instability after iron ingestion require aggressive management in the ED.

1. Place the patient on supplemental oxygen and a cardiac monitor, and establish 2 large-bore IVs.
2. Administer vigorous **intravenous (IV) crystalloid infusion** to help correct hypovolemia and hypoperfusion.
3. Perform **gastric lavage** in patients who present within 60 min of ingestion. Activated charcoal is not recommended.
4. Whole-bowel irrigation with a **polyethylene glycol** solution is efficacious. Administration of 250 to 500 mL/h in children or 2 L/h in adults via nasogastric tube may clear the GI tract of iron pills before absorption occurs.
5. Administer antiemetics such as **ondansetron** (4 milligrams IV in adults; 0.1 milligrams/kilogram to a maximum dose of 4 milligrams in pediatric patients) or **promethazine** 25 milligrams IV in adults.
6. Correct coagulopathy with vitamin **K<sub>1</sub>** (5 to 10 milligrams SC) and **fresh frozen plasma** (10 to 25 mL/kg in adults; 10 mL/kg in pediatric patients). Order blood for type and screen or crossmatch as necessary.
7. **Deferoxamine** is a chelating agent that can remove iron from tissues and free iron from plasma. Deferoxamine is safe to administer to children and pregnant women. Deferoxamine therapy is indicated in patients with systemic toxicity, metabolic acidosis, worsening symptoms, or a serum iron level predictive of moderate or severe toxicity.

Intravenous infusion is the preferred route of deferoxamine administration because IM absorption is unpredictable in the hypovolemic patient. The recommended initial dose is 1000 milligrams IV. Since hypotension is the rate-limiting factor for IV infusion, it is recommended to **begin with a slow IV infusion at 5 milligrams/kilogram/ hour**. The deferoxamine infusion rate can be increased to **15 milligrams/kilogram/h**, as tolerated, within the first hour of treatment. The recommended total amount of deferoxamine is 360 milligrams/kilogram or 6 grams during the first 24 hours. Initiate deferoxamine therapy without waiting for the serum iron level in any clinically ill patient with a known iron ingestion.

Evaluate the efficacy of deferoxamine treatment through serial urine samples. As ferrioxamine is excreted, urine changes to a classic *vin rose* appearance. Clinical recovery is the most important factor guiding the termination of deferoxamine therapy.

8. Patients who remain asymptomatic after 6 hours of observation, have a normal physical examination, and have a reliable history of an insignificant ingestion may

be considered for discharge. Patients initially symptomatic who become asymptomatic should be admitted for further evaluation since this may represent the second stage of iron toxicity. Admit all patients who receive deferoxamine therapy to an intensive care setting. Assess all patients for suicide risk. Consider child abuse or neglect in pediatric cases.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 192, "Iron," by Stephanie H. Hernandez and Lewis S. Nelson.

# CHAPTER 111

## Hydrocarbons and Volatile Substances

Allyson A. Kreshak

Products containing hydrocarbons are found in many household and workplace settings and include fuels, lighter fluids, paint removers, pesticides, polishers, degreasers, and lubricants. Some volatile substances may be recreationally abused. Exposure may cause mild to severe toxicity and, rarely, sudden death.

### CLINICAL FEATURES

Toxicity depends on route of exposure, physical characteristics, chemical characteristics, and the presence of toxic additives (eg, lead or pesticides). See [Table 111-1](#) for clinical features.

System	Clinical Manifestations
Pulmonary	Tachypnea, grunting respirations, wheezing, retractions
Cardiac	Ventricular dysrhythmias (may occur after exposure to halogenated hydrocarbons and aromatic hydrocarbons)
Central nervous	Slurred speech, ataxia, lethargy, coma
Peripheral nervous	Chronic numbness and paresthesias in the extremities
GI and hepatic	Nausea, vomiting, abdominal pain, loss of appetite (mostly with halogenated hydrocarbons)
Renal and metabolic	Muscle weakness or paralysis secondary to hypokalemia in patients who abuse toluene
Hematologic	Lethargy (anemia), shortness of breath (anemia), neurologic depression/syncope (carbon monoxide from methylene chloride), cyanosis (methemoglobinemia from amine-containing hydrocarbons)
Dermal	Local erythema, papules, vesicles, generalized scarlatiniform eruption, exfoliative dermatitis, "huffer's rash," cellulitis

**TABLE 111-1** Clinical Manifestations of Hydrocarbon Exposure

Chemical pneumonitis is the most common pulmonary complication, and is most likely to occur following aspiration of a hydrocarbon with low

viscosity, high volatility, and low surface tension. Symptoms occur quickly and include cough and dyspnea. Physical examination may reveal tachypnea, wheezing, grunting, and an elevated temperature. Radiographic findings may lag behind the clinical picture by 4 to 24 hours; however, most radiographic abnormalities are apparent within 6 hours. Less common pulmonary complications include pneumothorax, pneumomediastinum, and pneumatocele.

Cardiac toxicity manifests as potentially lethal dysrhythmias resulting from myocardial sensitization to circulating catecholamines (“sudden sniffing death syndrome”). Halogenated hydrocarbon solvents are most frequently implicated.

Central nervous system toxicity may present as intoxication, ranging from initial giddiness, agitation, and hallucinations to seizures, slurred speech, ataxia, and coma. Chronic exposure may cause recurrent headaches, cerebellar ataxia, and mood lability.

Gastrointestinal toxicity can include vomiting (which can lead to aspiration), abdominal pain, anorexia, and hepatic damage (particularly from halogenated hydrocarbons).

Dermal toxicity includes contact dermatitis and blistering with progression to full-thickness burns. Injection of hydrocarbons can cause tissue necrosis.

Less common acute toxicities include hematologic disorders such as hemolysis, methemoglobinemia, carboxyhemoglobinemia (from methylene chloride), and renal disorders.

## **DIAGNOSIS AND DIFFERENTIAL**

Diagnosis is made by history and physical examination findings, bedside monitoring, laboratory tests, and chest radiograph. An abdominal radiograph may reveal ingestion of radiopaque substances (eg, chlorinated hydrocarbons).

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Secure the airway and maintain ventilation support. Administer oxygen to symptomatic patients and place them on a cardiac monitor (see [Table 111-2](#)).

<b>Airway and breathing</b>	Secure airway. Antidotes: Administer oxygen for carboxyhemoglobinemia and methylene blue for methemoglobinemia. Provide supplemental oxygen. Administer inhaled $\beta_2$ -agonists. Ventilatory support: Provide positive end-expiratory pressure or continuous positive airway pressure as needed to achieve adequate oxygenation.
<b>Cardiac</b>	Circulation: Administer IV crystalloid fluid for initial volume resuscitation of hypotensive patients. Do not use catecholamines in cases of halogenated hydrocarbon exposure. Consider propranolol, esmolol, or lidocaine for ventricular dysrhythmias induced by halogenated hydrocarbon exposure. Consult the poison control center, toxicologist, and other appropriate specialists as needed.
<b>Decontamination</b>	Dermal: Remove hydrocarbon-soaked clothes, decontaminate skin with soap and water, decontaminate eyes with saline irrigation. GI: Not indicated for nonhalogenated hydrocarbon ingestion. Consider nasogastric aspiration or activated charcoal administration if a toxic halogenated or aromatic hydrocarbon was ingested within 1 hour prior to presentation and airway is protected.
<b>Other</b>	Laboratory tests: Order complete blood count, basic metabolic panel, liver function tests (serum transaminase, bilirubin, albumin levels), prothrombin time, partial thromboplastin time, carboxyhemoglobin level, methemoglobin level, and/or radiologic studies as indicated (see text). Correct electrolyte abnormalities. Tar removal: Commercial solvents and ointments containing polyoxyethylene sorbitan monolaurate (Polysorbate) or petrolatum may work to remove tar from the skin. Administer blood products as needed.

**TABLE 111-2** Management of Hydrocarbon Exposures

2. Treat hypotension with intravenous crystalloid infusion. Avoid catecholamines except in cases of cardiac arrest. Treat tachydysrhythmias with propranolol, esmolol, or lidocaine. Avoid class IA and III agents.
3. Follow standard hazardous material measures for decontamination of the patient. Most hydrocarbon ingestions do not require GI decontamination. GI decontamination within 1 hour of ingestion is suggested only for a few

hydrocarbons. Check with the local Poison Control Center before attempting GI decontamination as this can lead to further aspiration of volatile substances.

4. Meticulous wound care with potential surgical debridement is indicated for dermal exposures. Treat tar and asphalt injuries with debridement of blistered skin and application of petrolatum ointment or polyoxyethylene sorbitan monolaurate (Polysorbate).
5. Hyperbaric oxygen therapy may be indicated for patients who develop significant carbon monoxide toxicity after exposure to methylene chloride.
6. Admit symptomatic patients, those exposed to hydrocarbons capable of producing delayed toxicity (eg, halogenated hydrocarbons), and those exposed to hydrocarbons with toxic additives (eg, pesticides or organic metal compounds). Patients with a normal chest radiograph who remain asymptomatic after 6 to 8 hours of observation may be discharged with strict return precautions.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 193, "Hydrocarbons and Volatile Substances," by Paul M. Wax and Stella C. Wong.



# CHAPTER 112

## Caustics

Christian A. Tomaszewski

Caustics are substances that can cause histological and functional damage on contact and include both alkalis ( $\text{pH} > 7$ ) and acids ( $\text{pH} < 7$ ). The most common alkali exposure is household bleach (sodium hypochlorite with hydroxide), which is usually benign except in intentional ingestions. The most common acid exposures are sulfuric acid (drain cleaners) and hydrochloric acid (automobile batteries and masonry cleaners).

### CLINICAL FEATURES

Common features of caustic ingestions include dysphagia, odynophagia, epigastric pain, and vomiting with gastrointestinal (GI) tract injuries. Dysphonia, stridor, and respiratory distress can be seen with laryngotracheal injury. Esophageal injuries are graded by direct visualization: (1) edema and hyperemia; (2) ulcerations, blisters and exudates (2a-noncircumferential; 2b-circumferential); (3) deep ulceration and necrosis. Intentional ingestions are associated with higher-grade injury that can lead to the development of strictures. Most ingestions with serious injury are symptomatic with stridor, drooling, or vomiting, although distal GI injury without oral or facial burns is possible. Disc battery ingestions may be asymptomatic, though batteries  $> 15$  mm in diameter can become lodged in the esophagus and cause pressure necrosis.

Caustic exposures to the cornea are particularly serious if they involve alkalis. Dermal exposures to caustics usually produce only local pain and irritation. However, alkali and sodium hydrofluoric acid burns can penetrate deeply and lead to liquefactive necrosis. Hydrofluoric acid can cause systemic hypocalcemia, hypomagnesemia, and hyperkalemia with subsequent ventricular dysrhythmias.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis is clinical. Routine laboratory tests are recommended for severely affected patients and include electrolytes, assessment of acid-base status, and monitoring for potential gastrointestinal blood loss. Monitor serum calcium and magnesium levels and perform an ECG in patients with hydrofluoric acid exposures, especially ingestions. Consider chest and/or abdominal radiographs in symptomatic caustic ingestions to assess for free air or to investigate for foreign body in cases of suspected disc battery ingestion. Noncontrast CT of the chest and abdomen may be useful if perforated viscus is suspected, especially after ingestion of strong acids. Early endoscopic evaluation (<12 to 24 hours post ingestion) is indicated for intentional caustic ingestions, and unintentional cases presenting with stridor, oral burns, vomiting, drooling, or inability to tolerate oral intake.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Focus treatment on decontamination, early anticipatory airway management, stabilization of hemodynamic status, and delineation of extent of injury.

### **Decontamination**

1. Remove contaminated clothing and irrigate exposed skin with copious amounts of water. Alkali burns may require local debridement and removal of devitalized tissue followed by additional irrigation.
2. Perform aggressive ocular decontamination with normal saline for a minimum of 15 min with frequent monitoring of ocular pH until a pH of 7.5 to 8.0 is achieved.
3. Gastric decontamination in the form of activated charcoal, ipecac, or gastric lavage is contraindicated. Intentional strong acid ingestions may

benefit from gastric decontamination with a nasogastric tube if performed within 30 min of ingestion.

4. Dilution or neutralization is generally reserved for immediate prehospital or home care of the unintentional pediatric ingestion and is not recommended more than 30 min post-ingestion.

## Supportive Care

1. Perform early **awake oral intubation** with direct visualization in symptomatic patients with stridor, significant drooling, or dysphonia. Blind nasotracheal intubation is contraindicated.
2. Obtain IV access and administer isotonic IV fluids for hypotension.
3. Obtain surgical consultation for suspected or confirmed peritonitis or free air.

## Special Considerations

1. Treat hydrofluoric acid dermal exposures with topical **calcium gluconate gel** (3.5 grams mixed with 150 mL water-soluble lubricant). Consider intradermal **5% calcium gluconate** for large burns and **calcium gluconate** infusion into the radial artery over 4 hours or given as a Bier block (10 mL of 10% calcium gluconate in 40 mL saline or 5% dextrose) for refractory distal extremity burns.
2. Oral ingestions of hydrofluoric acid within an hour can be suctioned via nasogastric tube followed by instillation of up to 300 mL of 10% **calcium gluconate**. High doses of IV calcium and magnesium may be needed to treat systemic deficiencies and dysrhythmias.
3. Disc batteries lodged in the esophagus require **emergent endoscopic removal**.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 194, “Caustics,” by Nicole C. Brouchard and Wallace A. Carter.

# CHAPTER 113

## Pesticides

Christian A. Tomaszewski

Pesticides include insecticides, herbicides, and rodenticides. In addition to active ingredients, many also contain “inert” products such as petroleum distillates, which may be toxic as well. Although the mainstay of treatment is supportive care, some antidotes are essential.

## INSECTICIDES

### Clinical Features

Organophosphorus insecticides include diazinon, acephate, malathion, parathion, and chlorpyrifos. Absorption occurs through ingestion, inhalation (eg, nerve gas agents), and dermal routes. Toxicity is produced through binding to acetylcholinesterase, which becomes irreversible within hours, causing excess stimulation of acetylcholine receptors; this results in a cholinergic crisis known as “sludging” ([Table 113-1](#)). Most patients become symptomatic within 8 hours of dermal exposure, though some fat-soluble agents (eg, fenthion) can cause delayed symptoms. Nicotinic stimulation leads to fasciculations and muscle weakness, which is most pronounced in the respiratory muscles, worsening the pulmonary muscarinic effects. Nicotinic effects can also cause paradoxical tachycardia and mydriasis. Central nervous system (CNS) effects, which often predominate in children, include tremor, restlessness, confusion, seizures, and coma.

S	Salivation
L	Lacrimation
U	Urinary incontinence
D	Defecation
G	GI pain
E	Emesis
D	Defecation
U	Urination
M	Muscle weakness, miosis
B	Bradycardia, bronchorrhea, bronchospasm
E	Emesis
L	Lacrimation
S	Salivation
Killer Bees	Bradycardia, bronchorrhea, bronchospasm

**TABLE 113-1** SLUDGE, DUMBELS, and “Killer Bees” Mnemonics for the Muscarinic Effects of Cholinesterase Inhibition

A variety of sub-acute and chronic effects are associated with organophosphorus insecticide poisoning. An intermediate syndrome, 1 to 4 days after acute poisoning, may present with paralysis or weakness of neck, facial, and respiratory muscles, which can result in respiratory arrest if not treated. Organophosphate-induced delayed neuropathy can occur 1 to 3 weeks after acute poisoning, resulting in a distal motor-sensory polyneuropathy with leg weakness and paralysis.

## Diagnosis and Differential

Organophosphate poisoning is typically a clinical diagnosis based on the toxidrome ([Table 113-1](#)), with confirmation available through laboratory cholinesterase assay. An ECG may be useful to monitor for prolonged QT, which is associated with increased morbidity and mortality in organophosphate poisoning.

## Emergency Department Care and Disposition

Treatment of organophosphate poisoning is listed in [Table 113-2](#), and should not be delayed pending confirmatory tests.

Decontamination	Protective clothing must be worn to prevent secondary poisoning of health care workers. Handle and dispose of all clothes as hazardous waste. Wash patient with soap and water. Handle and dispose of water runoff as hazardous waste.
Monitoring	Cardiac monitor, pulse oximeter, 100% oxygen.
Gastric lavage	No proven benefit.
Activated charcoal	No proven benefit.
Urinary alkalinization	No proven benefit.
Atropine	1 milligram or more IV in an adult or 0.01 to 0.04 milligram/kilogram (but never <0.1 milligram) IV in children. Repeat every 5 min until tracheobronchial secretions attenuate.
Pralidoxime	1 to 2 grams for adults or 20 to 40 milligrams/kilogram (up to 1 gram) in children, mixed with normal saline and infused IV over 5 to 10 min. Continuous infusion often necessary.
Seizures	Benzodiazepines.

**TABLE 113-2** Treatment for Organophosphate Poisoning

1. In symptomatic patients, administer 100% oxygen and focus on airway management, with gentle suctioning of secretions. The use of succinylcholine for intubation, which is metabolized by plasma cholinesterase, may result in prolonged paralysis.
2. The key in treatment is large amounts of **atropine** titrated to attenuation of tracheobronchial secretions. Tachycardia and dilated pupils are not a contraindication to atropine. High dose **diphenhydramine** can be substituted for atropine.
3. Minimal exposures require only 6 to 8 hours observation. Recrudescence can occur due to reexposure to contaminated clothing, particularly leather. Significant poisonings require intensive care monitoring.
4. Other commonly encountered pesticides and their treatment are listed in [Table 113-3](#).

Agent	Example	Clinical Feature	Diagnosis	Treatment
Carbamates	Carbaryl, pirimicarb, propoxur, trimethacarb	Similar to organophosphates	Depressed cholinesterase levels	Atropine
Organochlorines	Chlordane, heptachlor, dieldrin, aldrin	Neurological: excitability, seizures	History, special lab	Benzodiazepine, cooling
Pyrethroids	Pyrethrins	Allergic hypersensitivity	History	Bronchodilators and antihistamines
Neonicotinoids	Imidacloprid	Nausea, headache, sedation	History	Supportive
<i>N,N</i> -Diethyl-3-methylbenzamide	DEET	Seizures	History	Benzodiazepines

**TABLE 113-3** Nonorganophosphate Insecticide Poisoning

## HERBICIDES

Herbicides are agents used to kill weeds. In addition to intrinsic toxicity, they may also be packaged with surfactants or solvents with their own toxic effects. The most dangerous are bipyridyl herbicides, namely paraquat and diquat. Nonbipyridyl herbicides and their treatment are depicted in [Table 113-4](#).

Agent	Example	Clinical Feature	Treatment
Chlorophenoxy	2,4-dichlorophenoxyacetic acid	Mucous membrane irritation, pulmonary edema, muscle toxicity, hyperthermia	Urine alkalization Hemodialysis
Glyphosate		Mucous membrane irritation, erosions, multiorgan failure, respiratory distress	Observe asymptomatic patients for 6 hours
Urea-substituted	Chlorimuron, diuron, fluometuron, isoproturon	Methemoglobinuria	Methylene blue
Organophosphates	Butiphos	SLUDGE	Same as pesticides
<i>N,N</i> -Diethyl-3-methylbenzamide	DEET	Seizures	Benzodiazepines

**TABLE 113-4** Nonbipyridyl Herbicides

Paraquat is especially toxic with lethal oral doses of 20% solution being only 10 to 20 mL in an adult and 4 to 5 mL in children. Both agents are toxic via inhalation, dermal exposure, or ingestion. Due to their caustic properties, ulceration of skin and mucous membranes and gastrointestinal corrosion can occur. Paraquat ingestions result in renal, cardiac, and hepatic failure along with progressive pulmonary fibrosis. Due to the latter, treatment often includes restriction of supplemental oxygen. Decontamination of skin is important to prevent continued absorption. Early after ingestion, GI decontamination with **activated charcoal**, **fuller's earth**, or **bentonite** may be helpful. Charcoal hemoperfusion may remove paraquat. Because of poor prognosis, especially with paraquat ingestions, admit patients with ingestion for further observation and treatment.

## **RODENTICIDES**

The most commonly used rodenticides are superwarfarins, including bordifacoum, diphenacoum, and bormadoline. Coagulopathy typically develops within 48 hours and lasts for weeks to months due to the long half-lives of these agents. Single ingestions in children usually do not result in toxic effects. Acute intentional or repeated ingestions may present with delayed hemorrhage, including hematuria, gastrointestinal hemorrhage, or epistaxis. Screening for toxic effect can be performed with an INR obtained 24 to 48 hours after ingestion. If the INR is elevated ( $> 2$ ), then start oral **vitamin K<sub>1</sub>**, typically at doses of 20 milligrams a day in adults (1 to 5 milligrams for children) and continue for up to 10 months. Acute hemorrhage requires more aggressive therapy with fresh frozen plasma, IV vitamin **K<sub>1</sub>**, and addition of **prothrombin complex** or **recombinant activated factor VII for refractory hemorrhage**. Nonanticoagulant rodenticides are described in [Table 113-5](#).



Agent	Toxicity	Clinical Effects	Treatment
Arsenic	Severe	Vomiting and diarrhea, cardiovascular collapse	Succimer
Barium carbonate	Severe	Vomiting and diarrhea, dysrhythmia, respiratory failure, paralysis	Gastric lavage with sodium or magnesium sulfate; potassium replacement
Phosphorus	Severe	Burns, cardiovascular collapse	Lavage with potassium permanganate
<i>N</i> -3-Pyridylmethyl- <i>N'</i> - <i>p</i> -nitrophenyl urea (Vacor)	Severe	GI symptoms, insulin deficient hyperglycemia and DKA	Nicotinamide IV
Sodium fluoroacetate	Severe	Vomiting, respiratory depression, pulmonary edema	Supportive care
Strychnine	Severe	Awake seizure like activity	Benzodiazepines
Thallium	Severe	Early GI irritation, respiratory failure and dysrhythmias	Oral Prussian blue
Zinc phosphide	Severe	Vomiting, shock, hypocalcemia	Intragastric alkalization
$\alpha$ -Naphthyl-thiourea	Moderate	Pulmonary edema	Supportive care
Cholecalciferol	Moderate	Hypercalcemia	Saline, furosemide, steroids
Bromethalin	Low	Tremors, focal seizures	Benzodiazepines
Norbormide	Low	Vasoconstrictive tissue hypoxia	Supportive care
Red squill	Low	Vomiting, diarrhea, hyperkalemia, heart block with ventricular dysrhythmias	Supportive care

**TABLE 113-5** Nonanticoagulant Rodenticides

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 195, “Pesticides,” by Walter C. Robey III and William J. Meggs.

# CHAPTER 114

## Metals and Metalloids

D. Adam Algren

Metal poisoning often results from occupational, recreational, or environmental exposure and results in a combination of neurologic, cardiovascular, gastrointestinal, hematologic, and renal findings.

### LEAD POISONING

#### Clinical Features

Lead is the most common cause of chronic metal poisoning and manifests as signs and symptoms affecting a variety of organ systems ([Table 114-1](#)).

System	Clinical Manifestations
Central nervous system	Acute toxicity: encephalopathy, seizures, altered mental status, papilledema, optic neuritis, ataxia Chronic toxicity: headache, irritability, depression, fatigue, mood and behavioral changes, memory deficit, sleep disturbance
Peripheral nervous system	Paresthesias, motor weakness (classic is wrist drop), depressed or absent deep tendon reflexes, sensory function intact
GI	Abdominal pain (mostly with acute poisoning), constipation, diarrhea, toxic hepatitis
Renal	Acute toxicity: Fanconi syndrome (renal tubular acidosis with aminoaciduria, glucosuria, and phosphaturia) Chronic toxicity: interstitial nephritis, renal insufficiency, hypertension, gout
Hematologic	Hypoproliferative and/or hemolytic anemia; basophilic stippling (rare and nonspecific)
Reproductive	Decreased libido, impotence, sterility, abortions, premature births, decreased or abnormal sperm production

**TABLE 114-1** Common Manifestations of Lead Poisoning

## **Diagnosis and Differential**

Suspect lead poisoning in any individual demonstrating a combination of abdominal pain, nausea, vomiting, and neurologic symptoms (particularly children with encephalopathy), especially in the setting of anemia. A CBC may demonstrate normocytic or microcytic anemia with hemolysis and basophilic stippling; however, hematologic findings are neither sensitive nor specific for lead poisoning. Lead toxicity is confirmed by an elevated blood lead level, though results are often not immediately available. Radiographs may identify metaphyseal long bone lead lines, radiopaque material in the GI tract, or retained bullet fragments.

## **Emergency Department Care and Disposition**

1. Address life-threatening ventilatory and circulatory problems through advanced airway management and fluid resuscitation. Avoid lumbar puncture in the setting of lead induced encephalopathy as this can precipitate herniation.
2. Decontaminate the GI tract with **whole-bowel irrigation** using polyethylene glycol solution in those with retained lead in the GI tract.
3. Chelation therapy is the mainstay of treatment and often must be started empirically ([Table 114-2](#)).

Severity [blood lead level (micrograms/dL)]	Dose
Encephalopathy	Dimercaprol, 75 milligrams/m <sup>2</sup> (or 4 milligrams/kilogram) IM every 4 h for 5 d <i>and</i> Edetate calcium disodium, 1500 milligrams/m <sup>2</sup> /d, for 5 d as a continuous IV infusion started 4 h after dimercaprol
Symptomatic Adults: blood lead > 100 Children: blood lead > 69	Dimercaprol <i>and</i> Edetate calcium disodium (as described above) <i>or</i> Edetate calcium disodium (alone) <i>or</i> Succimer (as described below)
Asymptomatic Adults: blood lead 70 to 100 Children: blood lead 45 to 69	Succimer, 350 milligrams/m <sup>2</sup> (or 10 milligrams/kilogram) PO every 8 h for 5 d, then every 12 h for 14 d
Asymptomatic Adults: blood lead < 70 Children: blood lead < 45	Routine chelation not indicated Remove patient from source of exposure

\*General guidelines. Consult with medical toxicologist or regional poison center for specifics and dosing.

**TABLE 114-2** Guidelines for Chelation Therapy in Lead-Poisoned Patients\*

- Admit patients requiring parenteral chelation therapy or those who cannot avoid further environmental lead exposure. Arrange follow up for patients started on succimer.

## ARSENIC POISONING

### Clinical Features

Arsenic is used in a variety of insecticides and herbicides as well as mining and smelting processes. Acute ingestion results in a profound vomiting and diarrhea within hours of exposure. Hypotension and tachycardia may develop secondary to hypovolemia and direct myocardial dysfunction. Encephalopathy, pulmonary edema, and acute renal failure have been described. Chronic poisoning causes peripheral neuropathy, malaise, and confusion. Skin findings include alopecia, hyperpigmentation, keratoses, and transverse white nail lines (Mees lines).

## Diagnosis and Differential

An exposure history is most useful in identifying arsenic poisoning. However, consider the diagnosis in patients with hypotension preceded by profound vomiting and diarrhea. An ECG may show QT-interval prolongation. The diagnosis is confirmed by demonstrating an elevated 24-hour urine arsenic level. Other diagnoses to consider include septic shock, encephalopathy, peripheral neuropathy, Addison disease, and lead, thallium, or mercury poisoning.

## Emergency Department Care and Disposition

1. Support the ABCs: endotracheal intubation may be required to protect the airway; treat hypotension with volume resuscitation and vasopressors, but avoid over resuscitation that may result in pulmonary or cerebral edema. Manage dysrhythmias according to ACLS/PALS protocols, but avoid agents that prolong the QT interval (class IA, IC and III anti-dysrhythmics). Magnesium sulfate or isoproterenol should be considered for treatment of torsades de pointes.
2. Decontaminate the gastrointestinal tract with **whole-bowel irrigation** using polyethylene glycol solution in those with radiopaque GI fragments.
3. Treat symptomatic patients empirically with chelation therapy ([Table 114-3](#)) using **dimercaprol (BAL) IM**. **Succimer (DMSA) PO** is preferred for clinically stable patients who can tolerate oral intake.

Chelator	Dose
Dimercaprol	3 to 5 milligrams/kilogram IM every 4 h for 2 d, followed by 3 to 5 milligrams/kilogram IM every 6 to 12 h until able to switch to succimer
Succimer	10 milligrams/kilogram PO every 8 h for 5 d, followed by 10 milligrams/kilogram PO every 12 h

**TABLE 114-3** Guidelines for Chelation Therapy in Arsenic-Poisoned Patients

4. Hospitalize symptomatic patients. Discharge stable patients with sub-acute or chronic poisoning if follow-up is ensured.

## **MERCURY POISONING**

Mercury poisoning can result from exposure to elemental, inorganic, or organic mercury compounds.

### **Clinical Features**

*Elemental mercury* exposure is most likely to occur after contact with a broken thermometer, and mercury is primarily absorbed via inhalation (especially with heating or vacuuming). Ingestions of elemental mercury are nontoxic in those with normal GI tracts. Vapor exposure results in cough, fever, dyspnea, vomiting, and headache. Acute lung injury can progress to respiratory failure. *Inorganic mercury* is used as a disinfectant and in manufacturing. Ingestion of inorganic mercury results in corrosive injury to the GI tract, with vomiting, diarrhea, abdominal pain, and GI bleeding early, followed by acute renal failure. *Organic mercury* is found in some fungicides and pesticides and can be absorbed when ingested. Poisoning tends to occur with chronic exposures and results in profound central nervous system dysfunction.

### **Diagnosis and Differential**

A history of exposure to mercury is key to diagnosis and is confirmed by an elevated 24-hour urine mercury level when toxicity is due to elemental or inorganic mercury; an elevated whole blood mercury level is necessary in cases of organic mercury exposure. The differential diagnosis is extensive and includes causes of metal fume fever (elemental), encephalopathy or tremor (elemental/inorganic/organic). Consider alternative causes of corrosive gastroenteritis (ingestion of iron, arsenic, phosphorus, acids, and alkalis) if mercury salt ingestion is suspected.

## Emergency Department Care and Disposition

1. Support the ABCs with airway management and crystalloid infusion.
2. Consider gastric lavage in cases of *inorganic* mercury ingestion or activated charcoal in cases of *organic* mercury ingestion.
3. Begin chelation therapy prior to confirming the diagnosis ([Table 114-4](#)). **Dimercaprol (BAL)** is preferred for *elemental* and *inorganic* mercury poisonings. **Succimer (DMSA)** is the agent of choice in *organic* mercury poisonings and in cases of mild or chronic elemental/inorganic mercury toxicity.

	Inorganic Mercury, Elemental Mercury	Organic Mercury
Severe poisoning	Dimercaprol, 5 milligrams/kilogram IM every 4 h for 2 d, followed by 2.5 milligrams/kilogram IM every 6 h for 2 d, followed by 2.5 milligrams/kilogram IM every 12 to 24 h until clinical improvement occurs or until able to switch to succimer therapy	Succimer, 10 milligrams/kilogram PO every 8 h for 5 d, then every 12 h for 14 d
Mild poisoning and chronic poisoning	Succimer, 10 milligrams/kilogram PO every 8 h for 5 d, then every 12 h for 14 d	No proven benefit for chelation therapy

**TABLE 114-4** Guidelines for Chelation Therapy in Mercury-Poisoned Patients

4. Admit patients with respiratory symptoms following mercury vapor exposure and those with inorganic mercury ingestions.

## POISONING WITH OTHER METALS

Less common toxic heavy metals include bismuth, cadmium, chromium, cobalt, copper, silver, thallium, and zinc. Unique manifestations and treatments of these exposures are outlined in [Table 114-5](#).

<b>Metal</b>	<b>Poisoning Source</b>	<b>Acute Clinical Manifestations</b>	<b>Chronic Clinical Manifestations</b>	<b>Specific Treatment</b>
Bismuth	Antidiarrheals (bismuth subsalicylate), impregnated surgical packing paste	Abdominal pain, acute renal failure	Myoclonic encephalopathy	Dimercaprol (limited evidence)
Cadmium	Contaminated soil in cadmium-rich areas; alloys used in welding, soldering, jewelry, and batteries	Ingestion: hemorrhagic gastroenteritis Inhalation: pneumonitis, acute lung injury	Proteinuria, osteomalacia, lung cancer (questionable)	Ingestion: succimer (limited evidence; not generally indicated) Pneumonitis: chelation not indicated
Chromium	Corrosion inhibitors (eg, heating systems), pigment production	Skin irritation and ulceration, contact dermatitis; GI irritation, renal and pulmonary failure	Mucous membrane irritation, perforation of nasal septum, chronic cough, contact dermatitis, skin ulcers ("chrome holes"), lung cancer	Acetylcysteine (animal studies suggest efficacy as chelator)
Cobalt	"Hard metal dust" (tungsten-cobalt mixture), flexible magnets, drying agents	Contact dermatitis, asthma	Hard metal lung disease (spectrum ranging from alveolitis to fibrosis), cardiomyopathy, thyroid hyperplasia	Acetylcysteine (animal studies suggest efficacy as chelator)
Copper	Leaching from copper pipes and containers; fungicide (copper sulfate); welding (copper oxide)	Ingestion: resembles iron poisoning; blue vomitus (copper salts), hepatotoxicity, hemolysis, methemoglobinemia Inhalation: metal fume fever (self-limited fever, chills, cough, dyspnea)	Hepatotoxicity (Indian childhood cirrhosis)	Dimercaprol for hepatic or hematologic toxicity Succimer in mild poisoning



Silver	Colloidal (metallic) silver used for medicinal purposes as oral solutions, aerosols, and douches; cauterizing and antiseptic agent (silver nitrate); jewelry, wire	Mucosal irritation (silver oxide and nitrate)	Argyria (permanent skin discoloration due to silver deposition and melanocyte stimulation)	Selenium (possible role)
Thallium	Rodenticides (use prohibited in the U.S.); contaminated herbal products; medical radioisotope (miniscule dose); most poisonings related to homicide	Early: nausea, vomiting, abdominal pain, tachycardia Intermediate (>24 h): painful ascending neuropathy, cardiac dysrhythmias, altered mental status Delayed (2 wk): alopecia	Sensorimotor neuropathy, psychosis, dermatitis, hepatotoxicity	Multidose activated charcoal Prussian blue, 125 milligrams/ kilogram PO every 12 h
Zinc	Smelting, electroplating, military smoke bombs, zinc lozenges, welding/galvanizing (zinc oxide)	Ingestion: nausea, vomiting, abdominal pain (resembles iron poisoning) Inhalation: mucosal irritation, metal fume fever (zinc oxide)	Copper deficiency, sideroblastic anemia, neutropenia	Edetate calcium disodium Supportive care for metal fume fever

**TABLE 114-5** Miscellaneous Metal Poisoning: Unique Manifestations and Treatments of Patients Poisoned by Less Common Metals

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 197, “Metals and Metalloids,” by Heather Long and Lewis S. Nelson.

# CHAPTER 115

## Industrial Toxins and Cyanide

Christian A. Tomaszewski

Hazardous chemicals are defined as agents capable of causing adverse health effects or physical danger (combustion or explosion) and are summarized in [Table 115-1](#). Useful resources in managing patients exposed to industrial chemicals include material safety data sheets and local poison control centers. Special at risk groups include pregnant women (the medical focus should be on treating the mother first, with early obstetric consultation) and children.

	Substances	Symptoms	Antidotes
Respiratory toxins	Phosgene Chlorine Vinyl chloride Nitrogen oxides Ammonia	Respiratory distress Pulmonary edema	Supportive care
Metabolic toxins	Cyanide Hydrogen sulfide Carbon monoxide Ricin	Coma Seizures Cardiac arrest	Cyanide kit for cyanides and hydrogen sulfide 100% oxygen or hyperbaric oxygenation for carbon monoxide
Hydrocarbons	Halogenated hydrocarbons Aromatic hydrocarbons	Confusion, lethargy Coma Cardiac dysrhythmias Respiratory distress	Symptomatic treatment Avoid sympathomimetic agents

**TABLE 115-1** Common Signs and Symptoms of Exposure to Hazardous chemicals

## RESPIRATORY TOXINS

### Clinical Features

Inhaled toxins include gases, dusts, fumes, and aerosols and generally cause acute dyspnea, burning of the mucous membranes, cough, and bronchospasm. Common irritating gases include chlorine, ammonia, hydrogen sulfide, and nitrogen dioxide. Some gases, particularly nitrogen dioxide, chlorine, and phosgene, can cause delayed pulmonary edema. Consider systemic toxicity from carbon monoxide and cyanide whenever there is a history of combustion. Some of the more common dangerous inhalations are described in [Table 115-2](#).

Agent	Source	Initial Irritation	Clinical Features	Diagnosis
Ammonia	Fertilizers, plastics, and explosive industry	High	Immediate mucous membrane burning; potential for pulmonary edema	Pungent odor
Chlorine	Paper manufacturing and water treatment	Intermediate	Early upper airway irritation followed by pulmonary edema	Green-yellow gas
Nitrogen dioxide	Blast weapon industry and silos	Low	Triphasic: initial dyspnea, improvement, then delayed pulmonary edema	Reddish-brown gas
Phosgene	Production of plastics, dyes, and pesticides	Low	Pulmonary edema	Odor of newly mown hay

**TABLE 115-2** Characteristics of Selected Toxic Industrial Exposures That Cause Dyspnea

## Diagnosis and Differential

ED evaluation includes chest radiography and laboratory studies (arterial blood gas, carboxyhemoglobin, methemoglobin, and lactate) in selected cases. The role of early bronchoscopy is controversial.

## Emergency Department Care and Disposition

1. Administer 100% oxygen, usually humidified, along with bronchodilators as needed. Have a low threshold for intubation because of the potential for pulmonary edema.
2. Prophylactic steroids and antibiotics are generally not indicated, though steroids may be considered for patients with underlying reactive airway disease and may also reduce the risk of delayed pulmonary edema from nitrogen dioxide inhalation.

## **CYANIDE**

### **Clinical Features**

Cyanide exposure most commonly results from fires that involve synthetic materials, wool, or plastics, but may also be associated with vermicidals, precious metal reclamation, chemical laboratories, and Prunus seeds. Cyanide is highly toxic.

The initial clinical features of cyanide poisoning involve the cardiovascular and central nervous systems, with subsequent lactic acidosis causing dyspnea and ultimately coma, cardiovascular collapse, and death.

The most common presentation is unexplained confusion, hyperventilation, hypotension, and/or bradycardia, especially when accompanied by unexplained metabolic acidosis. [Table 115-3](#) lists the signs and symptoms of acute cyanide toxicity.

	Toxic Level
Cardiovascular	
Tachycardia	Mild
Hypertension	
Bradycardia	
Hypotension	
Cardiovascular collapse	
Asystole	Severe
Central nervous system	
Headache	Mild
Drowsiness	
Seizures	
Coma	Severe
Pulmonary	
Dyspnea	Mild
Tachypnea	
Apnea	Severe

**TABLE 115-3** Signs and Symptoms of Acute Cyanide Toxicity

## Diagnosis and Differential

Because delays in treatment can lead to death, cyanide poisoning is usually diagnosed clinically at the bedside. Blood cyanide levels are not rapidly available.

## Emergency Department Care and Disposition

1. Administer 100% oxygen and secure the airway. Administer crystalloids and vasopressors for hypotension.
2. Consider decontamination with activated charcoal if the airway is stable and the patient presents within an hour of cyanide ingestion.
3. Antidotal therapy for adults is outlined in [Table 115-4](#), while [Table 115-5](#) summarizes the weight-based treatment of children. Administer empiric treatment prior to confirmatory tests in patients presenting with coma, cardiovascular collapse, and severe unexplained metabolic acidosis in the appropriate clinical setting (industrial fires or accidents, smoke inhalation). Patients with mild to moderate symptoms can be closely

observed prior to treatment. Traditionally, sodium nitrite is given first to induce methemoglobin, which helps remove cyanide from cytochrome. This is followed by sodium thiosulfate, which enhances enzymatic formation of less toxic thiocyanate from cyanide. This agent is safer than nitrites, especially for victims of smoke inhalation or when the diagnosis is unclear.

	Route	Treatment
PREHOSPITAL	inhaled	Amyl nitrite vial (crack and inhale over 30 s)
TRADITIONAL	IV	(1) Sodium nitrite 3% solution: 10 mL (300 milligrams) IV given over no less than 5 min (2) Sodium thiosulfate 25% solution: 50 mL (12.5 grams) IV (may repeat sodium thiosulfate once at half-dose 25 mL if symptoms persist)
ALTERNATE	IV	Hydroxocobalamin 5 grams over 15 min (may combine with sodium thiosulfate)

**TABLE 115-4** Treatment of Cyanide Poisoning in Adults

100% oxygen	
Sodium nitrite 3% solution: adjusted according to hemoglobin level, given IV over no less than 5 min*	
Hemoglobin (grams/100 mL)	Sodium Thiosulfate 3% Solution (mL/kg)
7	0.19
8	0.22
9	0.25
10	0.27
11	0.30
12	0.33
13	0.36
14	0.39
Sodium thiosulfate 25% solution: 1.65 mL/kg IV	
Repeat sodium thiosulfate once at half dose (0.825 mL/kg) if symptoms persist	
Monitor methemoglobin and keep level <30%	

\*Avoid nitrites in the presence of severe hypotension if diagnosis is unclear.

**TABLE 115-5** Treatment of Cyanide Poisoning in Children

4. **Hydroxocobalamin** is a newer antidote for cyanide. It is administered as 2 vials, each 2.5 grams reconstituted in 100 mL NS, given over 7.5 min (for a total dose of 5 grams over 15 min). This can be repeated within 15

min as clinically indicated. Although not approved for pediatric use, a dose of 70 milligrams/kilogram has been used. There may be added benefits if sodium thiosulfate is given concurrently, though it must be administered through a separate IV line.

## **HYDROGEN SULFIDE**

Hydrogen sulfide is a colorless gas used in the petrochemical industry and can emanate from sewage or manure. Although it has a distinct “rotten egg” odor, this olfactory warning is lost with extended exposure and high concentrations. Hydrogen sulfide causes cellular asphyxia that leads to lactic acidosis. In high concentrations, rapid loss of consciousness, seizures, and death can occur after only a few breaths. Treat with 100% oxygen, followed by administration of sodium nitrite IV, as with cyanide poisoning. The resultant methemoglobin enhances formation of less toxic sulfmethemoglobin from sulfide.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 198, “Industrial Toxins,” by Chip Gresham and Frank LoVecchio.

# **CHAPTER 116**

## **Herbals and Vitamins**

Stephen L. Thornton

Over-the-counter herbal and vitamin preparations are widely used and considered innocuous by most of the public. Many of these herbal and vitamin products, however, can produce significant toxicity, especially if used in excess.

### **CLINICAL FEATURES**

Common symptoms of vitamin toxicities are listed in [Table 116-1](#).



Vitamin	Symptoms
Vitamin A	Chronic toxicity: blurred vision, appetite loss, abnormal skin pigmentation, hair loss, dry skin, pruritus, long-bone pain, bone fractures, rare cases of pseudotumor cerebri, and hepatic failure
Vitamin D	Subacute toxicity: hypercalcemia, anorexia, nausea, abdominal pain, lethargy, weight loss, polyuria, constipation, confusion, and coma
Vitamin E	Chronic toxicity: nausea, fatigue, headache, weakness, and blurred vision
Vitamin B <sub>1</sub> (thiamine)	No toxicity observed, even with ingestion of large doses over prolonged periods
Vitamin B <sub>2</sub> (riboflavin) Vitamin B <sub>3</sub> (niacin)	No toxicity observed, regardless of amount ingested Acute toxicity: niacin flush, dose > 100 milligrams, redness, burning, and itching of the face, neck, and chest, rarely hypotension Chronic toxicity: doses > 2000 milligrams/d, abnormalities of liver function, impaired glucose tolerance, hyperuricemia, skin dryness, and discoloration
Vitamin B <sub>6</sub> (pyridoxine)	Subacute and chronic toxicity: doses > 5 grams/d or more over several weeks, peripheral neuropathy with unstable gait, numbness of the feet, similar symptoms in the hands and arms, marked loss of position and vibration senses
Vitamin B <sub>12</sub>	No toxicity observed, even with ingestion of large doses
Folate	No toxicity observed, even with ingestion of large doses
Vitamin C (ascorbate)	Chronic toxicity: attacks of gout, nephrolithiasis, intrarenal deposition of oxalate crystals with renal failure, large doses can produce diarrhea and abdominal cramps

**TABLE 116-1** Symptoms of Hypervitaminosis

Many popular herbal preparations have potential for serious toxicity. Nutmeg can cause hallucinations, agitation, gastrointestinal upset, miosis, coma, and hypertension. Ephedra, used for weight loss, contains ephedrine and can produce sympathomimetic toxicity, leading to strokes, seizures, and cardiac ischemia and dysrhythmias. Yohimbine is an  $\alpha_2$ -adrenergic receptor antagonist that may produce hallucinations, weakness, hypertension, and paralysis. Pennyroyal oil can cause hepatotoxicity. Absinthe (wormwood) contains volatile oils that can cause psychosis, intellectual deterioration, ataxia, headache, and vomiting. Black (or blue) cohosh, used to treat menopause, can induce nausea, vomiting, dizziness, and weakness. Juniper, used as a diuretic, can cause renal toxicity, nausea, and vomiting. Lobelia, used for asthma, can produce anticholinergic syndrome. Garlic, ginkgo, and ginseng have antithrombotic activity, which may precipitate bleeding in

patients on warfarin. St John wort, in conjunction with other antidepressants, may precipitate serotonin toxicity.

## **DIAGNOSIS AND DIFFERENTIAL**

Diagnosis is usually made clinically. A history of massive acute ingestion or chronic supratherapeutic use should be sought. Laboratory studies that may be helpful include a basic metabolic panel, hepatic enzymes, coagulation studies, bleeding time, toxicology screen, and urine pregnancy test. An ECG may be indicated with signs of sympathomimetic stimulation.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Basic supportive care and discontinuation of the vitamin or herbal preparation is usually all that is needed if a patient presents with mild toxicity.

1. Consider **activated charcoal** 1 gram/kilogram PO for large vitamin A or vitamin D overdoses.
2. Treat hypercalcemia from vitamin A or D overdose with normal saline, furosemide, and prednisone (to reduce GI absorption).
3. Consider diagnostic and therapeutic lumbar puncture to treat increased intracranial pressure of pseudotumor from hypervitaminosis A.
4. Administer **diphenhydramine** 25 to 50 milligrams IV (1 milligram/kilogram in children) or PO to patients with “niacin flush” symptoms.
5. Consider **N-acetylcysteine** 140 milligrams/kilogram PO or IV for treating severe hepatotoxicity from herbal preparations such as pennyroyal oil.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 199, “Herbals and Vitamins,” by G. Richard Braen and Prashant Joshi.

# CHAPTER 117

## Dyshemoglobinemias

B. Zane Horowitz

Dyshemoglobinemias result from the alteration of the hemoglobin molecule, which prevent it from carrying oxygen. Carboxyhemoglobin is created following exposure to carbon monoxide and is discussed in [Chapter 127](#). [Table 117-1](#) lists common pharmaceuticals capable of causing toxicity.

Oxidant	Comments
Analgesics Phenazopyridine Phenacetin	Commonly reported Rarely used
Antimicrobials Antimalarials Dapsone	Common Hydroxylamine metabolite formation is inhibited by cimetidine
Local anesthetics Benzocaine Lidocaine Prilocaine Dibucaine	Most commonly reported of the local anesthetics Rare Common in topical anesthetics Rare
Nitrates/nitrites Amyl nitrite  Isobutyl nitrite Sodium nitrite Ammonium nitrate Silver nitrate Well water Nitroglycerin	Cyanide antidote kit and used to enhance sexual encounters  Used to enhance sexual encounters Cyanide antidote kit Cold packs Excessive topical use Problem in infants, due to nitrate fertilizer runoff Rare
Sulfonamides Sulfamethoxazole	Uncommon

**TABLE 117-1** Drugs Commonly Implicated in Patients With Methemoglobinemia

## CLINICAL FEATURES

Methemoglobinemia presents with cyanosis. Children up to the age of 4 months lack the key enzyme for normally reducing methemoglobin. These children are susceptible to oxidant stress-induced methemoglobinemia. Three scenarios occur with some frequency: children with acute febrile illness, especially with diarrhea and dehydration; children with exposure to benzocaine in over-the-counter teething gels; and children with exposure to nitrates in agricultural areas with fertilizer runoff into the water aquifer.

In drug-induced methemoglobinemia, patients present with slate-grey to blue discoloration of the skin when levels exceed 15%. Symptoms occur in proportion to declining oxygen delivery. Headache, nausea, and fatigue occur at low levels (20% to 30%). In those with coronary artery disease, dyspnea, angina, and dysrhythmias may result. Levels above 50% can cause loss of consciousness and metabolic acidosis, and above 70% may be lethal.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of methemoglobin should be considered in patients presenting with cyanosis that does not improve with administration of oxygen. During venipuncture blood may appear chocolate brown, a visible effect that is easily identified when the blood is placed on filter paper with a normal patient's blood for comparison. Levels are measured by co-oximetry on an arterial blood gas analyzer, with either an arterial or venous sample. Standard pulse oximetry will almost always generate a reading of 85% that does not change despite administration of 100% oxygen. Newer generation pulse oximeters have been developed that can accurately measure abnormal hemoglobin (both methemoglobin and carboxyhemoglobin) noninvasively.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Methemoglobinemia should be treated initially with close monitoring and high concentrations of inspired oxygen ([Table 117-2](#)). Methemoglobinemia

at levels above 25%, and symptomatic patients with lower levels should be treated with **methylene blue**. The initial dose of methylene blue is 1 to 2 milligrams/kilogram as a 10% solution IV, given over 15 min, which may be repeated. Failure to respond to a second dose is usually due to 1 of 5 causes (in order or likelihood of occurrence):

Assess airway, breathing, and circulation
Place an IV line
Administer oxygen
Attach the patient to a cardiac and pulse oximetry monitor
Obtain an ECG
Decontaminate the patient as needed
Administer methylene blue—if symptomatic or methemoglobin >25%
Consider: cimetidine for patients taking dapsone

**TABLE 117-2** Management of Methemoglobinemia

1. Glucose-6-phosphate dehydrogenase deficiency (G6PD): consider transfusion of packed red blood cells for severely elevated methemoglobin levels in patients with suspected G6PD deficiency.
2. Dapsone: several compounds, most commonly dapsone, generate hydroxylamine that causes oxidation to methemoglobin. Treat dapsone-induced methemoglobinemia with repetitive dosing of methylene blue; consider the addition of IV **cimetidine** to impede the metabolism of dapsone to hydroxylamine.
3. NADPH-methemoglobin reductase deficiency: patients with congenital absence of this enzyme are not chronically cyanotic nor do they have resting methemoglobin levels above normal. However, they lack the ability to convert methylene blue to its active metabolite. As with G6PD deficiency, consider packed red cell or exchange transfusions for severe cases, especially those with hemolysis.
4. Methylene blue induced hemolysis: paradoxically, methylene blue can be a source of oxidant stress. Methylene blue doses, therefore, should not exceed 7 milligrams/kilogram/d.
5. Sulfhemoglobinemia: this rare drug-induced dyshemoglobinemia can occur with sulfur-containing pharmaceuticals and phenacetin. Patients appear cyanotic at sulfhemoglobin levels of 5%, and pulse oximetry may read in the 70% to 80 % range, but are rarely symptomatic. Treat sulfhemoglobinemia with supplement oxygen.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 201, “Dyshemoglobinemias,” by Brenna M. Farmer and Lewis S. Nelson.

## **SECTION 12 Environmental Injuries**

# CHAPTER 118

## Frostbite and Hypothermia

Michael C. Wadman

### NONFREEZING COLD INJURIES

Trench foot is a direct soft tissue injury that results from prolonged exposure to nonfreezing cold and moisture. The foot is initially pale, mottled, pulseless, and anesthetic and does not improve quickly with rewarming. Several hours after rewarming, the foot becomes hyperemic and painful and perfusion returns after 2 to 3 days. Bullae and edema are late findings. Chilblains (pernio) are painful inflammatory lesions typically affecting the ears, hands, and feet caused by chronic exposure to intermittent damp, nonfreezing conditions. Localized edema, erythema, and cyanosis appear up to 12 hours after the exposure and are accompanied by pruritis and burning paresthesias. Tender blue nodules may form after rewarming. Treatment of trench foot and chilblains include elevation, warming, and bandaging of the affected body part. **Nifedipine** 20 milligrams PO 3 times daily, **pentoxifyline** 400 milligrams PO 3 times daily, **or limaprost** 20 micrograms PO 3 times daily, as well as topical corticosteroids, such as **0.025% fluocinolone cream** or a brief burst of oral steroids may be added.

### FROSTBITE

#### Clinical Features

Freezing of the tissue causes frostbite. Patients initially complain of stinging, burning, and numbness. Frostbite injuries are classified by the depth of injury and amount of tissue damage based on appearance after



rewarming. First-degree frostbite (frostnip) is characterized by partial thickness skin freezing, erythema, edema, lack of blistering, and no tissue loss. Second-degree frostbite is characterized by deeper skin freezing and results in the formation of clear bullae. The patient complains of numbness, followed by aching and throbbing. Deep cold injury, third-degree frostbite, involves freezing of the skin and subdermal plexus leading to hemorrhagic bullae and skin necrosis. Fourth-degree frostbite, extends deeper to muscle, tendon, and bone with mottled skin, nonblanching cyanosis, and eventual dry, black, mummified eschar formation. Because it is difficult to initially evaluate the depth of injury, early injuries are better classified as superficial or deep. Laboratory testing or imaging is not needed to diagnosis frostbite.

## **Treatment**

1. Provide rapid rewarming in circulating water at 40°C to 42°C (104.0°F to 107.6°F) until tissue is pliable and erythematous.
2. Debridement of clear blisters and aspiration of hemorrhagic blisters are controversial. Consult with a surgeon for local preference.
3. Apply topical aloe vera every 6 hours.
4. Provide pain management, local wound care and dressing. Splint and elevate affected extremities. Patients may require parenteral opioids initially, followed by oral NSAIDs.
5. Update tetanus immunoprophylaxis.
6. Patients with superficial local frostbite may be discharged home with close follow-up arranged.
7. Patients with deeper injuries require admission for ongoing care.
8. The use of prophylactic bacitracin ointment, prophylactic antibiotics, and silver sulfadiazine is controversial.

## **HYPOTHERMIA**

Hypothermia, a core body temperature of <35°C (95°F), results from heat loss due to conduction, convection, radiation, or evaporation.

## **Clinical Features**

Patients with mild hypothermia (32°C to 35°C [90°F to 95°F]) present with shivering, tachycardia, tachypnea, and hypertension. When core temperatures fall below 32°C (90°F), shivering ceases and heart rate and blood pressure decrease. As temperature falls, patients become confused, lethargic, and then comatose. Pupillary reflexes are lost. Respiratory rate decreases, gag and cough reflexes are diminished, bronchorrhea occurs. Aspiration is common. Impaired renal concentration results in a cold diuresis and hemoconcentration. As temperature decreases, the typical progression is from sinus bradycardia, to atrial fibrillation with slow ventricular response, to ventricular fibrillation, to asystole. At temperatures <30°C the risk for dysrhythmias increases.

## **Diagnosis and Differential**

The diagnosis of hypothermia is based on core temperature and may not be initially obvious, especially in cases where a history of prolonged environmental exposure is missing. Low reading thermometers are required to measure and monitor temperature. Laboratory investigation is directed at determining the underlying cause and complications and includes glucose, CBC, electrolytes, clotting profile, blood gas and EKG. Acid-base disorders are common, but do not follow a predictable pattern. Intravascular thrombosis, embolism, and DIC may occur. Electrocardiographic changes include PR, QRS, and QT prolongation, T-wave inversion, and a slow positive deflection at the end of the QRS (Osborn J wave). In addition to environmental exposure, causes of hypothermia include hypoglycemia, hypothyroidism, hypoadrenalism, hypopituitarism, CNS dysfunction, drug intoxication, sepsis, and dermal disease.

## **Emergency Department Care and Disposition**

1. Place patient in a warm environment. Initiate continuous monitoring of vital signs, pulse oximetry, and core temperature (rectal, bladder, or esophageal thermometer). Indications for intubation are similar to those for normothermic patients. Initiate warmed crystalloid intravenous fluids. Remove wet clothing, dry, and cover patients.
2. Handle patients gently to avoid precipitation of ventricular fibrillation.
3. Attempt to palpate a pulse and detect respirations for 30 to 45 seconds. If none is detected, initiate CPR.
4. Sinus bradycardia, atrial fibrillation, or flutter usually requires no therapy and will resolve with rewarming. Ventricular fibrillation is typically refractory to therapy until the patient is rewarmed, but a single defibrillation is recommended.
5. Rewarming techniques include passive rewarming, active external rewarming, and active core rewarming ([Table 118-1](#)). Choice of technique depends primarily on cardiovascular status. Temperature is a secondary consideration. Patients with a stable cardiovascular status (including sinus bradycardia and atrial fibrillation) and temperature above 30°C may be passively rewarmed. All patients with cardiovascular instability require rapid core rewarming; extracorporeal circuit rewarming is the technique of choice for these patients. Invasive rapid core rewarming in patients without cardiac instability is controversial.

Passive rewarming
Removal from cold environment
Insulation
Active external rewarming
Warm water immersion
Heating blankets set at 40°C (104°F)
Radiant heat
Forced air
Active core rewarming at 40°C (104°F)
Inhalation rewarming
Heated IV fluids
GI tract lavage
Bladder lavage
Peritoneal lavage
Pleural lavage
Extracorporeal rewarming
Mediastinal lavage by thoracotomy

**TABLE 118-1** Rewarming Techniques

6. Continue resuscitative efforts until core temperature reaches 30°C to 32°C.
7. Address and treat underlying causes (eg, **dextrose** 50 mL IV for hypoglycemia, **thiamine** 100 milligrams IV/IM in thiamine deficient alcoholism; treat suspected hypothyroidism and hypoadrenalism with hormone replacement).
8. Admit all patients with symptomatic hypothermia. Healthy patients with mild environmental hypothermia that resolves quickly may be discharged home if social circumstances allow.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 202, “Frostbite and Other Localized Cold Injuries,” by Tiina M. Ikaheimo, Juhani Junila, Jorma Hirvonen, and Juhani Hassi; and Chapter 203, “Hypothermia,” by Howard A. Bessen and Bryan Ngo.

# CHAPTER 119

## Heat Emergencies

T. Paul Tran

Heat-related illnesses range from minor heat disorders, such as prickly heat and heat cramps, to life-threatening *heat stroke*. In heat stroke, thermal regulation breaks down, resulting in hyperthermia (temperature > 40°C) and end-organ damage.

### MINOR HEAT ILLNESSES

*Heat edema* is a self-limited, mild swelling of dependent extremities (hands and feet) that occurs in the first few days of exposure to a new hot environment. It is due to cutaneous vasodilation and pooling of interstitial fluid in dependent extremities. Treatment consists of elevation of the extremities or compressive stockings. Administration of diuretics may exacerbate volume depletion and should be avoided.

*Heat rash* (aka: *prickly heat*, *lichen tropicus*, *miliaria rubra*) is a vesiculopapular eruption that is found most commonly over clothed areas of the body. It results from inflammation and obstruction of sweat ducts. Antihistamines, low potency topical corticosteroids, or calamine lotion may provide symptomatic relief. Advise patients to wear light, loose fitting clothing.

*Heat syncope* results from volume depletion, peripheral vasodilation, and decreased vasomotor tone. It occurs most commonly in the elderly and poorly acclimatized individuals. Postural vital signs may or may not be demonstrable on presentation to the emergency department. Potentially serious causes of syncope (eg, cardiovascular, neurologic, infectious, endocrine, and electrolyte abnormalities) should be investigated, especially in the elderly. Treatment consists of rest and oral or IV rehydration.

*Heat cramps* are characterized by painful muscle spasms, especially in the calves, thighs, and shoulders during athletic events. They usually occur

when individuals replace evaporative losses with free water but not with salt. Core body temperature may be normal or elevated. Treatment consists of rest and administration of oral electrolyte solution or IV normal saline.

*Heat tetany* is due to the effects of respiratory alkalosis that results when an individual hyperventilates in response to an intense heat stress. Patients may complain of paresthesia of the extremities, circumoral paresthesia, and carpopedal spasm. Muscle cramps are minimal or nonexistent. Treatment consists of removal from the heat source and decreasing the respiratory rate.

## **HEAT STROKE**

### **Clinical Features**

Exertional heat stroke usually occurs after strenuous physical activity in a hot environment, whereas nonexertional heat stroke more commonly affects chronically ill or debilitated patients and persons at the extremes of age, especially during a prolonged heat wave. The cardinal features are hyperthermia (core temperature  $> 40^{\circ}\text{C}$  [ $104^{\circ}\text{F}$ ]) and altered mental status. Anhidrosis or profuse sweating may be seen. Prominent neurologic abnormalities include confusion, agitation, bizarre behavior, ataxia, seizures, obtundation, and coma. Other findings include hyperventilation, nausea, vomiting, diarrhea, muscle cramps, and oliguria.

### **Diagnosis and Differential**

Heat stroke should be considered in the clinical context of environmental heat stress, hyperthermia, and altered mental status. The differential diagnosis includes infection (eg, sepsis, meningitis, encephalitis, malaria, typhoid, tetanus), endocrine disorders (eg, diabetic ketoacidosis, thyroid storm), neurologic disorders (eg, cerebrovascular accident, status epilepticus), and toxicologic causes (eg, anticholinergics, sympathomimetics, salicylates, serotonin syndrome, malignant hyperthermia, neuroleptic malignant syndrome, alcohol or benzodiazepine withdrawal). About 20% of heat stroke patients are hypotensive. Initial

diagnostic studies are directed at detecting end-organ damage and excluding other disease processes. Respiratory alkalosis and lactic acidosis are seen in exertional heat stroke; respiratory alkalosis in nonexertional heatstroke. Early laboratory abnormalities associated with exertional heat stroke include hypoglycemia, hypophosphatemia and hypokalemia, elevated liver enzymes due to hepatocellular damage, hypercalcemia and an elevated hematocrit due to hemoconcentration, and elevated creatine phosphokinase and myoglobin from rhabdomyolysis. Laboratory abnormalities of DIC, renal failure develop with time. Obtain an ECG and CXR. Neuroimaging studies and other evaluations (eg, septic workup, toxicology screens) can be individualized as clinically indicated.

## **Emergency Department Care and Disposition**

1. Emergent priorities are airway, breathing, circulation, rapid initiation of cooling, and supportive care. Intubate patients with significantly altered mental status, diminished gag reflex, or hypoxia. Initiate continuous monitoring of vital signs, pulse oximetry, and core temperature (rectal, bladder, or esophageal thermometer). Provide high flow oxygen and begin IV crystalloids to maintain mean arterial pressure above **80 to 90 mm Hg**. Avoid volume overload. Vasopressors may be required.
2. **Evaporative cooling** is the most efficient and practical means of cooling hyperthermic patients in the emergency department. Place fans near the completely disrobed patient and spray the patient with tepid water. The goal is to decrease core temperature to below 39°C while avoiding hypothermia.
  - a. Avoid spraying with ice water because this may cause shivering, which induces thermogenesis.
  - b. Excessive shivering can be treated with short-acting benzodiazepines.
  - c. Other cooling methods are listed in [Table 119-1](#).

Cooling Method	Advantages	Disadvantages	Recommendations
Exposive methods	Provides effective cooling Readily available Practical Well tolerated	Can cause shivering Less effective in humid environments Makes it difficult to maintain electrode positions	Strongly recommended
Ice water immersion	Provides effective cooling	Can cause shivering Poorly tolerated Not compatible with resuscitation settings	Recommended
Ice packs on neck, axillae, and groin	Practical Can be added to other cooling methods	Has limited cooling efficacy Poorly tolerated	Can be used as adjunct cooling method
Cardiopulmonary bypass	Provides fast and effective cooling	Invasive Not readily available Setup is labor intensive	Recommended in severe or resistant cases when available
Cooling blankets	Easy to apply	Has limited cooling efficacy Impedes use of other cooling methods	Not recommended when other methods available
Cold water gastric, urinary bladder, rectal, or peritoneal lavage	-	Invasive Labor intensive May lead to water intoxication Human experience is limited	Not recommended
Cold water IV infusion	-	Carries unjustified complication rate	Not recommended



**TABLE 119-1** Summary of Cooling Techniques

3. Treat seizures with **benzodiazepines**, such as **lorazepam** 1 to 2 milligrams IV **or diazepam** 5 milligrams IV.
4. Treat rhabdomyolysis with IV hydration. To date, no prospective control studies have shown improved outcomes from alkalinization of the urine or forced diuresis with mannitol or loop diuretics.
5. Monitor serum electrolytes every hour initially. Treat hyperkalemia with standard regimens.
6. Most heat stroke patients require admission to the ICU.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 204, “Heat Emergencies,” by Thomas A. Waters and Majid A. Al-Salamah.

# CHAPTER 120

## Bites and Stings

Burton Bentley II

### WASPS, BEES, AND STINGING ANTS (*HYMENOPTERA*)

Wasps, bees, and stinging ants are members of the order Hymenoptera. Local and generalized reactions may occur in response to an encounter. Africanized bees often attack in massive numbers with a venom load that may result in lethal toxicity. Fire ant venom may cross-react in individuals sensitized to other Hymenoptera stings.

#### Clinical Features

*Local reactions* consist of pain, erythema, edema, and pruritus at the sting site. Severe local reactions increase the likelihood of serious systemic reactions if the patient is reexposed. The local reaction to a fire ant sting consists of a sterile pustule that evolves over 6 to 24 hours, sometimes resulting in necrosis and scarring. *Toxic reactions* are the nonantigenic result of a direct venom effect. They have many of the same features of true systemic (allergic) reactions, but there is a greater frequency of gastrointestinal disturbance while bronchospasm and urticaria are infrequent. Symptoms typically subside within 48 hours, though severe cases last longer and lead to rhabdomyolysis and hepatorenal failure. *Systemic or anaphylactic reactions* are true allergic reactions that range from mild to fatal. In general, the shorter the interval between the sting and the onset of symptoms; the more severe the reaction. Nearly all episodes of anaphylaxis occur within 6 hours; the majority occur within 15 min. Initial symptoms usually consist of itchy eyes, urticaria, and cough. As the

reaction progresses, patients may experience respiratory failure and cardiovascular collapse. *Delayed reactions* may occur 5 to 14 days after a sting, resemble serum sickness. Symptoms include fever, malaise, headache, urticaria, lymphadenopathy, and polyarthritis.

## **Emergency Department Care and Disposition**

1. Remove the bee stinger and clean the wound with soap and water. Intermittent application of ice packs and elevation may reduce swelling.
2. For local reactions, oral antihistamines and analgesics provide symptomatic relief. Localized erythema and swelling may be difficult to distinguish from cellulitis but infection is uncommon.
3. Treat multisystem reactions (anaphylaxis) in the standard fashion. (See [Chapter 6](#) “Anaphylaxis, Acute Allergic Reactions, and Angioedema”)
  - a. First-line therapy for anaphylaxis is epinephrine. Administer 0.3 to 0.5 milligram (0.3 to 0.5 mL of 1:1000; pediatric dose, 0.01 milligram/kilogram to a maximum of 0.5 milligram) IM in the anterolateral thigh. Repeat every 5 min as needed. Patients refractory to IM dosing or in significant shock should receive intravenous epinephrine. A bolus of 100 micrograms of 1:100 000 dilution (0.1 mL of 1:1000 in 10 mL normal saline) can be given over 5 to 10 min followed by an infusion, with close observation for chest pain or arrhythmias.
  - b. Hypotensive patients require aggressive fluid resuscitation with normal saline 1 to 2 L (pediatric dose, 10 to 20 mL/kg).
  - c. After epinephrine, administer parenteral H<sub>1</sub> and H<sub>2</sub> receptor antagonists (eg, diphenhydramine 50 milligrams PO/IV/IM [pediatric dose, 1 milligram/kilogram] and ranitidine 50 milligrams IV [pediatric dose, 0.5 milligram/kilogram]).
  - d. Administer steroids to control persistent or delayed reactions, methylprednisolone 125 milligrams IV (pediatric dose, 2 milligrams/kilogram) or prednisone 60 milligrams PO (pediatric dose, 2 milligrams/kilogram).
  - e. Bronchospasm can be treated with nebulized  $\beta$ -agonists such as albuterol 2.5 milligrams.

4. Patients who respond well to conservative measures may be discharged after several hours of observation. Refer all patients with Hymenoptera reactions to an allergist for further evaluation, prescribe a premeasured epinephrine injector (EpiPen<sup>®</sup>), and advise them to carry allergy alert identification. Instruct patients to use epinephrine at the first sign of a systemic reaction.
5. Admit patients with prolonged severe reactions, > 100 stings, those with substantial comorbidities, and those at extremes of age.

## **BROWN RECLUSE SPIDER (*LOXOSCELES RECLUSA*)**

### **Clinical Features**

The initial *L. reclusa* bite is painless. It evolves into a firm erythematous lesion that heals over several days to weeks. Occasionally, a severe reaction with immediate pain, hemorrhagic blister formation, and local blanching may occur. These lesions often become necrotic over the next 3 to 4 days and form significant eschars. *Loxoscelism* is a common systemic reaction following the bite of some South American *Loxosceles* species; it rarely occurs from the bite of *L. reclusa*. Symptoms are more common in children and typically occur 1 to 3 days after envenomation. Signs and symptoms may include fever, chills, vomiting, arthralgias, myalgias, petechiae, and hemolysis; severe cases progress to seizure, renal failure, disseminated intravascular coagulation, and death.

### **Diagnosis and Differential**

*Loxosceles* species are distinguished by three sets of paired eyes; most other spiders have eight eyes in two rows. A pigmented, violin-shaped pattern on the cephalothorax of the brown recluse is characteristic but unreliable. The diagnosis of *L. reclusa* envenomation is commonly clinical since the bite is rarely witnessed. Assays to confirm *L. reclusa* poisoning are not clinically

available. Patients with significant envenomation may exhibit hemolysis, coagulopathy or abnormal renal function.

## **Emergency Department Care and Disposition**

1. Treatment of the brown recluse spider bite includes supportive measures, such as pain medication, tetanus prophylaxis, and antibiotics if infection is present. In the United States, antivenom is not commercially available and is usually not needed.
2. Most wounds heal without intervention. The role of dapsone (50 to 200 milligrams/day PO divided twice daily for 2 weeks) in preventing necrosis is controversial due to lack of supporting research and significant adverse effects. Arrange serial wound evaluations for outpatients.
3. Patients with systemic reactions and hemolysis should be hospitalized.
4. Surgery is reserved for lesions larger than 2 cm and is deferred for 2 to 3 weeks after the bite.

## **HOBO SPIDER (*TEGENARIA AGRESTIS*)**

The hobo spider, also known as the Northwestern brown spider, causes clinical signs and symptoms that are similar to those of the brown recluse spider bite. The skin site is initially painless before developing induration, erythema, blistering, and necrosis. Patients may experience headache, vomiting, and fatigue. There is no specific diagnostic test or therapeutic intervention for hobo spider bites. Surgical repair for severe ulcerative lesions is delayed until the necrotizing process is complete.

## **BLACK WIDOW SPIDER (*LATRODECTUS MACTANS*)**

### **Clinical Features**

Black widow spider bites induce an immediate pinprick sensation that quickly spreads to the entire extremity. Erythema at the site appears within 1 hour (often “target” shaped lesion) along with diffuse muscle cramps in the large muscle groups, especially involving the trunk, back and abdomen. Severe pain may wax and wane for several days. Other signs and symptoms include hypertension, tachycardia, headache, nausea, vomiting, and diaphoresis. Serious acute complications include hypertension, respiratory failure, shock, and coma.

## **Emergency Department Care and Disposition**

1. Initial therapy includes local wound treatment and supportive care. Liberal dosing of analgesics and benzodiazepines will relieve pain and cramping.
2. *Lacrodectus* antivenom, derived from horse serum, is rapidly effective for severe envenomation even when the presentation is delayed. Anaphylaxis rarely has been reported with this therapy. The package insert provides dosing instructions.
3. Patients receiving antivenom may be discharged after a short observation period if symptoms of envenomation resolve.

## **TARANTULAS**

When threatened, tarantulas may flick barbed hairs into their victim. Although North American tarantula hairs rarely penetrate human skin, they can embed deeply into the conjunctiva and cornea and cause an inflammatory response. Any patient complaining of ocular symptoms after exposure to a tarantula should undergo a thorough slit lamp examination to search for imbedded hairs. Treatment includes topical steroids and consultation with an ophthalmologist for surgical removal of the hairs.

Tarantula bites may also occur. They are painful, cause local erythema and edema. Provide local wound care and analgesia.

# **SCORPION (*SCORPIONIDA*)**

## **Clinical Features**

Although highly toxic species are found in the Caribbean, Asia, and Africa, the only North American scorpion that produces systemic toxicity is the bark scorpion (*Centruroides exilicauda*). Venom from *C exilicauda* causes immediate pain and paresthesia. A positive “tap test” (ie, exquisite local tenderness when the area is lightly tapped) may be seen. Systemic effects are infrequent and occur mainly in children. Somatic and autonomic symptoms include tachycardia, nausea, vomiting, excessive secretions, roving eye movements, opisthotonos, fasciculations. Cranial nerve dysfunction may affect vision and swallowing. Symptoms may last 24 to 48 hours without antivenom therapy.

## **Emergency Department Care and Disposition**

1. Treatment is supportive: local wound care, analgesics and benzodiazepines. Muscle spasm and fasciculations respond promptly to benzodiazepines. Patients without systemic symptoms may be observed briefly and discharged with analgesics.
2. Hypersalivation and respiratory distress due to *C exilicauda* may respond to atropine, though atropine is contraindicated for foreign scorpion stings due to exacerbation of adrenergic effects.
3. Severe systemic reactions are treated with antivenom. Scorpion-specific F (ab')<sub>2</sub> equine antivenom is available in the United States, especially in Arizona, but is not yet approved by the FDA. Protocols for use must be followed.

# **SCABIES (*SARCOPTES SCABIEI*)**

## **Clinical Features**

Scabies bites are concentrated in the web spaces between fingers and toes. Other common areas include the axilla and genital area, children's faces and scalps, and the female nipple. Transmission is typically by direct contact. The distinctive feature of scabies infestation is intense pruritus with "burrows." The female mite is easily scraped out with a blade edge. Associated vesicles, papules, crusts, and eczematization may obscure the diagnosis.

### **Emergency Department Care and Disposition**

Advise patients to apply permethrin cream from the neck down; infants may require additional application to the scalp, temple, and forehead. The patient should bathe before application, apply the medication, and then bathe again in 12 hours. Reapplication is necessary only if mites are found 2 weeks after treatment, although the pruritus may last for several weeks after successful therapy. Ivermectin, 200 micrograms/kilogram PO, followed by a second dose in 10 days, is an alternative treatment.

## **TICKS**

The spectrum of tick borne illness includes Lyme disease, Rocky Mountain spotted fever, ehrlichiosis, babesiosis, Colorado tick fever, tularemia, and tickborne encephalitis. Certain species of ticks have a neurotoxin capable of inducing tick paralysis, a symmetric ascending flaccid paralysis nearly identical to Guillain-Barré syndrome. Indeed, a diagnosis of Guillain-Barré should not be considered until a thorough search rules out the presence of an engorged tick. The recommended method of tick removal involves grasping the tick with forceps near the point of attachment and pulling with steady, gentle traction. Since disease transmission is time dependent, prompt tick removal is essential.

## **CHIGGERS (*TROMBICULIDAE*)**



## **Clinical Features**

Chiggers are tiny mite larvae that cause intense pruritus when they feed on host epidermal cells. They tend to attach to skin in areas of tight-fitting clothing such as near waistbands. Children who have been sitting on lawns are prone to chigger lesions in the genital area. Itchiness begins within a few hours, followed by a papule that enlarges to a nodule (“chigger bite”) over the next 1 to 2 days. Single bites may cause soft tissue edema, whereas infestation has been associated with fever and erythema multiforme. The diagnosis of chigger bites is based on typical skin lesions and intense pruritus in the context of known outdoor exposure.

## **Emergency Department Care and Disposition**

Treatment is symptomatic with oral or topical antihistamines, although oral steroids may be required in more severe cases. Annihilation of the mites requires topical application of permethrin or other topical scabicides. The package insert provides techniques for proper use.

## **FLEAS (*SIPHONAPTERA*)**

Flea bites are frequently found in zigzag lines, especially on the legs and waist. They are intensely pruritic lesions with hemorrhagic puncta, surrounding erythema, and urticaria. Discomfort is relieved with calamine lotion, cool soaks, and oral or topical antihistamines. Severe irritation may require topical steroid creams. Patients who develop impetigo and other local infections from should be treated with topical or oral antibiotics.

## **LICE (*ANOPLURA*)**

*Body lice* concentrate on the waist, shoulders, axillae, and neck. Their bites produce red spots that progress to papules and wheals. They are so intensely pruritic that linear scratch marks are suggestive of infestation. The white ova of *head lice* are adherent to the hair shaft and therefore can be distinguished from dandruff. *Pubic lice* are spread by sexual contact. They cause intense pruritus, and their small white eggs (nits) are visible on hair shafts. As with scabies, permethrin is the primary treatment for body lice infestation. Treatment of any hair-borne infestation requires application of pyrethrin with piperonyl butoxide after hair washing, with reapplication in 10 days. Wet combing hair with a fine-tooth comb will remove dead lice and nits. Clothing, bedding, and personal articles should be washed in hot (> 52°C [125.6°F]) water to prevent reinfestation.

## **KISSING BUGS AND BED BUGS (HEMIPTERA)**

Kissing bugs (also known as reduviid or conenose beetles) and bed bugs feed on blood of a sleeping victim. The initial bite is painless. Bedbug bites are often linear. Bites are often multiple and result in wheals or hemorrhagic papules and bullae. Dark lines of bedbug excrement on bed linens may be seen. Treatment consists of local wound care, topical steroids, and oral antihistamines. Allergic reactions may occur in sensitized individuals.

## **SNAKE BITES**

Venomous snake bites in North America are typically caused by pit vipers (Crotaline-rattlesnakes, copperhead, water moccasin and massasauga) or coral snakes (Elapidae).

## **PIT VIPER (CROTALINAE) BITES**

Crotaline snakes, commonly known as pit vipers, are identified by their 2 retractable fangs and by heat-sensitive depressions (“pits”) located bilaterally between each eye and nostril. Only 25% of bites result in envenomation.

## **Clinical Features**

The effects of crotaline envenomation depend on the size and species of snake, the age and size of the victim, the time elapsed since the bite, and the characteristics of the bite itself. The hallmark of pit viper envenomation is the presence of 1 or more fang marks combined with pain, erythema, ecchymosis, and progressive edema extending from the site. In general, envenomated patients will have swelling within 30 min, although swelling may be delayed up to 12 hours. Systemic symptoms include nausea and vomiting, weakness, perioral paresthesias, lethargy and weakness. More severe systemic effects include tachycardia, tachypnea, respiratory distress, and altered sensorium. A coagulopathy with elevated INR and prothrombin time, hypofibrinogenemia, and thrombocytopenia may develop.

## **Diagnosis and Differential**

The diagnosis of crotaline envenomation is based on the presence of the aforementioned local injuries, systemic symptoms or hematologic abnormalities. The absence of any of these findings after 8 to 12 hours indicates a dry bite.

*Minimal* envenomation is defined as local swelling, no systemic signs, and no laboratory abnormalities. *Severe* envenomation causes extensive swelling, potentially life-threatening systemic signs and markedly abnormal coagulation parameters that may result in hemorrhage. Initially mild envenomation syndromes may progress to severe syndromes over several hours. Pertinent laboratory tests include a complete blood count, coagulation tests, urinalysis, and blood typing.

## **Emergency Department Care and Disposition**

1. Consultation with a poison control center is recommended for all but the simplest cases. A properly placed constriction band (a band wrapped circumferentially proximal to the bite with only enough tension to gently impede venous flow) may delay venom absorption.
2. Cardiac monitoring and IV access should be established before removing constricting bands. The patient should be aggressively resuscitated according to ACLS protocols.
3. Provide local wound care and tetanus immunization. Measure limb circumference at several sites above and below the wound and check every 30 min while marking the border of advancing edema.
4. Treat patients with progressive local swelling, systemic effects, or coagulopathy immediately with antivenom therapy, **Polyvalent Crotalidae Immune Fab (FabAV)**, a sheep-derived antivenin. Administer an “initial control” dose of **FabAV** 4 to 6 vials IV; there is no need for prior skin testing. “Initial control” is defined as cessation of progression of all components of envenomation: local effects, systemic effects, and coagulopathy. The initial dose of FabAV, as well as any subsequent dose, is diluted in 250 mL normal saline and infused IV over 1 hour. The goal of therapy is to neutralize existing venom; the dose of FabAV is the same for children and adults although the amount of diluent may need to be decreased in small children. If an allergic reaction occurs, stop the infusion and treat with epinephrine and antihistamines, as indicated.
5. If the “initial control” is not achieved with the first infusion, give a repeat dose of 4 to 6 vials.
6. Monitor blood count and coagulation studies every 4 hours or after each course of antivenom, whichever is more frequent. Monitor renal function.
7. The endpoint of antivenom therapy is the arrest of progressive symptoms and coagulopathy. Administer an additional 2-vial dose at 6, 12, and 18 hours after “initial control” is achieved. The anti-venom package insert will guide in administration. The administration of antivenom must continue until complete control of the envenomation is achieved.
8. *Compartment syndrome* may occur secondary to envenomation. Treatment includes limb elevation, **mannitol** 1 to 2 grams/kilogram IV

over 30 min, and additional **FabAV**, 4 to 6 vials IV over 60 min. If compartment pressures stay high, consider fasciotomy.

9. Active bleeding due to severe coagulopathy may require blood component therapy, although FabAV remains the mainstay of uncomplicated coagulopathy.
10. Observe all patients with a pit viper bite for at least 8 hours. Admit patients with severe bites and those receiving antivenin to the intensive care unit. Patients with no evidence of envenomation after 8 hours may be discharged. All patients who receive FabAV should be counseled regarding the 5% risk of serum sickness. The standard treatment for serum sickness is oral **prednisone**, 1 milligram/kilogram/d tapering over 1 to 2 weeks.

## **CORAL SNAKE BITE**

### **Clinical Features**

Venomous coral snakes in the United States are brightly colored with adjacent red and yellow bands. In the United States, only bites from the eastern coral snake (*Micrurus fulvius fulvius*) require significant treatment as its venom is a potent neurotoxin that causes tremor, salivation, respiratory paralysis, seizures, and bulbar palsies (eg, dysarthria, diplopia, and dysphagia). Bites of the Sonoran (Arizona) coral snake are mild and only need local care.

### **Emergency Department Care and Disposition**

1. Consultation with a poison control center is recommended.
2. The toxic effects of toxic coral snake venom may be preventable, but they are not easily reversed. All patients who have been bitten should receive 3 to 5 vials of antivenom (*M fulvius*). Additional antivenom doses are required if symptoms appear. Access to coral snake antivenom is extremely limited, as it is no longer manufactured in the United States.

3. Follow pulmonary function parameters and neurological exams (eg, inspiratory pressure and vital capacity) and admit to the hospital for 24 to 48 hours of observation.

## **GILA MONSTER BITE**

Gila monster bites result in pain and swelling. Systemic toxicity is rare but may consist of diaphoresis, paresthesia, weakness, and hypertension. The bite may be tenacious, and the reptile should be removed as soon as possible. If the reptile is still attached, it may loosen its bite when placed on a solid surface where it is not suspended in midair. Once removed, perform standard wound care including a search for implanted teeth. No further treatment is required.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 205, “Bites and Stings,” by Aaron B. Schneir and Richard F. Clark; and Chapter 206, “Reptile Bites,” by Richard C. Dart and Frank F. S. Daly.

## CHAPTER 121

# Trauma and Envenomation from Marine Fauna

Christian A. Tomaszewski

The population growth along coastal areas has made exposure to hazardous marine fauna increasingly common. The popularity of home aquariums generates additional exposures inland. Marine fauna can inflict injury through direct traumatic bite or envenomation, usually via a stinging apparatus.

## CLINICAL FEATURES

Marine trauma includes bites from sharks, barracudas, moray eels, seals, crocodiles, needlefish, wahoos, piranhas, and trigger fish. Shark bites may also cause substantial tissue loss with hemorrhagic shock and delayed infection. Minor trauma is usually due to cuts and scrapes from coral that can cause local stinging pain, erythema, urticaria, and pruritus. Marine wounds can be infected with routine skin flora, such as *Staphylococcus* and *Streptococcus*, along with bacteria unique to the marine environment. The most serious halophilic organism is the gram-negative bacillus *Vibrio*, which can cause rapid infections marked by pain, swelling, hemorrhagic bullae, vasculitis, and even necrotizing fascitis and sepsis. Immunosuppressed patients, particularly those with liver disease, are susceptible to sepsis and death (up to 60%) from *Vibrio vulnificus*. Another bacterium, *Erysipelothrix rhusiopathiae*, implicated in fish handler's disease, can cause painful, marginating plaques after cutaneous puncture wounds. The unique marine bacterium *Mycobacterium marinum*, an acid-fast bacillus, can cause a chronic cutaneous granuloma 3 to 4 weeks after exposure.

Numerous invertebrate and vertebrate marine species are venomous. The invertebrates belong to 5 phyla: Cnidaria, Porifera, Echinodermata, Annelida, and Mollusca.

The 4 classes of Cnidaria all share stinging cells, known as nematocysts, which deliver venom subcutaneously when stimulated. The most common effect is pain, swelling, pruritus, urticaria, and even blistering and necrosis in severe cases. The Hydrozoans include hydroids, *Millepora* (fire corals), and *Physalia* (Portuguese man-of-war). The latter causes a linear erythematous eruption and rarely can cause respiratory arrest, possibly from anaphylaxis. In addition to local tissue injury, the Scyphozoans (true jellyfish) include Atlantic Ocean larval forms that can cause a persistent dermatitis under bathing suits lasting days after exposure (Seabather's eruption). The Cubozoans (box jellyfish), in particular *Chironex fleckeri* in Australia and *Chiropsalmus* in the Gulf of Mexico, can cause death after severe stings. A Hawaiian species, *Carybdea*, has been implicated in painful stings but no deaths. Another Australian box jellyfish, *Carukia barnesi*, can cause Irukandji syndrome, characterized by diffuse pain, hypertension, tachycardia, diaphoresis, and even pulmonary edema. The most innocuous cnidaria are the Anthozoans (anemones) that occasionally cause a mild local reaction.

Porifera (the sponges) can produce a stinging, pruritic dermatitis. Spicules of silica or calcium carbonate can become embedded in the skin along with toxic secretions from the sponge. Echinodermata include sea urchins and sea stars. Sea urchin spines produce immediate pain with trauma; some contain venom that leads to erythema and swelling. Retained spines can lead to infection and granuloma formation. The crown-of-thorns sea star, *Acanthaster planci*, has sharp rigid spines that cause burning pain and local inflammation. Annelida include bristle and fire worms, which embed bristles in the skin, causing pain and erythema. Mollusca include gastropods and octopuses. Both the Indo-Pacific cone shell, *Conus*, and the blue-ringed octopus, *Hapalochalena*, can deliver paralytic venom that can quickly lead to respiratory paralysis.

Vertebrate envenomations are primarily due to stingrays (order Rajiformes) and spined venomous fish (scorpion fish, lion fish, catfish, and weeverfish). The stingray whip tail has venomous spines, which puncture or lacerate causing an intense painful local reaction. The spines of venomous fish have glands that force venom into the wound after puncture and cause local pain, erythema, and edema. Retention of a spine can lead to infection.



# EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Copiously irrigate lacerations, punctures and bite wounds; explore for foreign matter and débride devitalized tissue. Soft tissue radiographs or ultrasound may help locate foreign bodies, which usually require removal especially if intraarticular. Leave lacerations open for delayed primary closure. Update tetanus, if needed.
2. Prophylactic antibiotic therapy is not indicated for routine minor wounds in healthy patients but may be considered in selected patients ([Table 121-1](#)). Antibiotic therapy for infected wounds is first directed toward likely pathogens and later by culture and sensitivity results. Cover *Staphylococcus* and *Streptococcus* species with a first generation cephalosporin, such as **cephalexin** 500 milligrams 4 times daily or **cefazolin** 1 to 2 grams every 8 hours, or **clindamycin** 300 milligrams PO/600 milligrams IV 4 times daily, or **doxycycline** 100 milligrams PO/IV twice daily. Addition of a third-generation cephalosporin, such as **ceftriaxone** 1 gram IV daily or **cefotaxime** 2 grams IV every 8 hours, or a fluoroquinolone, such as **levofloxacin** 500 milligrams PO/IV daily will cover ocean related infections from *Vibrio*. A **fluoroquinolone** or **third-generation cephalosporin**, or **trimethoprim-sulfamethoxazole double strength, 1 tablet PO twice each day**, or **imipenem, 500 milligrams IV every 6 hours**, will cover fresh water infections from *Aeromonas*. Granulomas from *Mycobacterium marinum* require several months of treatment with **clarithromycin** or **rifampin plus ethambutol**.

No Antibiotics Indicated	Prophylactic/Outpatient Antibiotics	Hospital Admission for IV Antibiotics
Healthy patient	Late wound care	Predisposing medical conditions
Prompt wound care	Large lacerations or injuries	Long delays before definitive wound care
No foreign body	Early or local inflammation	Deep wounds, significant trauma
No bone or joint involvement		Wounds with retained foreign bodies
Small or superficial injuries		Progressive inflammatory change Penetration of periosteum, joint space, or body cavity Major injuries associated with envenomation Systemic illness

**TABLE 121-1** Recommendations for Antibiotic Treatment of Marine-Associated Wounds

3. See [Table 121-2](#) for early treatment of envenomations.

<b>Marine Organism</b>	<b>Detoxification</b>	<b>Further Treatment</b>
Catfish, lionfish, scorpionfish, stingray	Submerge injury in hot water [45°C (113°F)] for up to 90 min.	Irrigate with normal saline (0.9%). Explore and debride wound. Administer antibiotics and analgesics. Update tetanus immunization if needed. Elevate extremity. Observe for development of systemic symptoms.
Stonefish	Submerge injury in hot water [45°C (113°F)] for up to 90 min.	Irrigate with normal saline (0.9%). Explore and debride wound. Administer antibiotics and analgesics. Update tetanus immunization if needed. Elevate extremity. Administer stonefish antivenin if severe systemic reaction occurs.
Sea snake	—	Use pressure immobilization. Administer antivenom if severe systemic reaction occurs. Provide supportive care.
Fire coral, hydroids, anemones	Blot area. Irrigate with saline. Apply 5% acetic acid (vinegar) topically.	Apply topical antihistamines or corticosteroid cream for itching.
Portuguese man-of-war, blue bottles	Blot area. Submerge injury in hot water [45°C (113°F)] for 20 to 30 min. Remove tentacles.	Apply topical antihistamines or corticosteroid cream for itching. Observe for development of systemic symptoms. Provide supportive care.

Box jellyfish	Blot area. Irrigate with saline. Apply 5% acetic acid (vinegar) topically. Remove tentacles.	Apply topical antihistamines or corticosteroid cream for itching. Observe for development of systemic symptoms. Provide supportive care. Administer <i>Chironex</i> antivenin.
Australian blue-ringed octopus	–	Use pressure immobilization. Provide supportive care.
Cone snail	–	Use pressure immobilization. Provide supportive care.
Sea urchin	Submerge injury in hot water [45°C (113°F)] for up to 90 min. Remove visible spines or pedicellariae.	Explore wound and remove any spines.
Sponge	Irrigate with water. Apply cold compresses.	Administer oral analgesics. Consider topical or oral antihistamines.
Fireworms	Apply 5% acetic acid (vinegar) topically. Remove bristles.	Consider topical corticosteroids.

**TABLE 121-2** Early Treatment of Marine Envenomations

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 207, “Trauma and Envenomation from Marine Fauna,” by Geoffrey K. Isbister.

## **CHAPTER 122**

# **High-Altitude Medical Problems**

Shaun D. Carstairs

High-altitude syndromes are due primarily to hypoxia; the rapidity and height of ascent influence the risk of occurrence.

## **ACUTE MOUNTAIN SICKNESS**

### **Clinical Features**

Acute mountain sickness (AMS) is usually seen in nonacclimated people making a rapid ascent to higher than 2000 m (6560 ft) above sea level. Symptoms resembling a hangover may develop within 6 hours after arrival at altitude but may be delayed as long as 1 day. Typical symptoms include bifrontal headache, anorexia, nausea, weakness, and fatigue. Worsening headache, vomiting, oliguria, dyspnea, and weakness indicate progression of AMS. Physical examination findings in early AMS are limited. Postural hypotension and peripheral and facial edema may occur. Localized rales are noted in 20% of cases. Funduscopy shows tortuous and dilated veins; retinal hemorrhages are common at altitudes higher than 5000 m (16,500 ft). Resting SaO<sub>2</sub> is typically normal for altitude and correlates poorly with the diagnosis of AMS.

### **Diagnosis and Differential**

The differential diagnosis includes hypothermia, carbon monoxide poisoning, pulmonary or central nervous system infections, migraine, dehydration, and exhaustion. The diagnosis is based largely on history of rapid ascent and symptoms.

## **Emergency Department Care and Disposition**

The goals of treatment are to prevent progression, abort the illness and improve acclimatization.

1. Terminate further ascent until symptoms resolve. For mild AMS, symptomatic therapy includes an analgesic, such acetaminophen or an NSAID, and an antiemetic, such as **ondansetron**, 4 milligrams every 4 to 6 hours PO, disintegrating tablet. Mild AMS usually improves or resolves in 12 to 36 hours if ascent is stopped.
2. A decrease in altitude of 300 to 1000 m should provide prompt relief of symptoms. Immediate descent and treatment are indicated for patients with moderate AMS or if there is a change in the level of consciousness, ataxia, or pulmonary edema.
3. Low flow **oxygen** also relieves symptoms.
4. Consider hyperbaric therapy for moderate AMS if descent is not possible.
5. Pharmacologic therapy for moderate AMS includes **acetazolamide**, 125 to 250 milligrams PO twice daily (in children 2.5 milligrams/kilogram twice daily), until symptoms resolve and **dexamethasone** 4 milligrams PO, IM, or IV every 6 hours with a taper over several days.
  - a. Indications for acetazolamide are (a) history of altitude illness, (b) abrupt ascent higher than 3000 m (9840 ft), (c) AMS, and (d) symptomatic periodic breathing during sleep at high altitude.
  - b. Acetazolamide pharmacologically produces an acclimatization response by inducing a bicarbonate diuresis and metabolic acidosis. Acetazolamide is effective for both prophylaxis and treatment.
  - c. Acetazolamide is contraindicated in sulfa-allergic patients.
6. Patients who respond well to treatment may be discharged. Provide counseling on preventing future episodes: graded ascent, avoidance of overexertion, alcohol, and respiratory depressants and prophylaxis using acetazolamide (start a day before ascent and continue for at least 2 days after reaching high altitude).

## **HIGH-ALTITUDE PULMONARY EDEMA**

Risk factors for high-altitude pulmonary edema (HAPE) include rapid ascent, heavy exertion, cold, pulmonary hypertension, and use of a sleep medication. Children with acute respiratory infections may be more susceptible to HAPE. HAPE may be fatal if not recognized and treated early.

## **Clinical Features**

HAPE usually begins on the second to fourth night at a new altitude and may progress quickly from dry cough and impaired exercise capacity to resting dyspnea, productive cough, severe weakness and cyanosis. Physical examination findings include tachycardia, tachypnea, localized or generalized rales, and signs of pulmonary hypertension, such as a prominent P<sub>2</sub> and right ventricular heave. Resting SaO<sub>2</sub> is low for altitude and drops significantly with exertion. CXR abnormalities progress from interstitial to localized to generalized alveolar infiltrates. Right axis deviation and a right ventricular strain pattern are seen on EKG with progressive disease.

## **Diagnosis and Differential**

The differential diagnosis includes pneumonia, acute asthma, congestive heart failure, myocardial ischemia, and pulmonary embolism. Decreased exercise performance and dry cough are enough to suspect early HAPE. A key to diagnosis is response to treatment.

## **Emergency Department Care and Disposition**

Early recognition of HAPE is essential to prevent progression. General measures include rest and keeping patients warm.

1. Initiate supplemental **oxygen** and titrate to  $\text{SaO}_2 \geq 90\%$ .

2. **Immediate descent** is the treatment of choice. Hyperbaric treatment may be used if descent is not an option. Patients with very mild cases of HAPE may be managed with bedrest and oxygen alone.
3. Pharmacologic treatment is usually unnecessary if descent and oxygen are available. In such cases (field conditions), options include **nifedipine** 20 to 30 milligrams extended release PO every 12 hours, **sildenafil** 50 milligrams PO 3 times daily or **tadalafil** 10 milligrams PO twice daily. Sildenafil and tadalafil blunt hypoxic pulmonary vasoconstriction. Nifedipine, sildenafil, or tadalafil may also be used for HAPE prophylaxis in persons with prior episodes. Inhaled **albuterol** 2 to 4 puffs every 4 to 6 hours may be used for both prophylaxis and treatment but is not well studied.
4. Patients may be discharged if clinical improvement is noted and room air SaO<sub>2</sub> remains >90%.

## **HIGH-ALTITUDE CEREBRAL EDEMA**

### **Clinical Features**

High-altitude cerebral edema (HACE) is defined as progressive neurologic deterioration of AMS or HAPE. Patients present with altered mental status, ataxia, stupor, and progress to coma if untreated. Focal neurologic signs such as third and sixth cranial nerve palsies may be present.

### **Diagnosis and Differential**

The differential diagnosis includes stroke or transient ischemic attack, tumor, meningitis, encephalitis, or metabolic disturbance. Increased T2 signaling in the splenium of the corpus callosum is seen on MRI. Laboratory testing to rule out other diagnoses is warranted but should not delay treatment.

### **Emergency Department Care and Disposition**



1. Initiate supplemental oxygen and titrate to  $\text{SaO}_2 \geq 90\%$ . Comatose patients require intubation and ventilation.
2. **Immediate descent** is needed. Initiate hyperbaric therapy if descent is not possible.
3. Administer **dexamethasone** 8 milligrams PO, IM or IV, followed by 4 milligrams PO, IM, IV every 6 hours.
4. In intubated patients, monitor arterial blood gases, taking care to avoid lowering  $\text{PaCO}_2$  below 30 mm Hg. Monitor intracranial pressures and cerebral blood velocities by transcranial Doppler US, if possible.
5. Patients remaining ataxic or confused after descent require admission.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 216, “High-Altitude Medical Problems,” by Peter H. Hackett and Jenny Hargrove.

## **CHAPTER 123**

# **Dysbarism and Complications of Diving**

Christian A. Tomaszewski

Dysbarism is commonly encountered in scuba divers and refers to complications associated with changes in environmental ambient pressure and with breathing compressed gases. These effects are governed by the gas laws: Boyle law states that pressure and volume are inversely related; Henry law states that, at equilibrium, the quantity of gas in solution is proportional to the partial pressure of that gas; Henry law states that total pressure exerted by a mixture of gases is the sum of the partial pressures of each gas.

## **CLINICAL FEATURES**

Barotrauma is the most common diving-related affliction and is caused by the direct mechanical effects of pressure, as gas-filled cavities in the body contract or expand with pressure. The most common form of barotrauma occurs during descent and is middle ear squeeze, or barotitis media. It is caused by inability to equalize pressure causing tympanic membrane bleeding or rupture and may result in conductive hearing loss. A forceful Valsalva during equalization can cause inner ear barotrauma with rupture of the round or oval window. Symptoms include tinnitus, sensorineural hearing loss, and vertigo. If the sinus ostia are occluded on descent, an impending squeeze can cause bleeding from the maxillary or frontal sinuses, resulting in pain and epistaxis.

Barotrauma during ascent is due to expansion of gas in body cavities. In the middle ear, the pressure differential from asymmetrical expansion can cause alternobaric vertigo. Although rare, “reverse squeeze” may affect the ear or sinuses during ascent with rupture. Pulmonary overinflation or burst lung can occur during rapid, panicked ascents if divers fail to exhale or if intrinsic pulmonary air trapping exists (eg, COPD) resulting in

pneumediastinum, subcutaneous emphysema or pneumothorax. The most serious consequence is cerebral arterial gas embolism (CAGE). Neurologic symptoms occur on ascent or immediately upon surfacing and include loss of consciousness, seizure, blindness, disorientation, hemiplegia, or other signs of stroke.

Divers using compressed air, caisson (tunnel) workers, and high-altitude pilots can all present with decompression sickness (DCS). In divers, this usually results from exceeding the dive table limits for depth and time. DCS can occur within minutes to hours of surfacing, rarely days later. Excessive bubble formation in tissue or circulation from saturated gas can cause both acute occlusive and delayed inflammatory effects. Type I DCS includes mottled skin and deep pain of the joints, usually the shoulder or knee, and is unaffected by movement. Type II, “serious,” DCS involves the central nervous system, typically the spine. Patients may initially complain of truncal constriction with ascending paralysis. Prolonged exposure at depth can lead to cardiopulmonary “chokes” or vestibular “staggers.” Because DCS and CAGE can be difficult to distinguish, or present simultaneously, the term “decompression illness” is now typically used.

## **DIAGNOSIS AND DIFFERENTIAL**

Dive profile (depth, duration, and repetitiveness) and time of symptom onset are the most useful historical factors in distinguishing dysbarism from other disorders. During descent, the most common maladies are the squeezes. A fistula test, insufflation of the tympanic membrane on the affected side causing the eyes to deviate to the contralateral side, may help diagnose inner ear barotrauma. During ascent, barotrauma or alternobaric vertigo is most likely to occur. A CXR may reveal pneumomediastinum, pneumothorax or subcutaneous air after pulmonary overinflation. If accompanied by early neurological symptoms, CAGE should be considered.

The differential diagnosis for DCS is broad. Musculoskeletal complaints could be joint strain or symptomatic herniated cervical disk. Chest pain may represent cardiac ischemia from overexertion. Immersion pulmonary edema

from noncardiogenic causes can occur during strenuous dives, particularly in cold water. Seizures at depth can result from breathing enriched mixtures of oxygen exceeding 1.4 atmospheres absolute. If DCS is suspected, a trial of pressure with hyperbaric oxygen usually results in some improvement.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

### **1. Decompression Illness (DCS/CAGE):**

- a. Administer **100% oxygen** and **IV fluids**.
  - b. If CAGE is suspected, place the patient in the supine position; place in the left lateral decubitus position if vomiting occurs.
  - c. Rapidly arrange for **recompression therapy (hyperbaric oxygen)**. Divers Alert Network (1-919-684-8111) may help provide chamber locations.
  - d. **Lidocaine** 1 milligram/kilogram IV bolus followed by a continuous infusion at 1 milligram/min may provide neuroprotection.
2. Treat middle ear barotitis with **decongestants** and **analgesics**. Consider antibiotics. Advise patients against diving until healing is completed. Inner ear barotrauma requires bed rest with the head upright until otolaryngologic evaluation for possible surgical exploration.
3. Pulmonary overinflation with ascent may require **needle decompression** or **tube thoracostomy** if a pneumothorax develops.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 208, "Dysbarism and Complications of Diving," by Brain Snyder and Tom Neuman.

# **CHAPTER 124**

## **Near Drowning**

Richard A. Walker

Prognosis after submersion injuries depends on the degree of pulmonary and central nervous system injury and therefore is highly dependent on early rescue and resuscitation. Prevention is the most important means to reduce associated morbidity and mortality.

### **CLINICAL FEATURES**

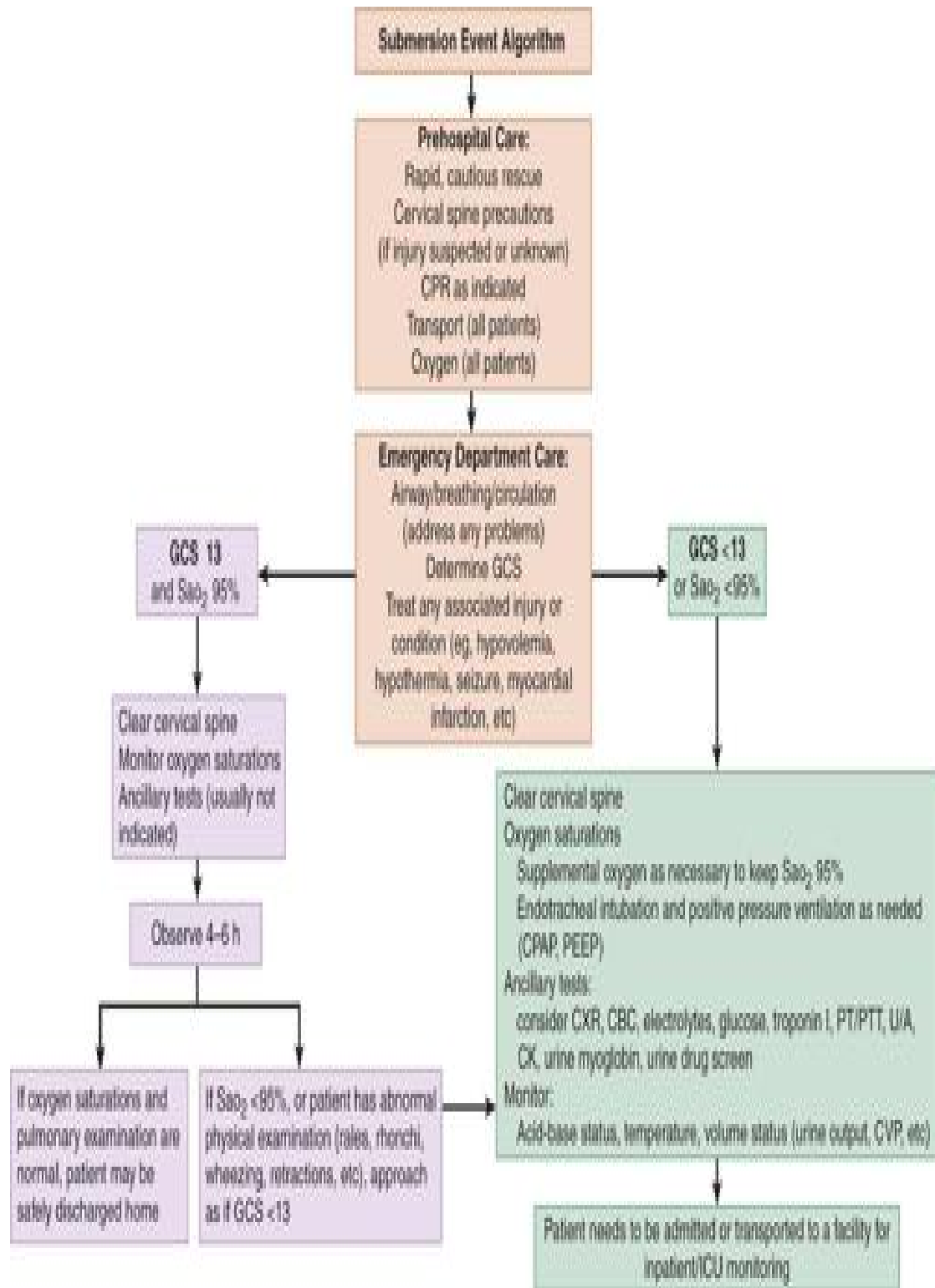
Up to 20% of patients who suffer submersion injuries do not aspirate water. Patients who aspirate water into their lungs have washout of surfactant, resulting in diminished alveolar gas transfer, atelectasis, ventilation perfusion mismatch, and hypoxia. Noncardiogenic pulmonary edema results from moderate to severe aspiration. Physical examination findings at presentation vary. Lungs may be clear or have rales, rhonchi, or wheezes. Mental status ranges from normal to comatose. Patients are at risk for hypothermia even in “warm water” submersions.

### **DIAGNOSIS AND DIFFERENTIAL**

Evaluate patients for associated injuries (spinal cord) and underlying precipitating disorders including syncope, seizures, hypoglycemia, and acute myocardial infarction or dysrhythmias. Respiratory acidosis may be present early followed by metabolic acidosis later. Early electrolyte disturbances are unusual. A CXR is usually obtained but is frequently normal in patients who are otherwise asymptomatic.

### **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Treatment for submersion events is summarized in [Fig. 124-1](#).



**FIGURE 124-1.** Submersion event algorithm.

2. Measure core temperature. Treat hypothermia if present. (See [Chapter 118](#) “Frostbite and Hypothermia”)
3. Data do not support routine antibiotic prophylaxis for pulmonary aspiration.
4. Efforts at “brain resuscitation,” including the use of mannitol, loop diuretics, hypertonic saline, fluid restriction, mechanical hyperventilation, controlled hypothermia, barbiturate coma, and intracranial pressure monitoring, have not shown benefit.
5. Hypothermic victims of cold-water submersion with cardiac arrest should undergo prolonged and aggressive resuscitation maneuvers until they are normothermic or considered not viable.
6. Patients who arrive in the ED in asystole or cardiac arrest after warm water submersion and are normothermic have a poor prognosis for recovery without significant neurologic handicaps.

For further reading in Tintinalli’s *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 209, “Near Drowning,” by Alan L. Causey and Mark A. Nichter.

# CHAPTER 125

## Thermal and Chemical Burns

Sandra L. Werner

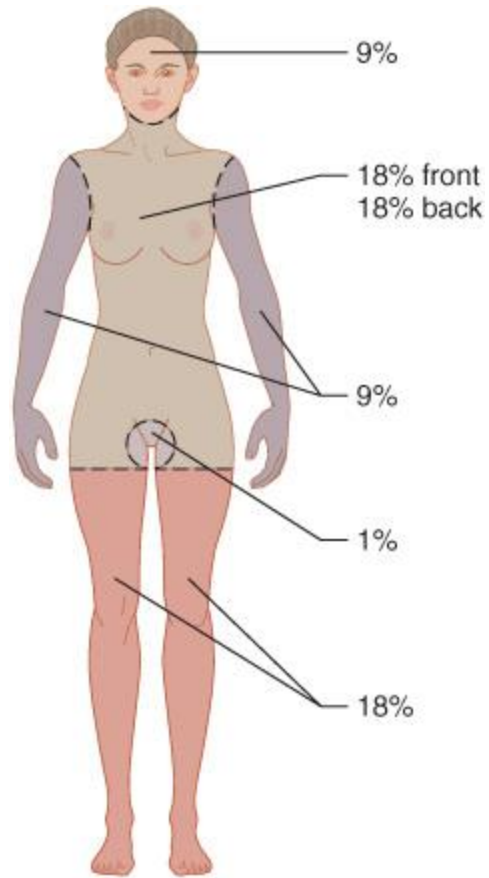
### THERMAL BURNS

The majority of burn patients are treated and released from the ED. Of those hospitalized, more than 60% are admitted to 1 of the country's 125 burn centers. The risk of death from a major burn is associated with larger burn size, advanced age, concomitant inhalation injury, and female sex.

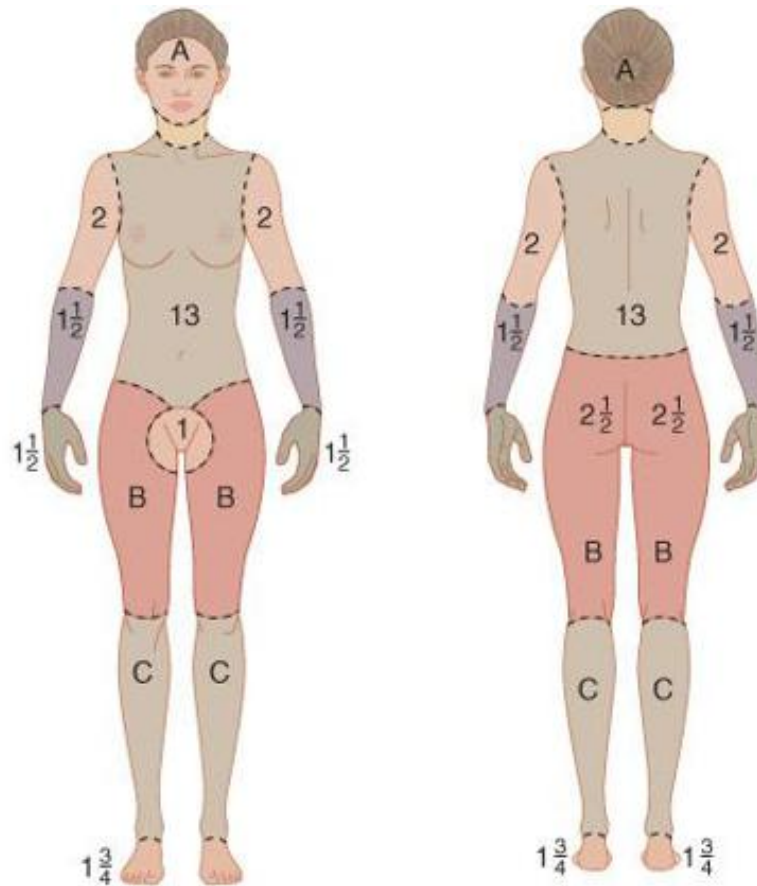
#### Clinical Features

Burns are categorized by their size and depth. Burn size is calculated as the percentage of body surface area (BSA) involved. The most common method to estimate this is the rule of 9s ([Fig. 125-1](#)). A more accurate tool, especially in infants and children, is the Lund and Browder burn diagram ([Fig. 125-2](#)). For smaller burns, the patient's hand can be used to estimate the percentage of BSA, as the area of the back of the patient's hand represents approximately 1% of BSA.





**FIGURE 125-1.** Rule of 9s to estimate the percentage of burn.



Relative percentages of areas affected by growth (age in years)

	0	1	5	10	15	Adult
A: half of head	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$	$3\frac{1}{2}$
B: half of thigh	$2\frac{3}{4}$	$3\frac{1}{4}$	4	$4\frac{1}{4}$	$4\frac{1}{2}$	$4\frac{3}{4}$
C: half of leg	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{3}{4}$	3	$3\frac{1}{4}$	$3\frac{1}{2}$

Second-degree \_\_\_\_\_ and

Third-degree \_\_\_\_\_ =

Total percent burned \_\_\_\_\_

**FIGURE 125-2.** Lund and Browder diagram to estimate the percentage of a burn.

Burn depth historically has been described in degrees: first, second, third, and fourth. A more clinically relevant classification scheme categorizes burns as superficial partial thickness, deep partial thickness, and full thickness. [Table 125-1](#) summarizes the characteristics of each type of burn.

Burn Depth	Histology/Anatomy	Example	Healing
First-degree	Epidermis No blisters, painful	Sunburn	7 days
Superficial second-degree or superficial partial thickness	Epidermis and superficial dermis Blisters, very painful	Hot water scald	14 to 21 days, no scar
Deep second-degree or deep partial thickness	Epidermis and deep dermis, sweat glands, and hair follicles Blisters, very painful	Hot liquid, steam, grease, flame	3 to 8 weeks, permanent scar
Third-degree	Entire epidermis and dermis charred, pale, leathery; no pain	Flame	Months, severe scarring, skin grafts necessary
Fourth-degree	Entire epidermis and dermis, as well as bone, fat, and/or muscle	Flame	Months, multiple surgeries usually required

**TABLE 125-1** Burn Depth Features Classified by Degree of Burn

Inhalation injury occurs most frequently in closed-space fires and in patients with decreased cognition (intoxication, overdose, head injury). Both the upper and lower airway can be injured by heat, particulate matter, and toxic gases. Thermal injury is usually limited to the upper airway, and can result in acute airway compromise. Particulate matter can reach the terminal bronchioles and lead to bronchospasm and edema. Clinical indicators of inhalation injury include facial burns, singed nasal hair, soot in the upper airway, hoarseness, carbonaceous sputum, and wheezing. Carbon monoxide poisoning should be suspected in all patients with inhalation injuries. Hydrogen cyanide poisoning should be considered in fires involving nitrogen-containing polymer products such as wool, silk, polyurethane, and vinyl.

## Diagnosis and Differential

The American Burn Association (ABA) classifies burns into major, moderate, and minor. [Table 125-2](#) summarizes the ABA burn classifications.

Burn Classification	Burn Characteristics	Disposition
Major burn	Partial thickness >25% BSA, age 10 to 50 years Partial thickness >20% BSA, age <10 y or >50 years Full thickness >10% BSA in anyone Burns involving hands, face, feet, or perineum Burns crossing major joints Circumferential burns of an extremity Burns complicated by inhalation injury Electrical burns Burns complicated by fracture or other trauma Burns in high-risk patients	Burn center treatment
Moderate burn	Partial thickness 15% to 25% BSA, age 10 to 50 years Partial thickness 10% to 20% BSA, age <10 years or >50 years Full thickness burns ≤10% BSA in anyone No major burn characteristics present	Hospitalization
Minor burn	Partial thickness <15% BSA, age 10 to 50 years Partial thickness <10% BSA, age <10 years or >50 years Full thickness <2% in anyone No major burn characteristics present	Outpatient treatment

Key: BSA = body surface area.

**TABLE 125-2** Burn Depth Features: American Burn Association Burn Classification

## Emergency Department Care and Disposition

Management of patients with *moderate to major* burns is divided into 3 phases: prehospital care, ED resuscitation and stabilization, and transfer to a burn center. Prehospital burn care consists of stopping the burning process, establishing an airway, initiating fluid resuscitation, relieving pain and protecting the burn wound.

1. In the ED, reevaluate the airway, administer **100% O<sub>2</sub>**, and intubate and ventilate the patient if there are signs of airway compromise or an airway burn. Obtain an ABG, carboxyhemoglobin level, and CXR. Monitor vital signs and oxygen saturation. Arrange bronchoscopy if inhalation injury is a concern.
2. Establish 2 IV lines in unburned areas. Use a burn formula, such as the Parkland formula, to guide initial **fluid resuscitation** ([Table 125-3](#)).

Ongoing fluid resuscitation is further guided by vital signs, cerebral and peripheral perfusion, and adequate urine output. Consult with a burn specialist as soon as possible.

Adults LR 4 mL × weight (kg) × % BSA burned* over initial 24 h Half over the first 8 h from the time of burn Other half over the subsequent 16 h Example: 70-kg adult with 40% second and third degree burns: 4 mL × 70 kg × 40 = 11 200 mL over 24 h
Children LR 3 mL × weight (kg) × % BSA burned* over initial 24 h plus maintenance Half over the first 8 h from the time of burn Other half over the subsequent 16 h

Key: BSA = body surface area; LR = lactated Ringer's solution.

\*Second and third degree burns only.

**TABLE 125-3** Parkland Formula for Fluid Resuscitation

3. Evaluate and treat traumatic injuries using standard trauma resuscitation guidelines. ([Chapter 156](#) “Trauma in Adults,” [Chapter 157](#) “Trauma in Children,” [Chapter 158](#) “Trauma in Elderly”)
4. After initiating resuscitative measures, address burn wound care. Apply cool compresses to small burns. Cover large burns with sterile dry sheets as saline soaked dressings may induce hypothermia. ED administration of empiric antibiotics and application of topical antimicrobials during resuscitation are not recommended.
5. Administer intravenous opioid analgesia early and titrate to pain.
6. Treat inhalation injuries with humidified oxygen, endotracheal intubation and mechanical ventilation, bronchodilators, and pulmonary toilet. **Hyperbaric oxygen therapy** is used for severe carbon monoxide poisoning.
7. Circumferential burns of the neck, chest or limbs may compromise breathing and circulation. **Escharotomy** may be required.
8. Update tetanus prophylaxis, if needed.
9. Hospitalize patients with moderate and major burns. The ABA’s criteria for referral to a burn unit are listed in [Table 125-4](#).

Third degree burns in any age group Electrical burns, including lightning injury Chemical burns Inhalation injury Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality Burn injury in any patients with concomitant trauma (such as fractures) in whom the burn injury poses the greatest risk of morbidity or mortality Burn injury in children in hospitals without qualified personnel or equipment to care for children Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention Burn injury in children <10 years and adults >50 years of age
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**TABLE 125-4** American Burn Association Burn Unit Referral Criteria

10. [Table 125-5](#) summarizes the care of minor burns. Patients with minor burns may be discharged after ED treatment provided close follow-up is available.

Provide appropriate analgesics before burn care and for outpatient use Cleanse burn with mild soap and water or dilute antiseptic solution Debride wound as needed Apply topical antimicrobial: 1% silver sulfadiazine cream (not on the face or in patients with a sulfa allergy) Bacitracin ointment Triple-antibiotic ointment (neomycin, polymyxin B, bacitracin zinc) Consider use of synthetic occlusive dressings Provide detailed burn care instructions with follow-up in 24 to 48 h
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**TABLE 125-5** ED Care of Minor Burns

## CHEMICAL BURNS

More than 25,000 products are capable of producing chemical burns. Chemical burn injuries account for 5% to 10% of burn center admissions.

### Clinical Features

Clinical features depend on the type of agent, concentration, volume, and duration of exposure. Alkalis usually produce more damage than acids. Acids typically cause coagulation necrosis which produces an eschar that

limits further damage. Alkalis produce liquefaction necrosis, allowing deeper damage to occur. Hydrofluoric (HF) acid is a special case as it rapidly penetrates intact skin and can cause progressive pain and deep tissue destruction without obvious superficial tissue damage. Systemic toxicity, including hypotension, acidosis, and shock, may occur if certain chemicals are absorbed.

Chemical burns of the eye are true ocular emergencies. Acid ocular burns quickly precipitate proteins in the superficial eye structures resulting in a “ground glass” appearance of the cornea. Alkali ocular burns are more severe due to deeper ongoing penetration. Lacrimators (tear gas and pepper mace) cause ocular, mucous membrane, and pulmonary irritation.

## Diagnosis and Differential

The diagnosis of chemical burn usually is made by history of exposure to a chemical agent. Chemical topical exposures should be considered in all cases of skin irritation and/or pain. For ocular exposures, pH paper can distinguish alkali from acid exposure.

## Emergency Department Care and Disposition

1. The first priority in the treatment of chemical burns is to terminate the burning process. Remove garments. Brush off dry chemical particles. **Immediately irrigate the skin copiously with water.** Cover elemental metals (sodium, lithium, calcium, and magnesium) with **mineral oil** because exposure to water may cause a severe exothermic reaction.
2. For ocular burns, begin irrigation of each involved eye with 1 to 2 L of normal saline. In patients with acid or alkali burns, continue irrigation until the pH is normal. Patients with alkali burns may require prolonged irrigation. *Visual acuity check and pH testing should follow, not precede, initial ocular irrigation.* Consult with an ophthalmologist.
3. Treatment for specific chemical burns is provided in [Table 125-6](#). Options for treating cutaneous HF acid burns are provided in [Table 125-7](#).

Consult with a plastic surgeon for patients with HF acid burns of the hands, feet, digits, or nails.

Chemical	Treatment	Comments
<b>Acids</b>		
All acid burns require prompt decontamination and copious irrigation with water.		
Acetic acid	Copious irrigation.	Consider systemic antibiotics for extensive scalp burns.
Phenol (carbolic acid)	Copious irrigation.  Swab with polyethylene glycol 300 and industrial methylated spirits in a 2:1 mixture.	Isopropyl alcohol may also be used.
Chromic acid	Copious irrigation. 5% thiosulfate soaks or ascorbic acid creams.	Observe for systemic toxicity.
Formic acid	Copious irrigation.	Dialysis may be needed for severe toxicity.
Hydrofluoric acid	Copious irrigation. Calcium gluconate gel.	Consider SC or intradermal injection of 5% calcium gluconate or intraarterial calcium gluconate for severe cases.
Nitric acid	Copious irrigation.	Consult with burn specialist.
Oxalic acid	Copious irrigation.  IV calcium may be required.	Evaluate serum electrolytes and renal function. Cardiac monitoring for serious dermal exposure.
<b>Alkalis</b>		
All alkali burns require prompt decontamination and copious, prolonged irrigation with water.		
Portland cement	Prolonged copious irrigation.	May need to remove cement particles with a brush, such as a preoperative scrubbing brush.
<b>Elemental Metals</b>		
Water is generally contraindicated in extinguishing burning metal fragments embedded in the skin.		
Elemental metals (sodium, lithium, potassium, magnesium, aluminum, and calcium)	Cover metal fragments with sand, foam from a Class D fire extinguisher, or with mineral oil. Excise metal fragments that cannot be wiped away.	—



<b>Hydrocarbons</b>		
Gasoline Tar	Decontamination. Cool before removal. Remove using ointment containing polyoxylene sorbitan (polysorbate) or De-solv-it.	— Mayonnaise can be used.
<b>Vesicants</b>		
Mustards	Decontaminate. Copious irrigation.	If limited water supply, adsorbent powders (flour, talcum powder, fuller's earth) can be applied to the mustard and then wiped away with a moist towel.
<b>Reducing Agents</b>		
Alkyl mercury compounds	Copious irrigation.  Debride, drain, and copiously irrigate blisters.	Blister fluid is high in metallic mercury content.
<b>Lacrimators</b>		
Tear gas	Copious irrigation.	May cause respiratory symptoms if inhaled.
Pepper spray	Copious irrigation.	May cause respiratory symptoms if inhaled.
<b>Miscellaneous</b>		
White phosphorus	Remove clothing. Copious irrigation. Debride visible particles.	Systemic toxicity is a significant concern.
Airbag	Prolonged copious irrigation.	

**TABLE 125-6** Treatment of Select Chemical Burns

1. Copious irrigation for 15 to 30 min immediately.
2. Application of calcium gluconate gel, 25 mL of 10% calcium gluconate in 75 mL of sterile water-soluble lubricant.
3. Further treatment options as dictated by patient response: <ul style="list-style-type: none"> <li>a. Dermal injection of 5% calcium gluconate up to a maximum of 1 mL per cm<sup>2</sup> of skin surface using a small gauge needle.</li> <li>b. Regional block using Bier method with 40 mL 10% calcium gluconate.</li> <li>c. Arterial infusion over 2 to 4 h (40 mL of 5% dextrose in water with 10 mL of 10% calcium gluconate).</li> <li>d. Consider supplemental magnesium IV.</li> </ul>

**TABLE 125-7** Options for Treatment of Hydrofluoric Acid Skin Burns

4. After initial decontamination measures, initiate IV fluid resuscitation, analgesia, tetanus immunoprophylaxis, and address systemic toxicity, as needed.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 210, “Thermal Burns,” by Lawrence R. Schwartz and Chenicheri Balakrishnan; and Chapter 211, “Chemical Burns,” by Fred P. Harchelroad Jr. and David M. Rottinghaus.

## **CHAPTER 126**

# **Electrical and Lightning Injuries**

Sachita Shah

## **ELECTRICAL INJURIES**

Electrical injuries present with a wide spectrum of damage, from superficial skin burns to multisystem injury. Electrical injuries are arbitrarily classified as low voltage ( $\leq 1000$  V) and high voltage ( $> 1000$  V). At very high voltages, an electric arc may also travel from a voltage source to a person and cause severe burns. Standard household electricity is low voltage alternating current (AC), power lines are high voltage. Young children most often sustain low voltage injuries in the home from electrical outlets or chewing on electrical wires, whereas adults more often sustain high voltage injuries while at work.

### **Clinical Features**

Electrical injuries cause injury via several mechanisms: direct tissue damage from electrical energy, thermal damage from heat created by tissue resistance, and mechanical injury induced by a fall or tetanic muscle contraction. Severity of injury and tissues affected depend on voltage, duration of contact, tissue resistance, and path of current. The risk for serious injury increases with as voltage increases. Patients may sustain immediate cardiac dysrhythmias (including ventricular fibrillation), respiratory arrest or seizures. Cardiac complications, such as arrhythmias and QT prolongation are more commonly seen in high voltage injuries. Temporary loss of consciousness is common. Severe burns may result from contact with high voltage lines. Entrance and exit burns are typically painless, gray to yellow depressed areas. The size of the skin injury does

not correlate well with internal injuries. Traumatic injuries frequently accompany electrical injuries. Details of specific immediate and delayed systemic injuries and complications are summarized in [Table 126-1](#).

Cardiovascular	Sudden death (ventricular fibrillation, asystole), chest pain, dysrhythmias, ST-T segment abnormalities, bundle branch block, myocardial damage, myocardial infarction (rare), hypotension (volume depletion), hypertension (catecholamine release)
Neurologic	Altered mental status, agitation, coma, seizures, cerebral edema, hypoxic encephalopathy, headache, aphasia, weakness, paraplegia, quadriplegia, spinal cord dysfunction (may be delayed), peripheral neuropathy, cognitive impairment, insomnia, emotional lability
Cutaneous	Electrothermal contact injuries, noncontact arc and "flash" burns, secondary thermal burns (clothing ignition, heating of metal)
Vascular	Thrombosis, coagulation necrosis, disseminated intravascular coagulation, delayed vessel rupture, aneurysm, compartment syndrome
Pulmonary	Respiratory arrest (central or peripheral, eg, muscular tetany), aspiration pneumonia, pulmonary edema, pulmonary contusion, inhalation injury
Renal/metabolic	Acute renal failure (due to heme pigment deposition and hypovolemia), myoglobinuria, metabolic (lactic) acidosis, hypokalemia, hypocalcemia, hyperglycemia
Gastrointestinal	Perforation, stress ulcer (Curling ulcer), GI bleeding, GI tract dysfunction, various reports of lethal injuries at autopsy
Muscular	Myonecrosis, compartment syndrome
Skeletal	Vertebral compression fractures, long bone fractures, shoulder dislocations (anterior and posterior), scapular fractures
Ophthalmologic	Corneal burns, delayed cataracts, intraocular hemorrhage or thrombosis, uveitis, retinal detachment, orbital fracture
Auditory	Hearing loss, tinnitus, tympanic membrane perforation (rare), delayed mastoiditis or meningitis
Oral burns	Delayed labial artery hemorrhage, scarring and facial deformity, delayed speech development, impaired mandibular/dentition development
Obstetric	Spontaneous abortion, fetal death

Key: GI = gastrointestinal.

**TABLE 126-1** Immediate and Delayed Complications of Electrical Injuries

## Diagnosis and Differential

Diagnosis of electrical injury is usually based on history of contact with an electrical source. The type of current and surrounding circumstances may

help direct the initial evaluation.

Characteristic skin lesions or oral lesions (in children) may provide diagnostic clues if the patient or witnesses are unable to provide a history. Laboratory and radiographic evaluation of high voltage injuries should follow standard trauma guidelines. ([Chapter 156](#) “Trauma in Adults,” [Chapter 157](#) “Trauma in Children,” [Chapter 158](#) “Trauma in Elderly”). An elevated serum CK, myoglobin or urine myoglobin suggests extensive muscle injury and rhabdomyolysis. Atrial or ventricular arrhythmias, bradyarrhythmias, prolonged QT intervals or ST-T wave abnormalities may be noted on ECG. Assessment and treatment of complications associated with electrical injuries are summarized in [Table 126-2](#).

Circulatory	Use the Parkland fluid resuscitation formula.*
Renal	Initiate fluid resuscitation.
Nervous system	Order head CT as needed for altered mental status; assess for spinal cord and peripheral nerve injury.
Skin	Assess and treat cutaneous burns.†
Musculoskeletal	Perform a careful assessment of spine, pelvis, long bones, and joints. Assess for compartment syndrome and need for fasciotomy.
Vascular	Spasm may occur leading to delayed thrombosis, aneurysm formation, or muscle damage.
Coagulation	Treat coagulation disorders by eliminating the precipitating factor through early surgical debridement. If hemorrhage is present, replace coagulation factors.
Lungs	Assess for inhalation injury, carbon monoxide, alveolar injury from blast.
Eyes	Document complete eye examination. Delayed cataracts may develop.
Ears	Assess for blast injury. Document hearing. Middle and inner ear disorders and hearing loss may occur.
GI	Intraabdominal injury may occur from current or blast.
Lips and oral cavity	Watch for delayed bleeding.

\*Refer to Chapter 125 “Thermal and Chemical Burns.”

†Refer to Chapter 51 “Rhabdomyolysis.”

**TABLE 126-2** Assessment and Treatment of Complications of Electrical Injuries

The differential diagnosis includes other causes of arrhythmias, such as myocardial ischemia, and neurologic dysfunction, such as stroke, closed head injury, spinal cord injury.

## Emergency Department Care and Disposition

1. Assess and stabilize airway, breathing, and circulation abnormalities. Treat ventricular fibrillation, asystole, or ventricular tachycardia using standard ACLS protocols. Other dysrhythmias are usually transient and do not need immediate therapy. Treat traumatic injuries using standard trauma protocols. Treat seizures with standard therapy.
2. Provide continuous monitoring of vital signs, heart rate and rhythm, and pulse oximetry. Provide high-flow oxygen and begin IV crystalloids. Fluid requirements are usually greater than initially predicted using the Parkland Burn formula ([Chapter 125](#)). Monitor urine output with a Foley catheter.
3. Cover large burns with dry sterile dressings.
4. Monitor for rhabdomyolysis, compartment syndrome and renal failure. Treat rhabdomyolysis with aggressive fluid rehydration aiming for a urine output of 2 mL/kg/h ([Chapter 51](#)).
5. Provide pain control with opioids.
6. Reduce and immobilize fractures and dislocations.
7. Administer tetanus prophylaxis, if not up to date.
8. Clean and treat small cutaneous burns in the standard fashion. Prophylactic systemic antibiotics are usually not necessary unless large open contaminated wounds are present.
9. Consult with a general surgeon, trauma surgeon or burn surgeon as patients may require formal wound exploration, debridement, fasciotomy or transfer to a facility with specialty care. For pregnant patients, consult with an obstetrician for fetal monitoring and admission.
10. [Table 126-3](#) summarizes admission criteria. Patients with more than a minor low voltage injury should be admitted for further monitoring.

High voltage > 600 V
Symptoms suggestive of systemic injury
Cardiovascular: chest pain, palpitations
Neurologic: loss of consciousness, confusion, weakness, headache, paresthesias
Respiratory: dyspnea
Gastrointestinal: abdominal pain, vomiting
Evidence of neurologic or vascular injury to a digit or extremity
Burns with evidence of subcutaneous tissue damage
Dysrhythmia or abnormal electrocardiogram
Suspected foul play, abuse, suicidal intent, or unreliable social situation
Pregnancy
Associated injuries requiring admission
Comorbid diseases (cardiac, renal, neurologic)

**TABLE 126-3** Indications for Admission for Patients with Electrical Injuries

11. Children with isolated oral injuries or isolated hand wounds can usually be discharged home after consultation with an ENT specialist or plastic surgeon. Provide parents with instructions for controlling delayed labial artery bleeding. Arrange close follow-up with the consulting surgeon to assess scarring and stricture.
12. Asymptomatic patients who sustained a low voltage injury ( $\geq 240$  V) and have a normal ECG on presentation and normal physical exam may be discharged home.

## **INJURIES DUE TO ELECTRONIC CONTROL DEVICES**

Electronic control devices, such as the cattle prod, stun gun, and the TASER<sup>®</sup>, deliver high-voltage, low-amperage electrical pulses that induce involuntary muscle contraction, neuromuscular incapacitation, and/or pain. The likelihood of electrical injury is minimal. Injuries are usually limited to superficial punctures, and minor lacerations and cutaneous burns. The majority of deaths that have followed the use of these devices have occurred in persons who were extremely ill and agitated due to psychosis, stimulant drugs, or other conditions. Ill-appearing and agitated patients should be evaluated and treated in the same way as all patients who may have sustained blunt trauma or who have ingested unknown substances.

Cardiac monitoring and other testing are not needed just because a TASER<sup>®</sup> has been used.

## **LIGHTNING INJURIES**

Lightning is a unidirectional extremely high voltage current that causes substantially different injuries from those caused by high voltage AC electricity. Lightning emits brief, but intense, thermal radiation. Lightning often flashes over the skin, rather than through the body, which helps explain how some people survive a lightning strike with little or no injury.

### **Clinical Features**

The most common immediate cause of death after lightning strike is cardiac arrest or respiratory arrest. In the patient with spontaneous circulation, hypertension and tachycardia are caused by sympathetic activation. Patients may experience temporary loss of consciousness, confusion, and amnesia. Feathering or fern-shaped burns on the skin are pathognomonic of lightning. Tympanic membrane rupture may occur. Deep tissue injuries, myoglobinuria, and renal failure are uncommon unless the lightning has traveled through, rather than over, the patient or as a result of associated trauma. Details of specific immediate and delayed systemic injuries and complications are summarized in [Table 126-4](#).



System	Injury
Cardiovascular	Dysrhythmias (asystole, ventricular fibrillation/tachycardia, premature ventricular contractions), electrocardiographic changes, myocardial infarction (unusual)
Neurologic	Immediate or delayed, permanent or transient; loss of consciousness, confusion, amnesia, intracranial hemorrhage, hemiplegia, amnesia, respiratory center paralysis, cerebral edema, neuritis, seizures, parkinsonian syndromes, cerebral infarction, myelopathy, progressive muscular atrophy, progressive cerebellar syndrome, transient paralysis, paresthesias, myelopathy, autonomic dysfunction
Cutaneous	Burns (first to third degree), scars, contractures
Ophthalmologic	Cataracts (often delayed), corneal lesions, uveitis, iridocyclitis, vitreous hemorrhage, macular degeneration, optic atrophy, diplopia, chorioretinitis, retinal detachment, hyphema
Otologic	Tympanic membrane rupture, temporary or permanent deafness, tinnitus, ataxia, vertigo, nystagmus
Renal	Myoglobinuria, hemoglobinuria, renal failure (rare)
Obstetric	Fetal death, placental abruption
Miscellaneous	Secondary blunt trauma, compartment syndrome, disseminated intravascular coagulation

**TABLE 126-4** Complications Associated with Lightning Injuries

## Diagnosis and Differential

The diagnosis of lightning injury is based on history and should be considered in any critically ill patient found outside during or after a thunderstorm. While increasingly rare, indoor exposures to lightning may occur via indoor pools and use of hard-wired telephones. Carefully assess patients for burns, neurologic and cardiovascular complications, otologic and ophthalmologic injuries, and blunt trauma. Laboratory and radiographic evaluation of lightning injures should follow standard trauma guidelines. ([Chapter 156](#) “Trauma in Adults,” [Chapter 157](#) “Trauma in Children,” [Chapter 158](#) “Trauma in Elderly”) Differential diagnosis includes stroke or intracranial hemorrhage, seizure disorder, and cerebral, spinal cord, or other neurologic trauma.

## Emergency Department Care and Disposition

1. Even in a mass casualty situation, provide aggressive resuscitation in patients with respiratory and cardiac arrest due to lightning strike. Respiratory arrest may outlast initial cardiac arrest and adequate ventilation can prevent hypoxic injury until return of spontaneous circulation. Survival has been reported even after prolonged respiratory arrest.
2. Treat traumatic injuries using standard trauma protocols. Treat arrhythmias using standard ACLS protocols. Treat seizures with standard therapy.
3. Provide continuous monitoring of vital signs, heart rate and rhythm, and pulse oximetry. Provide high flow oxygen and begin IV crystalloids.
4. Hypotension is unexpected and should prompt investigation for hemorrhage.
5. Treat keraunoparalysis with expectant management.
6. Administer tetanus prophylaxis, if not up to date.
7. Admit patients with persistent musculoskeletal symptoms, neurologic, cardiac rhythm or vascular abnormalities, or significant burns to a critical care unit. Consult with a general surgeon, trauma surgeon or burn surgeon. All pregnant patients require obstetric consultation and admission for fetal monitoring.
8. Patients with minor injuries and a negative workup may be discharged with outpatient follow-up to assess delayed effects of lightning injury.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 212, “Electrical Injuries,” and Chapter 213, “Lightning Injuries,” by Raymond M. Fish.

# CHAPTER 127

## Carbon Monoxide

Christian A. Tomaszewski

Carbon monoxide is a colorless, odorless, nonirritating gas that displaces oxygen from hemoglobin, resulting in early tissue hypoxia and delayed neurologic damage. Sources of exposure to carbon monoxide include the incomplete combustion of any carbonaceous fuel (eg, gasoline, kerosene, natural gas, and charcoal) or the metabolism of inhaled methylene chloride (paint stripper).

### CLINICAL FEATURES

A history of exposure to gas heat or smoke inhalation, or multiple victims with altered mental status, acidosis, or coma should alert one to the possibility of carbon monoxide poisoning. The clinical features of carbon monoxide poisoning are highly variable and primarily relate to hypoxic effects on the cardiovascular and neurologic systems ([Table 127-1](#)). Symptoms range from from “flu-like” symptoms, such as headache, dizziness, nausea, and vomiting, to coma. The “classic finding” of cherry red lips is rarely seen in living patients. Patients with significant poisoning may experience long-term neurological and cognitive problems.

Headache
Visual disturbances
Vomiting
Confusion
Ataxia
Dyspnea/tachypnea
Seizure
ECC changes/dysrhythmias
Syncope
Retinal hemorrhage
Chest pain
Bullous skin lesions
Focal neurologic deficit

**TABLE 127-1** Signs and Symptoms of Acute Carbon Monoxide Poisoning

## **DIAGNOSIS AND DIFFERENTIAL**

A venous, or arterial, blood sample for cooximetry is the most reliable test to diagnose carbon monoxide poisoning. Although carboxyhemoglobin (COHb) levels confirm exposure, they do not necessarily correlate with symptoms or prognosis. Baseline COHb may be as high as 3% in nonsmokers and 10% in smokers. Higher levels are suggestive of CO exposure. The use of bedside pulse cooximetry in the ED to screen for CO exposure is still under investigation.

Standard pulse oximetry is unreliable in the presence of increasing COHb as oxygen saturation readings will be artificially high. Additional laboratory and imaging abnormalities seen in symptomatic patients may include elevated anion gap metabolic acidosis, elevated lactate, elevated creatine phosphokinase, elevated troponin, ECG changes consistent with ischemia, and bilateral globus pallidus lesions on MRI.

The differential diagnosis is wide due to the nonspecific nature of the symptoms and includes flu-like illness, gastroenteritis, exposure to other toxins, and infectious causes of mental status changes. Cardiovascular compromise after poisoning may represent a concomitant myocardial infarction.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Remove patients from the source of exposure and address airway, breathing, and circulation.

1. Begin treatment in all patients suspected of CO poisoning with the **highest concentration of supplemental oxygen** available (eg, 100% oxygen via facemask with reservoir) and continue until the patient is

asymptomatic. Provide continuous monitoring of vital signs, heart rate, and rhythm. Establish IV access.

- Guidelines for **hyperbaric oxygen therapy (HBO)** in patients with severe poisoning are listed in [Table 127-2](#). Indications for pediatric and adult HBO are similar. The threshold COHb for initiating HBO in pregnant patients is lower because of concerns for the fetus. Consult with a hyperbaric specialist. Patients must have a secure airway and stable hemodynamics before transport and treatment with HBO as access may be limited en route and in the chamber.

Syncope Confusion/altered mental status Seizure Coma Focal neurologic deficit Pregnancy with carboxyhemoglobin level >15% Blood level >25% Evidence of acute myocardial ischemia
---

**TABLE 127-2** Commonly Utilized Indications for Referral for Hyperbaric Oxygen Treatment

- Guidelines for disposition are listed in [Table 127-3](#). Ensure that the home or work environment is no longer a source of carbon monoxide exposure.

Symptom Severity	Disposition	Comments
Minimal or no symptoms	Home	Assess safety issues
Headache	Home after symptom resolution	Administer 100% oxygen in ED
Vomiting Elevated carbon monoxide level		Observe 4 h Assess safety issues
Ataxia, seizure, syncope, chest pain, focal neurologic deficit, dyspnea, ECG changes	Hospitalize  Consult with hyperbaric specialist	Administer 100% oxygen in ED  Carbon monoxide level, comorbid conditions, including pregnancy, age and stability of the patient must be considered if considering transfer for hyperbaric oxygen

**TABLE 127-3** Disposition Considerations

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 217, “Carbon Monoxide,” by Gerald Maloney.

# **CHAPTER 128**

## **Mushroom and Plant Poisoning**

B. Zane Horowitz

### **MUSHROOMS**

Mushroom poisoning occurs in 4 groups: foragers who purposefully harvest mushrooms or plants for food; teenagers and young adults who use mushrooms to get “high”; preschool-age children who accidentally ingest mushrooms while playing outdoors; and, rarely, victims of attempted homicide or suicide.

#### **Clinical Features**

Determine if patients ingested only 1 type or multiple types of mushrooms and time elapsed from ingestion to symptoms. Foragers may be able to provide a description of the mushroom. Clinical features of common mushroom poisonings are listed in [Table 128-1](#).

Symptoms	Mushrooms	Toxicity	Treatment
Gastrointestinal symptoms			
Onset <2 h	<i>Chlorophyllum molybdites</i> <i>Omphalotus illudens</i> <i>Cantharellus cibarius</i>	Nausea, vomiting, diarrhea (occasional bloody)	IV hydration Antiemetics
Onset 6 to 24 h	<i>Amanita Sordiana</i> (Delayed renal failure) <i>Amanita phalloides</i> , <i>Amanita verna</i> , <i>Amanita virosa</i> <i>Lepiota</i> sp. (Delayed liver failure) <i>Gyromitra esculenta</i> (Delayed onset seizures)	Initial: nausea, vomiting, diarrhea Day 2: rise in AST, ALT Day 3: hepatic failure coagulopathy renal failure hemolysis As above + headache, tremor, ataxia, seizures	IV hydration, closely monitor electrolytes, glucose, renal, hepatic and coagulation function For <i>Amanita</i> and <i>Lepiota</i> Acetylcysteine, load with 140 milligrams/kilogram PO/NG Penicillin G 300,000 to 1,000,000 units/kilogram IV per day Silymarin 5 milligrams/kilogram IV over 1 h, then 20 milligrams/kilogram IV per day (available PO in the United States) For <i>Gyromitra</i> , treat seizures with both benzodiazepines AND pyridoxine 5 grams IV
Muscarinic (SLUDGE) syndrome Onset <30 min	<i>Inocybe</i> sp. <i>Citocybe</i> sp.	Salivation, lacrimation, diarrhea, gastrointestinal distress, emesis	Supportive, atropine 0.01 milligrams/kilogram IV repeated as needed for severe secretions
CNS excitement Onset <30 min	<i>Amanita muscaria</i> <i>Amanita pantherina</i> <i>Amanita gemmata</i>	Intoxication, delirium, ataxia, visual disturbances, seizures, tachycardia, hypertension, warm dry skin, dry mouth, mydriasis (anticholinergic effects)	Supportive, sedation with diazepam 2 to 5 milligrams IV is needed for adults
Hallucinations Onset <30 min	<i>Panaeolus</i> <i>Panaeolus</i> sp. <i>Gymnopilus spectabilis</i>	Visual hallucinations, ataxia	Supportive, sedation with or diazepam 2 to 5 milligrams IV for adults
Diazepam Onset 2 to 72 h after mushroom, and <30 min after alcohol	<i>Coprinus atramentarius</i> <i>Citocybe cloveae</i>	Headache, flushing, tachycardia, hyperventilation, palpitations	Supportive IV hydration

Key: ALT = alanine aminotransferase, AST = aspartate aminotransferase, BUN = blood urea nitrogen, CNS = central nervous system, IV = intravenous, PT = prothrombin time, PTT = partial thromboplastin time, SLUDGE syndrome = salivation, lacrimation, urination, defecation, gastrointestinal hypermotility, and emesis.



## TABLE 128-1 Mushrooms: Symptoms, Toxicity, and Treatment

### Diagnosis and Differential

Most patients that develop GI symptoms within 2 hours of ingestion have a reassuring clinical course and do not develop major organ failure ([Table 128-1](#)). An exception to this is *Amanita smithiana* ingestion, which results in early GI symptoms and delayed renal failure. Mushrooms with potential liver, kidney, and CNS effects are often associated with onset of vomiting that is delayed for 6 or more hours after ingestion. Toxic species include *Amanita*, *Galerina*, *Gyromitra*, and *Lepiota*. Ingestions may be misdiagnosed as viral gastrointestinal illness or food poisoning if a history of mushroom ingestion is not pursued.

### Emergency Department Care and Disposition

Consultation with a poison center is advised as regional differences in mushrooms types and toxicity exist.

1. Treatment regimens for mushroom poisonings are listed in [Table 128-1](#).
2. Admit all patients with delayed onset of vomiting or diarrhea for ongoing monitoring of renal and liver functions and fluid status for 48 hours.
3. Rapid progression to hepatic encephalopathy, hepatorenal syndrome, or coagulopathy are indications for liver transplantation. Consider transfer to a liver transplant setting early in the course of mushroom ingestion.
4. Patients who ingest hallucinogenic mushrooms or mushrooms with muscarinic effects only may be discharged when symptoms subside.

## PLANTS

Most patients with plant related exposures and ingestions require no treatment and may be discharged after a short period of observation. [Tables](#)

[128-2](#) and [128-3](#) describe the clinical symptoms and treatment regimens of common and severe poisonous plant ingestions.

Plant	Symptoms	Treatment
Castor bean ( <i>Ricinus communis</i> )	Delayed gastroenteritis, delirium, seizures, coma, death	Whole-bowel irrigation Supportive care
Coyotillo ( <i>Karwinskia humboldtiana</i> )	Ascending paralysis	Supportive care
Foxglove ( <i>Digitalis purpurea</i> )	Nausea, vomiting, diarrhea, abdominal pain, confusion, cardiac dys-rhythmias	GI decontamination with activated charcoal Monitoring of potassium level Antidysrhythmics Digoxin-specific Fab antibody for dysrhythmias
Jequirity bean ( <i>Abrus precatorius</i> )	Delayed gastroenteritis, delirium, seizures, coma, death	Whole-bowel irrigation Supportive care
Oleander ( <i>Nerium oleander</i> )	Nausea, vomiting, diarrhea, abdominal pain, confusion, cardiac dysrhythmias	GI decontamination with activated charcoal Monitoring of potassium level Antidysrhythmics Digoxin-specific Fab antibody for dysrhythmias
Poison hemlock ( <i>Conium maculatum</i> )	Tachycardia, tremors, diaphoresis, mydriasis, muscle weakness, seizures, neuromuscular blockade	GI decontamination with activated charcoal Supportive care
Water hemlock ( <i>Cicuta maculata</i> )	Nausea, vomiting, abdominal pain, delirium, seizures, death	GI decontamination Supportive care
Yew ( <i>Taxus</i> species)	Common: nausea, vomiting, abdominal pain Rare: seizures, cardiac dysrhythmias, coma	GI decontamination with activated charcoal Consider whole-bowel irrigation Supportive care

**TABLE 128-2** Symptoms and Treatment of Severely Poisonous Plant Ingestions

Plant	Symptoms	Treatment
Ackee ( <i>Blighia sapida</i> )	Hypoglycemia	Glucose
Aloe ( <i>Aloe barbadensis</i> )	Abdominal pain, diarrhea, red urine, nephritis	Supportive care
Azalea ( <i>Rhododendron</i> species)	Usually minor symptoms Severe intoxication: salivation, lacrimation, brady-cardia, hypotension, progressive paralysis	GI decontamination with activated charcoal Atropine for symptomatic bradycardia Fluids or vasopressors for hypotension
Cactus	Pain and irritation from embedded spines	Removal of spines Rubber cement peel
<i>Caladium</i> species	Usually minor symptoms Severe intoxication: burning and irritation of oral mucosa, swelling, drooling, dysphagia, respiratory compromise	Ingest cold milk or ice cream for oral burning bradycardia Fluids or vasopressors for hypotension
Colchicum (autumn crocus, meadow saffron, glory lily)	Delayed and severe gastroenteritis → severe multi-system organ failure	GI decontamination with activated charcoal Aggressive fluid resuscitation
Dumbcane ( <i>Dieffenbachia amoena</i> )	Usually minor symptoms Severe intoxication: burning and irritation of oral mucosa, swelling, drooling, dysphagia, respiratory compromise	Ingest cold milk or ice cream for oral burning Analgesics Consider steroids if severe symptoms
Fava beans ( <i>Vicia faba</i> )	In persons with glucose-6-phosphate dehydrogenase deficiency: GI upset, fever, headache, hemolytic anemia, hemoglobinuria, jaundice	Treatment varies depending on degree of hemolysis seen

Henbane ( <i>Hyoscyamus niger</i> )	Anticholinergic symptoms: hallucinations, mydriasis, tachycardia, agitation, seizures, coma	Consider physostigmine in severe cases
Jimsonweed ( <i>Datura</i> species)	Anticholinergic symptoms: hallucinations, mydriasis, tachycardia, agitation, seizures, coma	GI decontamination with activated charcoal Consider whole-bowel irrigation Supportive care
Lily of the valley ( <i>Convallaria majalis</i> )	Nausea, vomiting, diarrhea, abdominal pain, confusion, cardiac arrhythmias	GI decontamination with activated charcoal Monitoring of potassium level Antiarrhythmics Digoxin-specific Fab antibody for arrhythmias
Monkshood ( <i>Aconitum</i> species)	Bradycardia, heart block, torsades de pointes, ventricular fibrillation	GI decontamination with activated charcoal Supportive care
Nettle (stinging nettle, bull nettle) ( <i>Urtica</i> species)	Localized burning	Symptomatic care
Nightshade, common or woody ( <i>Solanum</i> species)	Nausea, vomiting, diarrhea, abdominal pain; With larger doses: delirium, hallucinations, coma	Supportive care
Nightshade, deadly ( <i>Atropa belladonna</i> )	Anticholinergic symptoms: hallucinations, mydriasis, tachycardia, agitation, seizures, coma	GI decontamination with activated charcoal Supportive care
Peach, apricot, pear, crab apple, yam bean, and hydrangea (pits or seeds)	Acute cyanide toxicity if large amounts are ingested: diaphoresis, nausea, vomiting, abdominal pain, lethargy	GI decontamination with activated charcoal Whole-bowel irrigation Cyanide antidote therapy
Pepper ( <i>Capsicum</i> species)	Irritation and pain on contact	Copious irrigation with water Milk or ice cream for oral irritation Analgesics
<i>Philodendron</i> species	Usually minor symptoms Severe intoxication: burning and irritation of oral mucosa, swelling, drooling, dysphagia, respiratory compromise	Cold milk or ice cream for oral irritation Analgesics Consider steroids
Pokeweed ( <i>Phytolacca americana</i> )	Mucosal irritation, abdominal pain, nausea, vomiting, profuse diarrhea Severe intoxication: coma, death	GI decontamination with activated charcoal Supportive care
Potato, eggplant (raw) ( <i>Solanum</i> species)	Nausea, vomiting, diarrhea, abdominal pain With larger doses: delirium, hallucinations, coma	Supportive care

Pothos (devil's ivy, <i>Epipremnum</i> species)	Usually minor symptoms Severe intoxication: burning and irritation of oral mucosa, swelling, drooling, dysphagia, respiratory compromise	Cold milk or ice cream for oral irritation Analgesics Consider steroids
Yellow sage ( <i>Lantana camara</i> )	Dilated pupils, vomiting, diarrhea, weakness, coma	GI decontamination with activated charcoal Fluids
<i>Toxicodendron</i> species (poison ivy, oak, and sumac)	Dermatitis	Skin protection Antipruritic and topical therapies Systemic steroids for facial, genital, or widespread involvement
Holly ( <i>Ilex</i> species)	Gastroenteritis Can be fatal if significant ingestion	GI decontamination with activated charcoal Supportive care
Poinsettia ( <i>Euphorbia pulcherrima</i> )	Occasional local irritation	–
American mistletoe ( <i>Phoradendron flavescens</i> )	Gastroenteritis	GI decontamination with activated charcoal Supportive care
Easter lily ( <i>Lilium longiflorum</i> )	Toxicity has not been reported in humans	No treatment necessary

**TABLE 128-3** Symptoms and Treatment of Common Poisonous Plant Ingestions or Exposures

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 214, “Mushroom Poisoning,” by Anne F. Brayer, Sandra M. Schneider, Arif Alper Cevik; and Chapter 215, “Poisonous Plants,” by Mark A. Hostetler and Sandra M. Schneider.

## **SECTION 13 Endocrine Emergencies**

# **CHAPTER 129**

## **Diabetic Emergencies**

Michael P. Kefer

### **HYPOGLYCEMIA**

Hypoglycemia is usually a complication of treatment of diabetics with insulin or sulfonylureas (chlorpropamide, glyburide, glipizide). Hypoglycemia is an unusual reaction from treatment with the glitizones (rosiglitazone, pioglitazone), glinides (repaglinide, nateglinide), alpha-glucosidase inhibitors (acarbose, miglitol), or the biguanide metformin. Patients with diabetes, alcoholism, sepsis, adrenal insufficiency, hypothyroidism, or malnutrition are at risk for severe hypoglycemia.

#### **Clinical Features**

Typical symptoms of hypoglycemia include sweating, shakiness, anxiety, nausea, dizziness, confusion, slurred speech, blurred vision, headache, lethargy, and coma. Focal neurologic findings may include cranial nerve palsies, hemiplegia, seizures, and decerebrate posturing.

#### **Diagnosis and Differential**

A blood glucose level alone does not define hypoglycemia. The diagnosis is based on the glucose level in conjunction with typical symptoms that resolve with treatment. Hypoglycemia can easily be misdiagnosed as a primary neurologic or psychiatric condition ([Table 129-1](#)).

Stroke
Transient ischemic attack
Seizure disorder
Traumatic head injury
Brain tumor
Narcolepsy
Multiple sclerosis
Psychosis
Sympathomimetic drug ingestion
Hysteria
Altered sleep patterns and nightmares
Depression

**TABLE 129-1** Differential Diagnosis of Hypoglycemia

## Emergency Department Care and Disposition

1. Treat hypoglycemic patients with altered mental status with **50% dextrose** 50 mL IV. A continuous infusion of **10% dextrose** solution may be required to maintain the blood glucose above 100 milligrams/ dL. Provide a carbohydrate meal if the patient can tolerate PO.
2. If there is no IV access, administer **glucagon** 1 milligram IM or SC.
3. Refractory hypoglycemia secondary to the sulfonylureas may respond to **octreotide** 50 to 100 micrograms SC. A continuous infusion of 125 micrograms/h may be required.
4. Monitor for rebound hypoglycemia by determining blood glucose every 30 min initially.
5. Disposition is determined by the patient's response to treatment, cause of hypoglycemia, comorbid conditions, and social situation. Most insulin reactions respond rapidly. Patients can be discharged with instructions to continue oral intake of carbohydrates and closely monitor their finger stick glucose. Patients with hypoglycemia due to the sulfonylureas or long acting insulins should be admitted due to the risk of recurrence from these agents. See [Table 129-2](#) for admission guidelines.

Inpatient care for type 2 diabetes mellitus is generally appropriate for the following clinical situations:
Life-threatening metabolic decompensation such as diabetic ketoacidosis or hyperglycemic hyperosmolar nonketotic state
Severe chronic complications of diabetes, acute comorbidities, or inadequate social situation
Hyperglycemia (> 400 milligrams/dL) associated with severe volume depletion or refractory to appropriate interventions
Hypoglycemia with neuroglycopenia (altered level of consciousness, altered behavior, coma, seizure) that does not rapidly resolve with correction of hypoglycemia
Hypoglycemia resulting from long-acting oral hypoglycemic agents
Fever without an obvious source in patients with poorly controlled diabetes



**TABLE 129-2** Disposition/Guidelines for Hospital Admission

## DIABETIC KETOACIDOSIS

Diabetic ketoacidosis (DKA) results from a relative insulin deficiency and counter-regulatory hormone excess causing hyperglycemia and ketonemia. [Table 129-3](#) lists causes.

Omission or reduced daily insulin injections
Dislodgement/occlusion of insulin pump catheter
Infection
Pregnancy
Hyperthyroidism
Substance abuse (cocaine)
Medications: steroids, thiazides, antipsychotics, sympathomimetics
Heat-related illness
Cerebrovascular accident
GI hemorrhage
Myocardial infarction
Pulmonary embolism
Pancreatitis
Major trauma
Surgery

**TABLE 129-3** Important Causes of Diabetic Ketoacidosis

### Clinical Features

Hyperglycemia causes an osmotic diuresis with dehydration, hypotension, and tachycardia. Ketonemia causes an acidosis with myocardial depression, vasodilation, and compensatory Kussmaul respiration. Nausea, vomiting, and abdominal pain are common. The absence of fever does not exclude infection. Acetone, formed from oxidation of ketone bodies, causes the characteristic fruity odor of the patient's breath.

### Diagnosis and Differential

Diagnosis of DKA is based on clinical presentation and laboratory values of a glucose > 250 milligrams/dL, bicarbonate <15 mEq/L, pH <7.3, and a moderate ketonemia.

An anion gap metabolic acidosis results from formation of ketone bodies. In DKA, the conversion of acetoacetate to  $\beta$ -hydroxybutyrate is favored. Therefore, the patient may have low levels of acetoacetate and high levels of  $\beta$ -hydroxybutyrate. If the nitroprusside test is used to detect serum or urine ketones, it may be falsely low or negative as it only detects acetoacetate, not  $\beta$ -hydroxybutyrate.

Osmotic diuresis results in loss of sodium, chloride, calcium, phosphorus, and magnesium, but initial serum levels may be normal from hemoconcentration. Serum and urine glucose and ketones are elevated. Pseudohyponatremia is common: for each 100 milligrams/dL increase in blood glucose, the sodium decreases by 1.6 mEq/L. Some recommend this sodium correction factor be 2.4, especially if the glucose is  $> 400$  milligrams/ dL. Serum potassium may be low from osmotic diuresis and vomiting, normal, or high from acidosis. In acidosis, potassium is driven extracellularly. Therefore, the acidotic patient with normal or low potassium has marked depletion of total body potassium.

Laboratory investigation includes serum pH, glucose, electrolytes, blood urea nitrogen, creatinine, phosphorus, magnesium, complete blood count, urinalysis (and pregnancy if indicated), electrocardiogram, and chest radiograph to assess the severity of DKA and search for the underlying cause. When ordering serum pH, consider that venous pH correlates closely with arterial pH and avoid the pain and risk associated with arterial puncture.

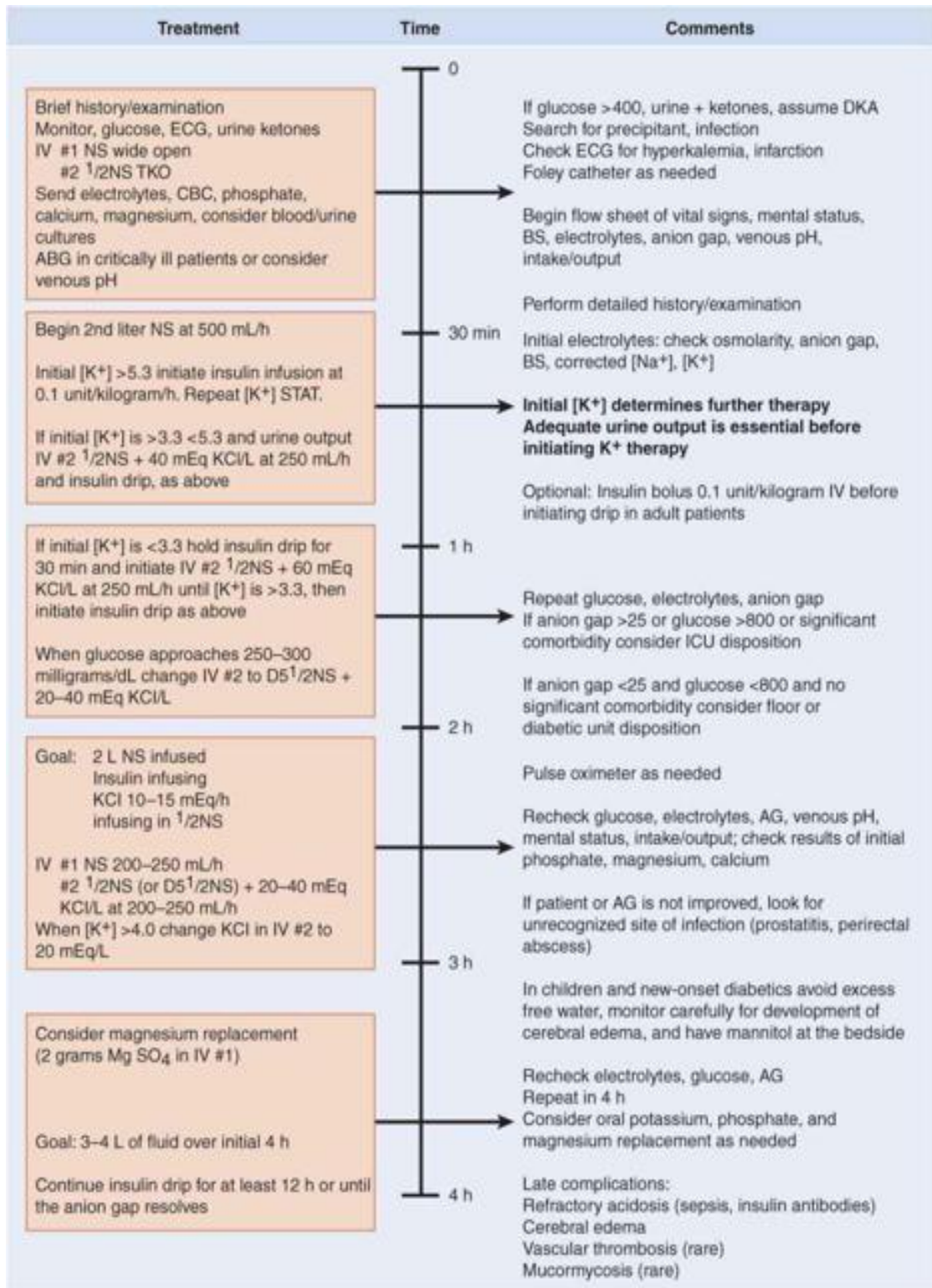
The differential diagnosis includes other causes of an anion gap metabolic acidosis ([Table 129-4](#)). Hypoglycemia and hyperosmolar hyperglycemic state should also be considered.

Alcoholic ketoacidosis
Starvation ketoacidosis
Renal failure
Lactic acidosis
Ingestions
Salicylates
Ethylene glycol
Methanol

**TABLE 129-4** Differential Diagnosis for Diabetic Ketoacidosis

## Emergency Department Care and Disposition

1. The goal of treatment is to correct the volume deficit, acid-base imbalance and electrolyte abnormalities, administer insulin, and treat the underlying cause ([Fig. 129-1](#)). See [Table 129-2](#) for admission guidelines.



**FIGURE 129-1** Timeline for the typical adult patient with suspected diabetic ketoacidosis.

*Key:* ABG = arterial blood gas; AG = anion gap; BS = blood sugar; CBC = complete blood count; chemistries = sodium, potassium, chloride, CO<sub>2</sub> content, blood urea nitrogen, creatinine; DKA = diabetic ketoacidosis; NS = normal saline; STAT = immediately; TKO = IV infusion just to keep the venous access patent.

2. Bicarbonate therapy remains controversial as to when the benefits of correcting the effects of acidosis (vasodilation, depression of cardiac contractility and respiration, CNS depression, severe hyperkalemia) outweigh the risk of treatment (paradoxical CSF acidosis, hypokalemia, impaired oxyhemoglobin dissociation, rebound alkalosis, sodium overload). It may be of benefit in patients with severe acidosis (pH < 6.9). It is indicated for the treatment of life-threatening hyperkalemia.
3. Monitor serum glucose, anion gap, potassium, and bicarbonate hourly until recovery is well established: glucose < 200 milligrams/dL, bicarbonate > 17, and pH > 7.3.
4. Cerebral edema is a complication of treatment that occurs predominately in children. Young age and new-onset diabetes are risk factors. It tends to develop 4 to 12 hours into treatment and typically manifests as deterioration in neurologic status. Begin treatment with **mannitol** 1 gram/kilogram before obtaining the diagnostic CT scan. Gradual correction of sodium, glucose, and hypovolemia may lessen the risk.

## **HYPEROSMOLAR HYPERGLYCEMIC STATE**

Hyperosmolar hyperglycemic state (HHS) is distinguished from DKA by the absence of significant ketosis. It is a common presentation of new-onset type 2 diabetes. Causes are similar to DKA ([Table 129-3](#)). Osmotic diuresis causes hypovolemia and electrolyte losses.

### **Clinical Features**

The typical patient is elderly with type 2 diabetes and presents with complaints of weakness or mental status changes, and has preexisting renal or heart disease. Because metabolic changes progress slowly, symptoms often signal advanced HHS.

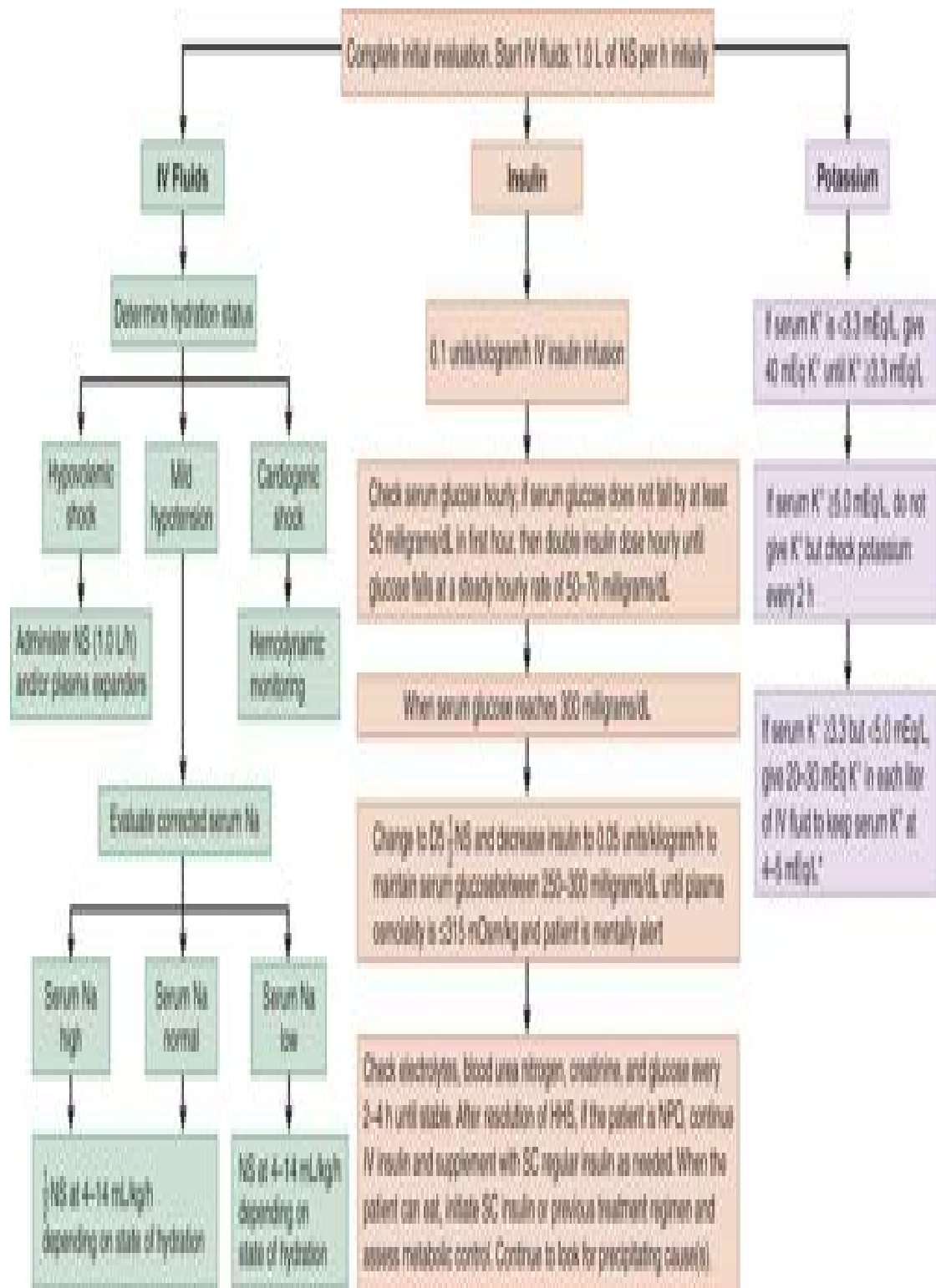
Physical examination reveals signs of dehydration with orthostasis, dry skin and mucous membranes, and altered mental status. Focal deficits and seizures may occur. Kussmaul respiration and the smell of acetone on the breath are absent as significant ketosis does not occur.

## **Diagnosis and Differential**

Defining laboratory parameters are serum glucose above 600 milligrams/ dL, serum osmolality > 320 mOsm/kg, pH > 7.3, and negative or mildly elevated ketones.

## **Emergency Department Care and Disposition**

Treatment consists of correcting the volume deficit, electrolyte imbalance, and hyperosmolality, and treating the underlying cause ([Fig. 129-2](#)). See [Table 129-2](#) for admission guidelines.



**FIGURE 129-2** Protocol for the management of severely ill adult patients with hyperosmolar hyperglycemic state (HHS). Diagnostic criteria for HHS: blood glucose

> 600 milligrams/dL, arterial pH > 7.3, bicarbonate > 15 mEq/L, mild ketonuria or ketonemia, and effective serum osmolality > 320 mOsm/kg of water. \*Concentrations of  $K^+ \geq 20$  mEq/L should be administered via central line. History and physical examination, appropriate ancillary studies. D5<sup>1/2</sup>/NS = 5% dextrose in half normal saline; HHS = hyperosmolar hyperglycemic state; NS = normal saline.

## DIABETIC FOOT ULCERS

These are classified and managed as nonlimb-threatening, limb-threatening, or life-threatening ([Table 129-5](#)). Antibiotic treatment is tailored accordingly ([Table 129-6](#)).

Extent of Infection	Nonlimb-Threatening Infection	Life or Limb-Threatening Infection
<b>Clinical Features</b>	<ul style="list-style-type: none"> <li>&lt;2 cm cellulitis</li> <li>Superficial ulcer</li> <li>Mild infection</li> <li>No systemic toxicity</li> <li>No ischemic changes</li> <li>No bone or joint involvement</li> <li>Does not probe to bone</li> </ul>	<ul style="list-style-type: none"> <li>&gt;2 cm cellulitis</li> <li>Deep ulcer</li> <li>Odor or purulent drainage from wound</li> <li>Fever</li> <li>Ischemic changes</li> <li>Lymphangitis, edema</li> <li>Sepsis or septic shock</li> </ul>
<b>Diagnostic Procedures</b>	<ul style="list-style-type: none"> <li>Cultures from base of ulcer</li> <li>Diagnostic imaging</li> <li>Serologic testing</li> <li>CBC with differential</li> <li>ESR</li> <li>Comprehensive metabolic panel</li> </ul>	<ul style="list-style-type: none"> <li>Cultures from base of ulcer</li> <li>Diagnostic imaging</li> <li>Serologic testing</li> <li>CBC with differential</li> <li>ESR</li> <li>Comprehensive metabolic panel</li> <li>Blood cultures</li> </ul>
<b>Treatment</b>	<ul style="list-style-type: none"> <li>Outpatient management with follow-up in 24 to 72 h</li> <li>Debridement of all necrotic tissue and callus</li> <li>Wound care/dressing</li> <li>Empiric antibiotic coverage, modified by culture findings</li> <li>Appropriate off-loading of weightbearing</li> <li>Wound care continued with packs, dressings, and debridement as needed</li> <li>Hospital admission if infection progresses or systemic signs or symptoms develop</li> <li>Refer to podiatrist for follow-up care, special shoes, and prostheses as needed</li> </ul>	<ul style="list-style-type: none"> <li>Hospital admission</li> <li>Surgical debridement with resection of all necrotic bone and soft tissue</li> <li>Exploration and drainage of deep abscess</li> <li>Empiric antibiotic coverage, modified by culture findings</li> <li>Surgical resection of osteomyelitis</li> <li>Wound care continued with packs, dressings, debridement as needed</li> <li>Foot-sparing reconstructive procedures</li> <li>Refer to podiatrist for follow-up care, special shoes, and prostheses as needed</li> </ul>

**TABLE 129-5** Clinical Practice Pathways for Diabetic Foot Ulcer and Infection

<p>Nonlimb-threatening (May give initial dose as IV equivalent) Cephalexin, 500 milligrams PO once a day, 10-day course (cefazolin, 1 gram IV) or Clindamycin, 300 milligrams PO once a day, 10-day course (clindamycin, 900 milligrams IV) or Dicloxacillin, 500 milligrams PO once a day, 10-day course or Amoxicillin-clavulanate, 875 milligrams PO twice a day, 10-day course</p> <p>Limb-threatening <i>Oral regimen</i>*: Fluoroquinolone and clindamycin <i>IV regimens</i>: Ampicillin-sulbactam, 3 grams IV every 6 h or Ticarcillin-clavulanate, 3.1 grams IV every 8 h or</p> <p>Second-generation cephalosporin (cefoxitin, cefotetan), 1 to 2 grams IV every 12 h or Clindamycin, 900 milligrams IV every 6 h, plus either ciprofloxacin, 400 milligrams IV every 12 h, or ceftriaxone, 1 gram IV every 12 h</p> <p>Life-threatening <i>IV regimens</i>: Imipenem-cilastatin, 1 gram every 6 h or Ampicillin-sulbactam, 3 grams every 8 h, plus antipseudomonal aminoglycoside tobramycin, 5 to 7 milligrams/kilogram once a day or Vancomycin, 1 gram every 12 h, plus metronidazole, 500 milligrams every 6 h, plus aztreonam, 2 grams every 8 h</p>
--

Note: Adjust all dosages for renal/hepatic function and monitor blood levels where appropriate.

\*This approach is acceptable under special circumstances with close follow-up.

### TABLE 129-6 Antimicrobial Therapy in Infected Diabetes-Related Lower Extremity Ulcers

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 218, “Type 1 Diabetes Mellitus,” by Nikhil Goyal and Adam B. Schlichting; Chapter 219, “Type 2 Diabetes Mellitus,” by Mohammad Jalili; Chapter 220, “Diabetic Ketoacidosis,” by Michael E. Chansky and Cary Lubkin; and Chapter 222, “Hyperosmolar Hyperglycemic State,” by Charles S. Graffeo.



# CHAPTER 130

## Alcoholic Ketoacidosis

Michael P. Kefer

Alcoholic ketoacidosis (AKA) results from heavy alcohol intake, either acute or chronic, and lack of food intake. Glycogen stores are depleted. Alcohol consumption along with the body's fat metabolism generates ketoacids, with a resultant anion gap metabolic acidosis.

### CLINICAL FEATURES

The patient typically presents with nausea, vomiting, orthostasis, and abdominal pain 24 to 72 hours after the last alcohol intake. On examination, the patient appears acutely ill and dehydrated. Abdominal tenderness is diffuse and nonspecific or is the result of other causes related to alcohol, such as gastritis, hepatitis, or pancreatitis.

### DIAGNOSIS AND DIFFERENTIAL

Laboratory investigation reveals an anion gap metabolic acidosis. However, the serum pH may vary as these patients often have mixed acid-base disorders such as a metabolic acidosis from AKA and a metabolic alkalosis from vomiting and dehydration. Blood glucose is low to mildly elevated. The alcohol level is usually low or 0 as symptoms limit intake. Serum ketones, acetoacetate, and  $\beta$ -hydroxybutyrate, are elevated. If the nitroprusside test is used to measure serum and urine ketones, acetoacetate is detected but not  $\beta$ -hydroxybutyrate. The redox state may be such that most or all acetoacetate is reduced to  $\beta$ -hydroxybutyrate, which may result in a false negative or falsely low result.

Diagnostic criteria for AKA are listed in [Table 130-1](#). The differential diagnosis of an anion gap metabolic acidosis is listed in [Table 130-2](#).

Low, normal, or slightly elevated serum glucose  
Binge drinking ending in nausea, vomiting, and decreased intake  
Wide anion gap metabolic acidosis  
Positive serum ketones\*  
Wide anion gap metabolic acidosis without alternate explanation

\*The absence of ketones in the serum based on the nitroprusside test does not exclude the diagnosis.

**TABLE 130-1** Diagnostic Criteria for Alcoholic Ketoacidosis\*

- Methanol
- Uremia
- Diabetic ketoacidosis
- Paraldehyde
- Iron, isoniazid, inhalants
- Lactic acidosis
- Ethanol, ethylene glycol
- Salicylates

**TABLE 130-2** The Differential Diagnosis of an Anion Gap Metabolic Acidosis Is Recalled by the Acronym MUDPILES

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Administer **D5NS**. The isotonic crystalloid solution restores intravascular volume. The glucose stimulates the patient's endogenous insulin release, which inhibits ketosis. Unlike treatment for DKA, insulin administration is not necessary.
2. **Thiamine** 100 milligrams IV before glucose administration may prevent precipitation of Wernicke disease.
3. Supplement other electrolytes and vitamins as warranted.
4. Continue treatment until the acidosis clears, which is usually within 12 to 24 hours.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 221, "Alcoholic Ketoacidosis," by William A. Woods and Debra G. Perina.

# CHAPTER 131

## Thyroid Disease Emergencies

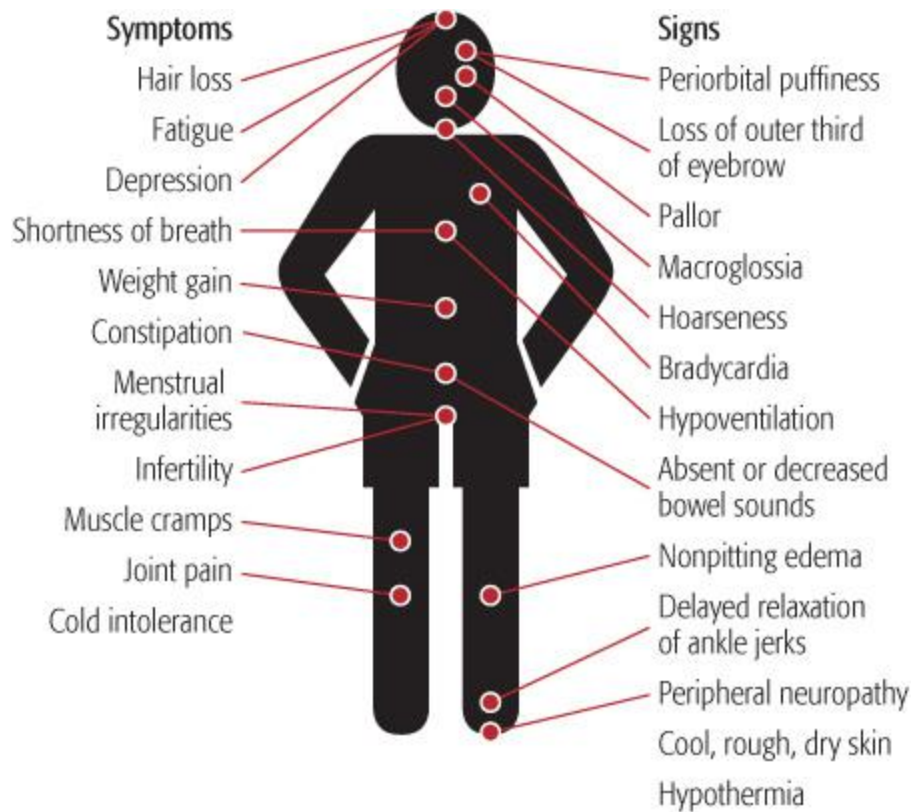
Katrina A. Leone

### **HYPOTHYROIDISM AND MYXEDEMA COMA**

Hypothyroidism may be caused by multiple factors. Myxedema coma is a rare, life-threatening expression of hypothyroidism. It may be precipitated by infection, cold exposure, trauma, medications, or myocardial infarction. It classically occurs during the winter months in elderly women with undiagnosed or undertreated hypothyroidism.

#### **Clinical Features**

The presentation of hypothyroidism is summarized in [Figure 131-1](#). Patients with myxedema coma have hypothyroidism and present with hypothermia, bradycardia, hypotension, and altered mental status. Respiratory failure is common and a difficult airway may be encountered due to macroglossia and oropharyngeal edema. Laboratory abnormalities include hypoglycemia and hyponatremia.



**FIGURE 131-1.** Symptoms and signs of hypothyroidism.

## **Diagnosis and Differential**

Myxedema coma is a clinical diagnosis. Send confirmatory thyroid studies, but do not delay treatment for test results. Low free thyroxine ( $FT_4$ ) and triiodothyronine ( $FT_3$ ), and elevated thyroid stimulating hormone (TSH) are diagnostic. The differential diagnosis for myxedema coma includes sepsis, adrenal crisis, congestive heart failure, hypoglycemia, stroke, hypothermia, and drug overdose.

## **Emergency Department Care and Disposition**

1. Provide supportive care with airway stabilization, mechanical ventilation, and cardiac monitoring ([Table 131-1](#)). Treat hypotension with fluid resuscitation. Vasopressors may be ineffective until thyroid hormone replacement is initiated. Passively rewarm hypothermic patients.

<p>Supportive care</p> <ul style="list-style-type: none"> <li>• Airway, breathing, and circulation support: ensure airway control, oxygen, IV access, and cardiac monitor</li> <li>• IV therapy: dextrose for hypoglycemia; water restriction for hyponatremia</li> <li>• Vasopressors: if indicated (ineffective without thyroid hormone replacement)</li> <li>• Hypothermia: treated with passive rewarming using blanket</li> <li>• Steroids: hydrocortisone (because of increased metabolic stress; 100 to 200 milligrams IV)</li> </ul>
<p>Thyroid replacement therapy (see discussion Thyroid Replacement in text)</p> <ul style="list-style-type: none"> <li>• IV T<sub>4</sub> (levothyroxine) at 4 micrograms/kilogram, followed in 24 h by 100 micrograms IV, then 50 micrograms IV until oral medication is tolerated. (Switch to 50 to 200 micrograms/d PO when patient is ambulatory.)</li> </ul> <p><i>or</i></p> <ul style="list-style-type: none"> <li>• IV T<sub>3</sub> (liothyronine or triiodothyronine) for myxedema coma at 20 micrograms followed by 10 micrograms IV every 8 h until the patient is conscious (given because of the risk of decreased T<sub>3</sub> generation from T<sub>4</sub> in severely hypothyroid patients). Start with no more than 10 micrograms IV for the elderly or those with coronary artery disease.</li> <li>• <i>Either T<sub>4</sub> or T<sub>3</sub> can be used, but in severe myxedema coma, T<sub>3</sub> should be considered (either combined with T<sub>4</sub> or used alone) but with cautious use on patients with myocardial compromise.</i></li> </ul>
<p>Identify and treat precipitating factors</p> <ul style="list-style-type: none"> <li>• Infections.</li> <li>• Sedatives.</li> <li>• Anesthetic agents (eg, etomidate).</li> <li>• Cold exposure.</li> <li>• Trauma.</li> <li>• Myocardial infarction or congestive heart failure.</li> <li>• Cerebrovascular accident.</li> <li>• GI hemorrhage.</li> <li>• Contributing metabolic conditions include hypoxia, hypercapnia, hyponatremia, and hypoglycemia.</li> </ul>

**TABLE 131-1** Treatment for Myxedema Coma Supportive Care

2. Seek out and treat precipitating causes. Administer **hydrocortisone** 100 milligrams IV for suspected adrenal insufficiency. Correct hypoglycemia.
3. Administer **levothyroxine** (T<sub>4</sub>) 4 micrograms/kilogram IV by slow infusion. Add **liothyronine** (T<sub>3</sub>) 20 micrograms IV for severe myxedema coma. Use liothyronine with caution in the elderly and patients with cardiovascular disease.
4. Patients should be admitted to a monitored or ICU setting.

# THYROTOXICOSIS AND THYROID STORM

Hyperthyroidism is defined as excess circulating thyroid hormone due to thyroid gland hyperactivity. Thyrotoxicosis is a general term for excess circulating thyroid hormone from any cause. Thyroid storm is an acute, life-threatening state of thyrotoxicosis that is most common in patients with antecedent Graves disease.

## Clinical Features

The clinical features of thyrotoxicosis are manifestations of enhanced adrenergic activity. Thyroid storm presents as fever, central nervous system and cardiovascular dysfunction, in addition to thyrotoxicosis signs and symptoms ([Table 131-2](#)).

<b>Constitutional</b>
Fever
<b>Central Nervous System</b>
Agitation
Confusion
Delirium
Coma
Seizure
<b>Cardiovascular</b>
Tachycardia
Arrhythmia
Congestive heart failure

**TABLE 131-2** Presenting Signs and Symptoms of Thyroid Storm

## Diagnosis and Differential

An elevated FT<sub>4</sub> or FT<sub>3</sub> level and a suppressed TSH level are diagnostic of thyrotoxicosis. Thyroid storm is a clinical diagnosis; laboratory tests cannot distinguish it from thyrotoxicosis. The differential diagnosis for thyroid

storm includes sepsis, heat stroke, delirium tremens, neuroleptic malignant syndrome, serotonin syndrome, pheochromocytoma, and sympathomimetic drug overdose.

## Emergency Department Care and Disposition

1. Provide supportive care, including airway stabilization, supplemental oxygen, and cardiac monitoring. Cooling is indicated for hyperthermia.
2. See [Table 131-3](#) for pharmacologic therapy for thyroid storm.

1. Inhibit thyroid hormone release with thionamides (PTU is the preferred over methimazole, also blocks conversion of T <sub>4</sub> to T <sub>3</sub> )	<b>Propylthiouracil</b> (PTU) 600 to 1000 milligrams PO loading dose followed by 200 to 250 milligrams PO every 4 h or <b>Methimazole</b> 40 milligrams PO loading dose followed by 25 milligrams PO every 4 h
2. Inhibit new thyroid hormone production (Give at least 1 h after step 1)	<b>Lugol Solution</b> 8 to 10 drops PO every 6 to 8 h or <b>Potassium Iodide</b> (SSKI) 5 drops PO every 6 h or <b>Iopanoic Acid</b> 1 gram IV every 8 h or <b>Lithium Carbonate*</b> 300 milligrams PO every 6 h
3. Block peripheral thyroid hormone effects	<b>Propranolol</b> 1 to 2 milligrams IV every 10 to 15 min or <b>Reserpine</b> <sup>†</sup> 1 milligram IM test dose followed by 2.5 to 5 milligrams IM every 4 to 6 h or <b>Guanethidine</b> <sup>b</sup> 30 to 40 milligrams PO every 6 h
4. Prevent conversion of T <sub>4</sub> to T <sub>3</sub>	<b>Hydrocortisone</b> 100 milligrams IV every 8 h or <b>Dexamethasone</b> 2 milligrams IV every 6 h

\*Lithium preferred over iodine for iodine or thionamide allergies and in the setting of iodine- or amiodarone-induced thyrotoxicosis.

<sup>†</sup>Use if β-blocker is contraindicated and congestive heart failure or hypotension is not present.

**TABLE 131-3** Treatment of Thyroid Storm

3. Evaluate and treat precipitating causes. Search for an infectious source in febrile patients and administer appropriate antibiotics for identified infections.
4. Admit patients to a monitored or ICU setting.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 223, “Thyroid Disorders: Hypothyroidism and Myxedema Crisis,” and Chapter 224, “Thyroid Disorders: Hyperthyroidism and Thyroid Storm,” by Alzamani Mohammad Idrose.



## **CHAPTER 132**

# **Adrenal Insufficiency and Adrenal Crisis**

Michael P. Kefer

Adrenal insufficiency results when the physiologic demand for glucocorticoids and mineralocorticoids exceeds the supply from the adrenal cortex. The pituitary secretes adrenocorticotropin hormone (ACTH) and associated melanocyte stimulating hormone (MSH). ACTH stimulates the adrenal cortex to secrete cortisol. Cortisol has negative feedback on the pituitary to inhibit secretion of ACTH and MSH.

## **CLINICAL FEATURES**

Primary adrenal insufficiency is due to adrenal gland failure, resulting in cortisol and aldosterone deficiency. Manifestations include weakness, dehydration, hypotension, anorexia, nausea, vomiting, weight loss, and abdominal pain. Hyperpigmentation of skin and mucous membrane occurs as a result of uninhibited MSH secretion in conjunction with ACTH.

Secondary adrenal insufficiency results from inadequate secretion of ACTH with resultant cortisol deficiency. Aldosterone levels are not significantly affected because of regulation through the renin-angiotensin system. Therefore, hyperpigmentation and hyperkalemia are not seen.

Adrenal crisis is the acute, life-threatening form of adrenal insufficiency. Clinical features are as described above, but to the extreme and accompanied by shock and altered mental status.

Congenital adrenal hyperplasia (CAH) results from an enzyme deficiency in cortisol production. Patients typically present in the first month of life with nonspecific symptoms of lethargy, vomiting, poor feeding, and poor weight gain. Examination reveals dehydration, hyperpigmentation, and, in females, clitoromegaly.

## **DIAGNOSIS AND DIFFERENTIAL**

The diagnosis of adrenal insufficiency may be difficult because the clinical features are nonspecific. The diagnosis of primary adrenal insufficiency and CAH is based on the presence of the clinical features and lab findings of hyponatremia, hyperkalemia, hypoglycemia, anemia, metabolic acidosis, and prerenal azotemia. All patients with adrenal insufficiency have low plasma cortisol levels. Secondary adrenal insufficiency will not have findings of hyperkalemia as there is not a deficiency of aldosterone.

The most common cause of acute adrenal insufficiency is adrenal suppression from prolonged steroid use with either abrupt steroid withdrawal or exposure to increased physiologic stress such as injury, illness, or surgery. It may take up to 1 year for the hypothalamic-pituitary-adrenal axis to recover following prolonged suppression with steroid treatment. [Tables 132-1](#) and [132-2](#) list causes of primary and secondary adrenal insufficiency, respectively.

<b>Primary Adrenal Insufficiency (disorders in the adrenal gland)</b>	<b>Examples</b>
Autoimmune	Isolated adrenal insufficiency or associated with polyglandular insufficiencies (polyglandular autoimmune syndrome types I or II)
Adrenal hemorrhage or thrombosis	Necrosis caused by meningococcal sepsis Coagulation disorders Overwhelming sepsis (Waterhouse-Friderichsen syndrome)
Drugs	Adrenolytic agents Metyrapone Aminoglutethimide Mitotane Ketoconazole
Infections involving adrenal glands	Tuberculosis Fungal, bacterial sepsis Acquired immunodeficiency syndrome involving adrenal glands
Infiltrative disorders involving adrenal glands	Sarcoidosis Hemochromatosis Amyloidosis Lymphoma Metastatic cancer
Surgery	Bilateral adrenalectomy
Hereditary	Adrenal hypoplasia Congenital adrenal hyperplasia Adrenoleukodystrophy Familial glucocorticoid deficiency
Idiopathic	—

**TABLE 132-1** Causes of Primary Adrenal Insufficiency

Secondary Adrenal Insufficiency (hypothalamic-pituitary dysfunction)	Examples
Sudden cessation of prolonged glucocorticoid therapy	Chronic use of steroid inhibits ACTH production
Pituitary necrosis or bleeding	Postpartum pituitary necrosis (Sheehan syndrome)
Exogenous glucocorticoid administration	Causes decreased production of ACTH at pituitary
Brain tumors	Pituitary tumor Hypothalamic tumor Local invasion (craniopharyngioma)
Pituitary irradiation Pituitary surgery Head trauma involving the pituitary gland	Disrupts corticotropin-releasing hormone and ACTH production capacity in hypothalamic-pituitary axis
Infiltrative disorders of the pituitary or hypothalamus	Sarcoidosis Hemosiderosis Hemochromatosis Histiocytosis X Metastatic cancer Lymphoma
Infectious diseases involving organs away from adrenal	Tuberculosis Meningitis Fungus Human immunodeficiency virus

Key: ACTH = adrenocorticotropic hormone.

**TABLE 132-2** Causes of Secondary Adrenal Insufficiency

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

[Table 132-3](#) outlines treatment for acute adrenal insufficiency in adults. Neonates with CAH are treated with NS 20 mL/kg for hypovolemia, **hydrocortisone** 25 milligrams IV/IO for glucocorticoid and mineralocorticoid deficiency, and D10 5 mL/kg for hypoglycemia. The dose of hydrocortisone for toddlers and school-age children is 50 milligrams and 100 milligrams for adolescents.

Begin therapy immediately in any suspected case of adrenal crisis (prognosis is related to rapidity of treatment delivery).
<b>Administer IV fluids</b> 5% dextrose in normal saline is the fluid of choice to correct both hypoglycemia and hyponatremia.
<b>Steroids</b> Hydrocortisone (100-milligram bolus) is the drug of choice for cases of adrenal crisis or insufficiency (provides both glucocorticoid and mineralocorticoid effects). <i>or</i> Dexamethasone, 4-milligram bolus (for accuracy of rapid adrenocorticotrophic hormone stimulation test results).
<b>Vasopressors</b> Administered after steroid therapy in patients unresponsive to fluid resuscitation [norepinephrine, dopamine, or phenylephrine (Neo-Synephrine®) preferred].
<b>Supplementation</b> Patients may require lifelong glucocorticoids ± mineralocorticoid supplementation.
<b>Maintenance</b> Increased maintenance doses of chronic steroids are required during periods of stress (eg, illness, surgery, trauma, etc) to satisfy increased physiologic need for cortisol.

**TABLE 132-3** Treatment Guide for Adrenal Insufficiency

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 137, “Hypoglycemia and Metabolic Emergencies in Infants and Children,” by Nadeemuddin Qureshi, Mohammed Al-Mogbil, and Osama Y. Kentab; and Chapter 225, “Adrenal Insufficiency and Adrenal Crisis,” by Alzamani Mohammed Idrose.

# **SECTION 14 Hematologic and Oncologic Emergencies**

## **CHAPTER 133**

# **Evaluation of Anemia and the Bleeding Patient**

Daniel A. Handel

Anemia may be chronic and unrelated to the chief complaint, or it may result from acute blood loss as seen in trauma, gastrointestinal bleeding, or other acute hemorrhage. Underlying bleeding disorders must be suspected in patients presenting with spontaneous bleeding from multiple sites, bleeding from nontraumatized sites, delayed bleeding several hours after injury, or bleeding into deep tissues or joints.

## **CLINICAL FEATURES**

The rate of the development of the anemia, the extent of the anemia, the age of the patient, and the ability of the cardiovascular system to compensate for the decreased oxygen-carrying capacity determine the severity of the patient's symptoms and clinical presentation. Patients may complain of weakness, fatigue, palpitations, orthostatic symptoms, and dyspnea with minimal exertion. Patients may have pale conjunctiva, skin, and nail beds. Tachycardia, hyperdynamic precordium, and systolic murmurs may be present. Tachypnea at rest and hypotension are late signs. Use of ethanol, prescription drugs, and recreational drugs may alter the patient's ability to compensate for the anemia.

Patients with bleeding may or may not have an obvious site of hemorrhage. A history of excessive or abnormal bleeding in the patient and other family members may indicate an underlying bleeding disorder. Historical data about liver disease and drug use, such as use of ethanol, aspirin, nonsteroidal anti-inflammatory drugs, warfarin, and antibiotics should be gathered. Mucocutaneous bleeding (including petechiae, ecchymoses, purpura, and epistaxis), gastrointestinal, genitourinary, or heavy menstrual bleeding are features associated with qualitative or quantitative platelet disorders. Patients with deficiencies of coagulation factors often present with delayed bleeding, hemarthroses, or bleeding into potential spaces between fascial planes and into the retroperitoneum. Patients with combination abnormalities of platelets and coagulation factors, such as disseminated intravascular coagulation, present with both mucocutaneous and potential space bleeding.

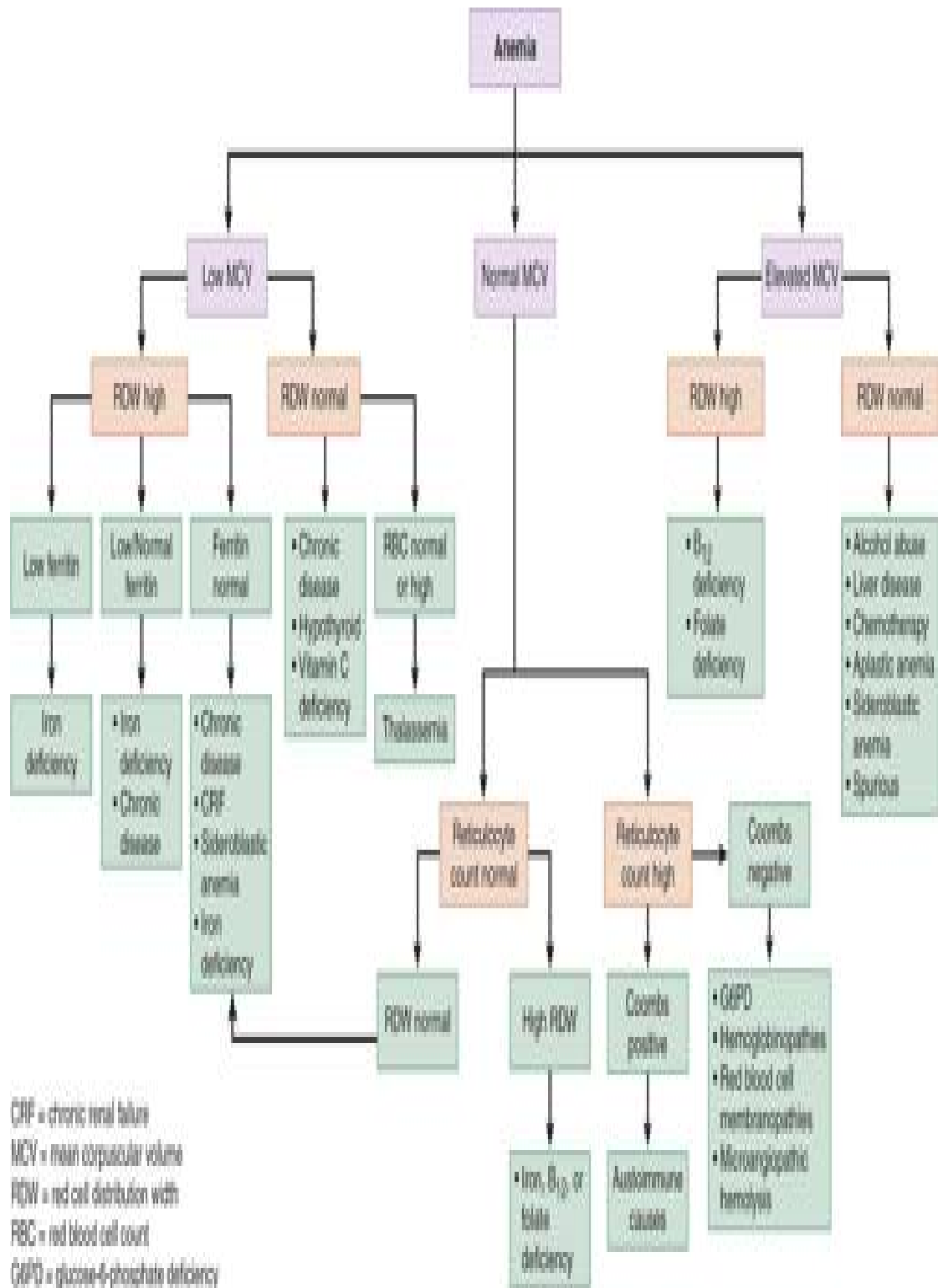
Acquired hemolytic anemia may be autoimmune or drug-induced. Microangiopathic syndromes include thrombotic thrombocytopenic purpura (TTP) and hemolytic-uremic syndrome (HUS). The classic pentad of TTP is CNS

abnormalities, renal disease, fever, microangiopathic hemolytic anemia, and thrombocytopenia. HUS consists of acute nephropathy or renal failure, microangiopathic hemolytic anemia, and thrombocytopenia. Macrovascular hemolysis can be caused by prosthetic heart valves.

## **DIAGNOSIS AND DIFFERENTIAL**

A decreased red blood count, hemoglobin, and hematocrit are diagnostic for anemia. The initial evaluation of newly diagnosed anemia should include a complete blood count, review of RBC indices, reticulocyte count, stool hemocult examination, urine pregnancy test, and examination of the peripheral blood smear. The mean corpuscular volume (MCV) and reticulocyte count can assist in classifying the anemia and can aid in differential diagnosis ([Fig. 133-1](#)).





**FIGURE 133-1.** Flowchart for the evaluation of anemia.

Laboratory studies used to diagnose bleeding disorders can be divided into the following 3 categories: (a) those that test the initial formation of a platelet plug (primary hemostasis); (b) those that assess the formation of crosslinked fibrin (secondary hemostasis); and (c) those that test the fibrinolytic system, which is responsible for limiting the size of the fibrin clots formed (see [Table 133-1](#)). A complete blood count with platelet count, prothrombin time, and partial thromboplastin time are the initial studies needed in patients with suspected bleeding disorders. If there is a suspicion for a hemolytic anemia, further studies may be warranted ([Table 133-2](#)).

Test	Reference Value	Component Measured	Clinical Correlations/Comments
Fibrin degradation product (FDP) and D-dimer levels	FDP: variable depending on specific test, typically <2.5 to 10 micrograms/ml D-Dimer: variable depending on specific test, typically <250 to 500 ng/ml	FDP test: measures breakdown products from fibrinogen and fibrin monomer D-Dimer test: measures breakdown products of cross-linked fibrin	Levels are elevated in diffuse intravascular coagulation, venous thrombosis, pulmonary embolus, and liver disease, and during pregnancy
Factor level assays	60% to 150% of reference value (0.80 to 1.50 units/ml)	Measures the percent activity of a specified factor compared to normal	To identify specific deficiencies and direct therapeutic management
Protein C level	Variable Typically 60% to 150% of reference value	Level of protein C in the blood	Vitamin K dependent Increases with age Values higher in males than females Deficiency associated with thromboembolism in people <50 y of age
Protein S level	Variable Typically 60% to 150% of reference value	Level of protein S in the blood	Vitamin K dependent Increases with age Values higher in males than females Deficiency associated with thromboembolism in people <50 y of age
Factor V Leiden (FVL)	Variable	Screening test looks for activated protein C resistance and confirmatory test analyzes DNA sequence of Factor V gene Screening assay uses activated partial thromboplastin time with and without added activated protein C	FVL not inactivated by activated protein C Heterozygotes have 7x and homozygotes have a 20x increased lifetime risk of venous thrombosis Mutation associated with thromboembolism in people <50 y of age

Antithrombin level	Variable depending on specific test Typically 20 to 45 milligrams/dL	Measures level of antithrombin in the blood	Not vitamin K dependent; patients with deficiency require higher dosages of heparin for anticoagulation therapy Deficiency associated with thromboembolism in people <50 y of age
Antiphospholipid antibodies	IGG <25 GPL units/mL and IGM <11 MPL units/mL	Tests for antibodies that bind to phospholipids  Lupus anticoagulant  Anticardiolipin antibody	Lupus anticoagulant: elevated in systemic lupus erythematosus (SLE) and other autoimmune diseases Anticardiolipin antibody: elevated in SLE, other autoimmune diseases, syphilis, and Behcet syndrome Increased risk of spontaneous abortions, fetal loss, and fetal growth retardation
Anti-Factor Xa activity	Therapeutic: 0.7 to 1.1 units/mL Prophylactic: 0.2 to 0.3 units/mL	Inhibition of Factor Xa activity	Used to monitor low-molecular-weight heparin therapy May be elevated in renal dysfunction
Platelet function assay	88 to 190 s Variable	Tests for platelet adhesion and aggregation	Affected by uremia, anemia, thrombocytopenia, antiplatelet medications, and von Willebrand disease
Peripheral blood smear	Qualitative and quantitative based on visualization	Estimates quantity and appearance of platelets, white blood cells, and red blood cells	Allows identification of clumped platelets, abnormal cells interfering with coagulation (leukemia), operator dependent
Dilute Russell viper venom time	23 to 27 s	Venom directly activates Factor X and converts prothrombin to thrombin when phospholipid and Factor V are present	Prolonged in the presence of antiphospholipid antibodies
Inhibitor screens	Variable	Verifies the presence or absence of antibodies directed against one or more of the coagulation factors	Specific inhibitors: directed against one coagulation factor, most commonly against Factor VIII Nonspecific inhibitors: directed against more than one coagulation factor, example is lupus-type anticoagulant
PIVKA II (proteins induced by vitamin K absence or antagonism) test	Variable	Measures nonfunctional precursors of vitamin K-dependent coagulation factors (II, VII, IX, X)	Increased in vitamin K-deficient states, such as hemorrhagic disease of the newborn, and can differentiate it from nonaccidental trauma Increased in overdoses of warfarin or cholestatic liver diseases that can respond to vitamin K therapy

Key: GPL = 1 microgram of affinity-purified IGG anticardiolipin antibody from an original index serum; MPL = 1 microgram of affinity-purified IGM anticardiolipin antibody from an original index serum.

**TABLE 133-1** Additional Hemostatic Tests

Purpose	Test	Finding
Confirm anemia/blood loss	Hemoglobin	Decreased
	Hematocrit	Decreased
Confirm compensatory RBC production	Reticulocyte count	Increased
Confirm hemolysis	Peripheral smear	Schistocytes—intravascular hemolysis, RBCs fragmented by shear mechanism Spherocytes—extravascular hemolysis, RBC phagocytosis by macrophages
Confirm hemolysis	Lactate dehydrogenase	Increased, released by RBCs
	Potassium	Increased, released by RBCs
Confirm hemolysis	Haptoglobin	Decreased, indicative of intravascular hemolysis
	Free hemoglobin	Increased, indicative of intravascular hemolysis
	Hemoglobinuria	Present
Confirm hemoglobin breakdown	Total bilirubin	Increased
	Indirect bilirubin	Increased (hepatic conjugation of bilirubin overwhelmed)
	Urinary urobilinogen	Increased

Key: RBC = red blood cell.

**TABLE 133-2** Basic Tests and Findings in the Evaluation of Hemolytic Anemia

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Type and crossmatch blood in patients with anemia and ongoing blood loss so that it is available for transfusion, if necessary.
2. Consider immediate transfusion of **packed RBCs** in symptomatic patients who are hemodynamically unstable and have evidence of tissue hypoxia.
3. Admit patients with anemia and ongoing blood loss for further evaluation and treatment. Admit patients with chronic anemia or newly diagnosed anemia with unclear etiology if they are hemodynamically unstable, hypoxic, acidotic, or demonstrate cardiac ischemia.
4. Consider **hematology consultation** to assist in evaluation of those patients with anemia of unclear etiology, anemic patients with concomitant abnormalities of platelets and white blood cell counts, and patients with suspected bleeding disorders.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 226, “Anemia,” by Robin R. Hemphill; Chapter 227, “Tests of

Hemostasis,” by Stephen John Cico and Robin R. Hemphill; and Chapter 232, “Acquired Hemolytic Anemia,” by Patricia Chu Klap and Robin R. Hemphill.

# CHAPTER 134

## Acquired Bleeding Disorders

Aaron Barksdale

Platelet abnormalities, drugs, systemic illness, and endogenous anticoagulants can cause acquired bleeding disorders.

### ACQUIRED PLATELET DEFECTS

#### Clinical Features

Patients with thrombocytopenia or dysfunctional platelets commonly present with petechiae, most evident in the lower extremities. Patients may also exhibit gingival bleeding, epistaxis, purpura, hematuria, and menorrhagia. Splenomegaly may be noted in patients experiencing platelet sequestration.

#### Diagnosis and Differential

Acquired platelet abnormalities include quantitative (thrombocytopenia) and qualitative (dysfunctional) defects. *Quantitative platelet disorders* include those caused by decreased platelet production (marrow infiltration, aplastic anemia, drugs, viral infections, and chronic alcohol use), and increased platelet destruction (idiopathic thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome, disseminated intravascular coagulation (DIC), viral infections, drugs and HELLP syndrome). Other causes include acute hemorrhage, hemodialysis and splenic sequestration. *Qualitative platelet disorders* are commonly associated with uremia, liver disease, DIC, drugs (aspirin, NSAIDs, clopidogrel), myeloproliferative disorders, and antiplatelet antibodies. Initial laboratory testing should include a CBC with peripheral smear.

## Emergency Department Care and Disposition

1. Consult with a hematologist as subtleties in diagnosis and treatment exist. For example, some conditions may be worsened by platelet transfusion (DIC and TTP).
2. Consider **platelet transfusion** in patients with a **platelet count <10 000/mm<sup>3</sup> or active bleeding with platelets <50 000/mm<sup>3</sup>**.
3. Treatment and disposition of patients with ITP varies with age, severity, and symptoms. In general, patients with a platelet count > 50 000/mm<sup>3</sup> require no treatment. Patients with a platelet count <20 000 to 30 000/mm<sup>3</sup> and patients with a platelet count of <50 000/mm<sup>3</sup> with bleeding typically require treatment. Treatment options include corticosteroids, such as **prednisone** 60 to 100 milligrams PO daily. **Immunoglobulin** 1gram/kilogram daily IV is usually reserved for patients with very low platelet counts and bleeding.

## BLEEDING IN LIVER DISEASE

Patients with liver disease have an increased risk of bleeding for multiple reasons including decreased synthesis of vitamin K–dependent coagulation factors (II, VII, IX, and X), thrombocytopenia, and increased fibrinolysis.

## Emergency Department Care and Disposition

1. Administer **vitamin K** 10 milligrams PO/IV to patients with significant liver disease who are actively bleeding.
2. Use **fresh-frozen plasma** 15 mL/kg IV to temporarily replace coagulation factors in patients with active bleeding and coagulopathy, or prior to any invasive procedure.
3. In patients with active bleeding: replace fibrinogen with **cryoprecipitate** **1 unit/5 kilograms IV** if fibrinogen levels <100 milligrams/dL, consider



**platelet transfusion** if thrombocytopenic; **desmopressin** 0.3 microgram/kilogram SC or IV may shorten bleeding times in some patients.

## **BLEEDING IN RENAL DISEASE**

A variety of hemostatic defects are associated with renal disease including platelet dysfunction due to uremic toxins, deficiency of coagulation factors, and thrombocytopenia.

### **Emergency Department Care and Disposition**

1. Treat acute bleeding with **transfusion of packed red blood cells**.
2. Hemodialysis improves platelet function transiently for 1 to 2 days.
3. **Desmopressin** 0.3 microgram/kilogram SC or IV shortens bleeding time in most patients.
4. **Conjugated estrogen 0.6 milligram/kilogram IV daily for 5 days** improves both the bleeding time and clinical bleeding in 80% of patients.
5. Transfuse **platelets** and **cryoprecipitate** for life-threatening bleeding only. Use in conjunction with red blood cells, desmopressin and estrogens.

## **DISSEMINATED COAGULATION**

## **INTRAVASCULAR**

### **Clinical Features**

The clinical features of DIC are a result of simultaneous hemorrhage and thrombosis and vary according to the underlying illness. Bleeding typically predominates and can range from petechiae to diffuse hemorrhaging from GI/GU tracts, surgical wounds, and venipuncture sites. Patients may present with mental status changes, focal ischemia, oliguria, renal cortical necrosis, acute lung injury, and may develop multisystem organ failure.

## Diagnosis and Differential

The diagnosis is based on history, clinical presentation, and associated laboratory abnormalities. Clinical conditions associated with DIC are listed in [Table 134-1](#). Laboratory abnormalities are listed in [Table 134-2](#). The differential diagnosis includes primary fibrinolysis and coagulopathy due to severe liver disease.

Clinical Setting	Comments
Infection Bacterial Viral Fungal	Probably the most common cause of DIC; 10% to 20% of patients with gram-negative sepsis have DIC; endotoxins stimulate monocytes and endothelial cells to express tissue factor; Rocky Mountain spotted fever causes direct endothelial damage; DIC more likely to develop in asplenic patients or cirrhosis; septic patients are more likely to have bleeding than thrombosis.
Carcinoma Adenocarcinoma Lymphoma	Malignant cells may cause endothelial damage and allow the expression of tissue factor as well as other procoagulant materials; most adenocarcinomas tend to have thrombosis (Trousseau syndrome), except prostate cancer tends to have more bleeding; DIC is often chronic and compensated.
Acute leukemia	DIC most common with promyelocytic leukemia; blast cells release procoagulant enzymes, there is excessive release at time of cell lysis (chemotherapy); more likely to have bleeding than thrombosis.
Trauma	DIC especially with brain injury, crush injury, burns, hypothermia, hyperthermia, rhabdomyolysis, fat embolism, hypoxia.

Organ injury Liver disease Pancreatitis	May have chronic compensated DIC; acute DIC may occur in the setting of acute hepatic failure, tissue factor is released from the injured hepatocytes. Pancreatitis can activate the coagulation cascade.
Pregnancy	Placental abruption, amniotic fluid embolus, septic abortion, intrauterine fetal death (can be chronic DIC); can have DIC in hemolysis-elevated liver enzymes-low platelets (HELLP) syndrome.
Vascular disease	Large aortic aneurysms (chronic DIC can become acute at time of surgery), giant hemangiomas, vasculitis, multiple telangiectasias.
Envenomation	DIC can develop with bites of rattlesnakes and other vipers; the venom damages the endothelial cells; bleeding is not as serious as expected from laboratory values.
Acute lung injury or adult respiratory distress syndrome	Microthrombi are deposited in the small pulmonary vessels, the pulmonary capillary endothelium is damaged; 20% of patients with ARDS develop DIC and 20% of patients with DIC develop ARDS.
Transfusion reactions, such as acute hemolytic reaction	DIC with severe bleeding, shock, and acute renal failure.

Key: ARDS = acute respiratory distress syndrome.

**TABLE 134-1** Common Conditions Associated with Disseminated Intravascular Coagulation (DIC)

Studies	Result
<b>Most Useful</b>	
Prothrombin time	Prolonged
Platelet count	Usually low, or dropping
Fibrinogen level	Usually low (fibrinogen is an acute phase reactant, so may actually start out elevated) fibrinogen level < 100 milligrams/dL correlates with severe DIC
<b>Helpful</b>	
Activated partial thromboplastin time	Usually prolonged
Thrombin clotting time	Prolonged (not sensitive)
Fragmented red blood cells	Should be present (not specific)
Fibrin degradation products and D-dimer*	Elevated
<i>Specific factor assays</i>	Extrinsic pathway factors are most affected (VII, X, V, and II)
Factor II, V, VII, <sup>†</sup> X	Low
Factor VIII (acute phase reactant)	Low, normal, high
Factor IX	Low (decreases later than other factors)

\*Levels may be chronically elevated in patients with liver or renal disease.

<sup>†</sup>Factor VII is usually low early because it has the shortest half-life.

**TABLE 134-2** Laboratory Abnormalities Characteristic of Disseminated Intravascular Coagulation (DIC)

## Emergency Department Care and Disposition

1. Treat the underlying illness and provide hemodynamic support (IV fluid resuscitation, red blood cells, and inotropic agents).
2. Administer **cryoprecipitate** to patients with hypofibrinogenemia and active bleeding, with the goal of raising fibrinogen levels to 100 to 150 milligrams/dL.
3. Transfuse **platelets** if counts <20 000/mm<sup>3</sup> or <50 000/mm<sup>3</sup> with active bleeding.
4. Transfuse **fresh frozen plasma** if bleeding is present.
5. Administer **vitamin K**.
6. The role of heparin remains unclear.

# BLEEDING DUE TO CIRCULATING ANTICOAGULANTS

Patients with acquired factor VIII inhibitors (hemophilia A) present with spontaneous bruising and hematomas, whereas those with antiphospholipid antibodies (lupus anticoagulant and anticardiolipin antibodies) typically present with thrombosis. Consult with a hematologist for management of acute bleeding in patients with factor VIII inhibitor. Treatment options include **factor VIII**, **factor XI complex**, **recombinant factor VIIa**, **desmopressin**, and **plasmapheresis**.

## CLOTTING DISORDERS

Hypercoagulable states may be either inherited (activated protein C resistance/ factor V Leiden mutation, protein C deficiency, protein S deficiency, antithrombin deficiency or acquired hypercysteinemia) or acquired (antiphospholipid syndrome, pregnancy, oral contraceptives/hormone therapy, malignancy, heparin-induced thrombocytopenia (HIT), and hyperviscosity syndrome). Suspect a hypercoagulable state in young patients without other risk factors and patients with a family history of thromboembolism, recurrent thromboembolic events, or thromboembolic events in unusual sites. Consider HIT in patients with a drop in platelet count of  $> 50\%$  5 to 15 days after starting heparin. Diagnostic testing for the specific hypercoagulable state is usually beyond the scope of the ED. Initial management of thrombosis consists of low molecular weight heparin or unfractionated heparin. ([Chapter 25](#)) An exception to this is patients with HIT in whom heparin must be stopped and another anticoagulant, such as lepirudin, argatroban, danaparoid, initiated in consultation with a hematologist.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 228, “Acquired Bleeding Disorders,” by Sally A.

Santen and Robin R. Hemphill and Chapter 229, "Clotting Disorders," by  
Jessie G. Nelson and Robin R. Hemphill.

## **CHAPTER 135**

# **Hemophilias and von Willebrand Disease**

Daniel A. Handel

## **HEMOPHILIAS**

The most common hemophilias are caused by genetic deficiencies of factor VIII (hemophilia A) or factor IX (hemophilia B).

### **Clinical Features**

Bleeding complications depend on severity of the disease. Patients with severe disease (factor VIII or factor IX activity level <1%) experience spontaneous bleeds and difficult to control bleeding after trauma. Patients with moderate disease (1% to 5% factor activity level) may bleed spontaneously but more commonly bleed after trauma. Patients with mild disease (5% to 40% factor activity level) usually only bleed after trauma. Easy bruising, recurrent hemarthrosis, and muscle hematomas are the most common clinical manifestations. Mucocutaneous, abdominal, retroperitoneal, GU and CNS bleeding also occur. Neck hematomas may obstruct the airway. Unless there is another underlying disease, most patients with hemophilia do not have problems with minor cuts or abrasions.

### **Diagnosis and Differential**

Clinically, it is impossible to differentiate between hemophilias A and B. Laboratory testing in patients with hemophilia most often shows a normal prothrombin time (PT), prolonged partial thromboplastin time (PTT), and a normal bleeding time. However, if greater than 30% to 40% of factor activity is present, the PTT may be normal. Specific factor assays may be

used to differentiate between the types of hemophilia. Ten percent to 25% of patients with hemophilia A and 1% to 2% of patients with hemophilia B will develop an inhibitor, which is an antibody against the deficient factor. The quantity of inhibitor is measured by the Bethesda inhibitor assay (BIA) and is reported in BIA units. The presence of an inhibitor makes treatment more difficult.

## Emergency Department Care and Disposition

The mainstay of therapy is early factor replacement. Replacement factor products are listed in [Table 135-1](#).

Hemophilia Type	Available Products*	Comments
Hemophilia A	<i>Human plasma-derived Factor VIII products</i>	All products have a low risk of HIV and hepatitis transmission.
	Koate-HP® (gel chromatography, solvent, and detergent treated)	
	Humate-P® (heat treated)	Both products have reduced amounts of von Willebrand factor.
	Alphanate® (solvent and detergent treated)	
	<i>Human plasma-derived Factor VIII with immunoaffinity purification</i>	
	Hemofil-M® (monoclonal antibody purification, solvent and detergent treated)	Highly purified source of Factor VIII.
	Monodate-P® (monoclonal antibody purification, heat treated)	All products have low to no risk of HIV and hepatitis transmission.
	<i>Recombinant Factor VIII products</i>	
	Recombinate® (recombinant DNA product)	
	Helixate® (recombinant DNA product)	
	Advate® (recombinant DNA product)	
	Kogenate-FS® (recombinant DNA product)	
Xyntha® (recombinant DNA product)	No evidence that human viral infection occurs.	
<i>Porcine Factor VIII product cryoprecipitate fractionation, screened for porcine viruses</i>		
Hyate:C®		



Hemophilia B	<i>Factor IX complex products</i>	Thrombotic risk.
	Koyne-80 <sup>®</sup> Factor IX complex (heat treated)	Low risk of HIV and hepatitis transmission.
	Proplex-T <sup>®</sup> Factor IX complex (heat treated)	
	Profilnine-SD <sup>®</sup> (solvent and detergent treated)	
	<i>Activated Factor IX complex products</i>	Low risk of HIV and hepatitis transmission.
	Autoplex-T <sup>®</sup> (heat treated)	
	<i>Purified Factor IX products</i>	Low to no transmissions of HIV or hepatitis reported.
	AlphaNine-SD <sup>®</sup> (purified, solvent, and detergent treated)	
	Mononine <sup>®</sup> (monoclonal antibody purification, ultrafiltration)	
	<i>Recombinant Factor IX products</i>	No known risk of HIV or hepatitis transmission.
	BeneFIX <sup>®</sup> (recombinant DNA product)	Product of choice for patients with significant inhibitor activity.

Key: HIV = human immunodeficiency virus.

\*Commercial trade names provided for ease of specific identification.

**TABLE 135-1** Replacement Factor Products for Hemophilia Treatment

- Determine the type of hemophilia and the presence or absence of inhibitor.** See [Table 135-2](#) for factor replacement guidelines. Factor replacement may need to be instituted before definitive imaging after head trauma and other life-threatening injuries. If an inhibitor is present, use therapy as outlined in [Table 135-3](#).

Site	Desired Initial Factor Level (%)	Hemophilia A Initial Dose (units/kilogram)	Hemophilia B Initial Dose (units/kilogram)	Details
Skin (deep laceration)	=	=	=	Abrasions and superficial lacerations usually do not require factor replacement. Treat with pressure and topical thrombin.
Deep muscle	40 to 80	20 to 40	40 to 60	Admit, monitor vital blood loss, watch for compartment syndrome. Duration of replacement: 1 to 5 days.
Joint (hemarthrosis)	30 to 50	15 to 25	30 to 40	Orthopedic consult may be required for splinting, physical therapy, and follow-up. Duration of replacement: 1 to 3 days.
Epistaxis	40 to 50	20 to 25	80 to 100	Local measures should be used. Replacement is given until bleeding resolves.
Oral mucosa	50	25	50	Local measures and antifibrinolytic therapy will decrease need for additional factor replacement (see Special Considerations: Oral and Mucosal Bleeding).
Hematuria	50	25	50	Common and typically not severe. Rest and hydration are important.
GI bleeding	100	50	100	Consultation with a gastroenterologist for endoscopy to locate potential lesion is appropriate.
Central nervous system	100	50	100	Treat before CT. Early neurosurgical consultation. Lumbar puncture requires factor replacement.

**TABLE 135-2** Initial Factor Replacement Guidelines in Severe Hemophilia

Type of Product	Hemophilia A Dose	Hemophilia B Dose	Comments
Factor VI concentrates	5000 to 10,000 units bolus followed by a continuous infusion	Not applicable	Not available in the U.S. Preferred if patient has Factor VI deficiency
Prothrombin complex concentrates; contains factors I, VI, IX, and X; small amount of activation occurs during processing Octaplex <sup>®</sup> Konyne-80 <sup>®</sup> Proplex-7 <sup>®</sup>	75 to 100 units/kg	Approximately 75 units/kg	Thrombotic risk Risk of contamination with other coagulation factors
Anti-inhibitor coagulant complex; contains factors I, VI, IX, and X, with Factor VII mainly in an activated form FEIBA-VH <sup>®</sup> Autoplex <sup>™</sup>	50 to 100 units/kg	50 to 100 units/kg	Thrombotic risk Used in patients with high BIA unit titer and high BIA units antibody response
Recombinant Factor VIIa NovSeven <sup>®</sup>	90 to 120 micrograms/kg	90 to 120 micrograms/kg	No risk of viral transmission
Highly purified Factor IX concentrates Aphaline SD <sup>®</sup> Monocine <sup>®</sup>	Not applicable	Variable	-
Recombinant Factor IX products BeneFIX <sup>®</sup>	Not applicable	Variable	Product of choice for hemophilia B patients with significant inhibitor activity

Key: BIA = Bethesda inhibitor assay

<sup>®</sup>Commercial trade names provided for ease of specific identification.

**TABLE 135-3** Replacement Therapy for Hemophilia A and B in Patients with Inhibitors

- Determine the desired factor activity level.** Factor activity level determines how much factor replacement is required. Calculate the amount of factor needed using the patient's weight and the desired increase in factor:

**Factor VIII required** = (Target factor – Base line factor)/2 × weight (kg)

**Factor IX required** = (Target factor – Base line factor)/2 × weight (kg)

For severe hemophilia, assume 0% intrinsic activity.

- Treat patients with undiagnosed bleeding disorders with fresh frozen plasma (**FFP**). FFP contains 1 unit of factor VIII/mL. **Specific factor assays** should guide further therapy.
- Treat minor bleeding in patients with mild hemophilia A with **desmopressin (DDAVP)** 0.3 microgram/kilogram IV over 15 to 30 min or DDAVP 150 micrograms single spray in each nostril (for children > 5 years, **DDAVP** 150 micrograms single spray in 1 nostril). Very mild mucosal bleeding can also be treated with antifibrinolytic agents, such as **ε-aminocaproic acid (EACA)** 75 to 100 milligrams/kilogram (up to 6 grams) IV/PO every 6 hours or **tranexamic acid** 10 to 25 milligrams/kilogram IV every 6 hours.
- Indications for admission include bleeding involving the head, neck, pharynx, retropharynx, or retroperitoneum, potential compartment syndrome, inability to control pain; and treatment requiring multiple factor replacement.

## **VON WILLEBRAND DISEASE**

### **Clinical Features**

Von Willebrand disease (vWD) is a group of disorders caused by a defect or deficiency of von Willebrand factor (vWF). vWF is a cofactor for platelet adhesion and a carrier protein for factor VIII in the plasma. Type 1 vWD is most common; patients have 20% to 50% of normal vWF levels and usually manifest with skin and mucosal bleeding symptoms. Patients with Type 2 vWF have abnormal and dysfunctional vWF. Patients with Type 3 vWF have complete vWF deficiency and have clinical presentations similar to hemophilia. Patients with mild vWF are frequently unaware of the disease until bleeding occurs after a traumatic episode or surgical procedure.

## **Diagnosis and Differential**

The PT and PTT are usually normal. The bleeding time is prolonged and vWF activity is low. Variability in vWF levels can make it difficult to distinguish vWD from hemophilia A.

## **Emergency Department Care and Disposition**

The treatment of vWD depends on the type of disease and the severity of bleeding. Indications for admission are similar to patients with hemophilia.

1. The mainstay of treatment for bleeding in type 1 vWD is **desmopressin (DDAVP)** 0.3 microgram/kilogram (up to 20 micrograms) SC/IV every 12 to 24 hours for up to 4 doses or **DDAVP** 150 micrograms single spray in each nostril (for children > 5 years, **DDAVP** 150 micrograms single spray in 1 nostril). If there is no response to DDAVP, administer factor VIII concentrate or cryoprecipitate as described for type 2 and 3 vWD.
2. **Factor VIII concentrate** containing vWF is used to treat bleeding in patients with type 2 or 3 disease ([Table 135-1](#)).
3. **Cryoprecipitate** 10 bags IV every 12 to 24 hours can be used for type 1 refractory to DDAVP or type 2 or 3 vWD. There is, however, a risk of viral transmission.
4. **Platelet transfusions** may benefit patients with type 3 vWD who do not respond to vWF-containing plasma products.

5. **Oral contraceptives** may help increase vWF levels and limit menstrual bleeding in women with vWD and menorrhagia.
6. Patients who have sustained dental injuries or who require dental procedures may need  **$\epsilon$ -aminocaproic acid (EACA)** 75 to 100 milligrams/kilogram (up to 6 grams) PO every 6 hours or **tranexamic acid** mouth-wash for 5 to 10 days.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 230, “Hemophilias and von Willebrand Disease,” by William Manson, Robin R. Hemphill, and Christine L. Kempton.

## CHAPTER 136

# Sickle Cell Disease and Other Hereditary Hemolytic Anemias

Jason B. Hack

The inherited hemoglobin disorders stem from abnormal Hb structure (eg, sickle cell disease [SCD] or abnormal Hb production (eg, the thalassemias). Anemia occurs when destruction of red blood cells outstrips production. Most patients are aware of their dyshemoglobinemia status.

## SICKLE CELL DISEASE

### Clinical Features

The most common complaints in SCD (homozygous (HbSS) or sickle trait (heterozygous [HbAS]) patients are pain, localized or generalized, weakness, or infectious complaints. Physical examination findings commonly include pale complexion, venous stasis changes, jaundice, hepatosplenomegaly, anemic cardiac flow murmurs, cardiomegaly, and high output CHF.

Painful vaso-occlusive crisis in the musculoskeletal system or diffusely in the abdomen are the most common presentation to the ED. Crisis occur when sickled RBCs mechanically obstruct blood flow, causing ischemia, organ damage, and infarcts. Crisis-causing stresses include fever or infection (especially encapsulated organisms *Haemophilus influenza* or *Pneumococcus*), cold exposure or high altitude, dehydration or overexertion, medication non-compliance, or drug use.

Acute symptomatic anemia results from splenic sequestration or bone marrow failure (aplastic crisis) and presents with weakness, dyspnea, CHF, or shock.

Life or limb-threatening events seen in SCD patients include acute chest syndrome (vaso-occlusive pulmonary insult), stroke, renal infarct,

mesenteric infarcts, sepsis, osteomyelitis, pneumonia, or priapism.

## Diagnosis and Differential

The degree of illness guides the evaluation of an acute crisis. Although workups should be individualized, the more common protean complaints (pain, weakness, fever) must include a search for a cryptic inciting event. See [Table 136-1](#).

History	Duration and location of pain History of fever History of focal swelling or redness Precipitating factors for acute episode Medications taken for pain relief
Physical examination	Assess degree of pain Inspect sites of pain, looking for swelling, warmth, redness General: respiratory distress, pallor, hydration, jaundice, rash Vital signs: especially temperature, pulse oximetry Respiratory: chest wall, lung sounds Heart: cardiomegaly and systolic murmur common with chronic anemia Abdomen: tenderness, organomegaly
Ancillary tests	If moderate to severe pain, focal pathology is present, or pain is atypical for acute episode Complete blood count, leukocyte differential, reticulocyte count, urinalysis Chest radiograph, if signs of lower respiratory tract pathology Blood cultures and additional blood tests: as indicated by clinical condition
General management	Bed rest, provide warmth, and a calm, relaxing atmosphere Distractions where appropriate—television, music, etc. Oral fluids: typically about 3 L per day IV fluids to correct dehydration or if reluctant to drink or vomiting is present Oxygen: not routinely required, unless hypoxemia is present Encourage deep breathing, incentive spirometry



Pain management	<p>Use analgesics appropriate to degree of pain</p> <p>Acetaminophen for mild pain</p> <p>NSAID for mild to moderate pain (avoid if renal insufficiency is present)</p> <p>Opioids for moderate to severe pain, typical initial doses include:</p> <p>Morphine, 0.3 milligram/kilogram PO or 0.1–0.15 milligram/kilogram IV</p> <p>Hydromorphone, 0.06–0.08 milligram/kilogram PO or 0.015–0.020 milligram/kilogram IV</p> <p>Reassess response in 15–30 min, may repeat with one-fourth to one-half initial dose, consider patient's known effective dose</p>
Disposition and follow-up	<p>Consider admission to the hospital if:</p> <p>Acute chest syndrome is suspected</p> <p>Sepsis, osteomyelitis, or other serious infection is suspected</p> <p>White blood cell count is <math>&gt;30,000/\text{mm}^3</math></p> <p>Platelet count is <math>&lt;100,000/\text{mm}^3</math></p> <p>Pain is not under control after two to three rounds of analgesics in the ED</p> <p>Consider discharge if:</p> <p>Pain is under control and patient can take oral fluids and medications</p> <p>Ensure appropriate oral analgesics are available</p> <p>Provide home care instructions</p> <p>Ensure resource for follow-up</p>

**TABLE 136-1** Guidelines for the Assessment and Management of Acute Vaso-Occlusive Crisis

Acute worsening of baseline anemia may suggest increased splenic sequestration if the reticulocyte count is elevated, or bone marrow failure if the reticulocyte count is depressed. Leukocytosis or left shift with increased bands suggests infection. Perform a pregnancy test on women. Assessment of electrolytes allows evaluation of dehydration and renal function. Liver function tests and lipase may help evaluate abdominal pain. Febrile SCD patients without localizing symptoms should have blood cultures, urinalysis, and chest radiographs performed.

Patients presenting with symptoms of acute chest syndrome (chest pain, cough, fever, dyspnea) need immediate evaluation ([Table 136-2](#)). Assess oxygenation; type and cross for possible exchange transfusion.

History	<p>Major presenting symptom: dyspnea, fever, cough</p> <p>Accompanying chest, rib, bone, or joint pain</p> <p>Assess degree or severity of pain</p> <p>Recent or previous sepsis, infection, pneumonia, or hospitalization</p> <p>Prior history of acute chest syndrome, especially if required intubation and ventilatory support</p> <p>Potentially infectious contacts</p> <p>Current medications</p> <p>Immunization history: especially pneumococcal and <i>Haemophilus influenzae</i> type b</p> <p>Baseline hemoglobin level and arterial oxygenation saturation</p>
Physical examination	<p>General: respiratory distress, pallor, hydration, jaundice, rash</p> <p>Vital signs: especially temperature, pulse oximetry</p> <p>Respiratory: chest wall, lung sounds</p> <p>Heart: cardiomegaly and systolic murmur common with chronic anemia</p> <p>Abdomen: tenderness, organomegaly</p>
Ancillary tests	<p>Complete blood count, leukocyte differential, reticulocyte count, urinalysis</p> <p>Cross-match sample: if red blood cell transfusion is contemplated</p> <p>Arterial blood gas: if moderate to severe respiratory distress and/or hypoxemia on pulse oximetry</p> <p>Chest radiography</p> <p>Blood cultures</p> <p>Additional blood tests: as indicated by clinical condition</p>
Treatment	<p>Oxygen: adjust according to pulse oximetry</p> <p>Oral hydration: preferable</p> <p>IV hydration: use hypotonic fluids, use a rate and dose at approximately 1.5 of maintenance (over aggressive IV fluids can worsen acute chest syndrome)</p> <p>Analgesics: if needed, generally potent parenteral opioids are used, monitor for signs of respiratory suppression</p> <p>Antibiotics: empiric antibiotics recommended to treat community acquired pneumonia</p> <p>Bronchodilators: nebulized <math>\beta_2</math>-adrenergic agonists</p> <p>Chest physiotherapy</p> <p>Transfusion: use if severe acute anemia is present</p>
Exchange transfusion	<p>Consider when</p> <ul style="list-style-type: none"> <li>Severe acute chest syndrome on admission and past history of requiring ventilatory support: useful to prevent intubation</li> <li>Deterioration despite above management: useful to prevent intensive care unit admission</li> <li>Patient already intubated and on ventilatory support: useful to shorten duration of ventilatory need</li> <li>Suspected or confirmed fat or bone marrow embolism</li> </ul>

**TABLE 136-2** Assessment and Treatment of Acute Chest Syndrome

Radiographs of the skeleton are indicated for atypical focal bone pain. Advanced imaging for abdominal pain or for neurologic manifestations are helpful with assessment of these symptoms.

The differential diagnosis includes osteomyelitis, bony infarcts, cellulitis, acute arthritides, pancreatitis, hepatitis, cholecystitis, pelvic inflammatory disease, pyelonephritis, pneumonia, pulmonary embolus, and meningitis.

## **Emergency Department Care and Disposition**

Initial management for acute crisis in SCD patients is primarily supportive, and includes pain management and an assessment for the underlying cause of the crisis (see [Tables 136-1](#) and [136-2](#)).

Oral **rehydration** should be encouraged if dehydration is suspected. IV crystalloid may be used as an alternative, at 1.5 times maintenance. **Opioid pain medications** should be administered for severe pain. Individualized **treatment plans** are warranted for patients with frequent relapses. Supplemental **oxygen** is indicated for hypoxia. **ECG** and **cardiac monitoring** is appropriate for patients with cardiopulmonary symptoms. For symptoms of acute infection, **cultures** should be obtained and **broad spectrum antibiotics** administered. **Exchange transfusion** for acute crisis or complications should be considered in specific circumstances—aplastic crisis, cardiopulmonary decompensation, pregnancy, stroke, respiratory failure, general surgery, and priapism (requires urologic consultation). **Admission** criteria include pulmonary, neurologic, aplastic, or infectious crises; splenic sequestration; intractable pain; persistent nausea and vomiting; or an uncertain diagnosis. **Discharged** patients should receive oral analgesics, close follow-up, and instructions to return immediately for temperature above 38°C or worsening symptoms.

## **VARIANTS OF SICKLE CELL DISEASE**

Other genetic variants of hemoglobinopathies exist and vary in presentation, from asymptomatic to SCD-like and depend upon the specific

abnormality and whether homozygous, heterozygous, or combined with sickle cell trait.

## **Thalassemias**

Thalassemias are hereditary disorders caused by defective synthesis of globin chains, resulting in microcytic, hypochromic, hemolytic anemia. The degree of illness depends upon the type and number of genetic abnormalities.

Patients with  $\beta$ -thalassemia minor have mild microcytic anemia and are generally asymptomatic. Patients with  $\beta$ -thalassemia major (Cooley's anemia), develop hepatosplenomegaly, jaundice, and bony changes. They are at increased risk for infection and may develop severe anemia requiring blood transfusions. Iron overload from transfusions causes significant morbidity and mortality.

## **Glucose-6-Phosphate Dehydrogenase Deficiency**

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzymopathy of RBCs. This causes Hb precipitation, RBC removal by the spleen and hemolysis. The amount of hemolysis depends on degree of enzyme abnormality. Most patients are asymptomatic until an exposure to an oxidative stress (eg, medication, infection, fava beans) causes hemolysis. Evaluation includes a complete blood count and reticulocyte count, bilirubin levels, serum aminotransferases, and lactate dehydrogenase. Treatment is determined by the patient's overall clinical condition and includes early treatment of infection and may include blood transfusion for severe anemia.

## **Hereditary Spherocytosis**

Hereditary spherocytosis results from an erythrocyte membrane defect creating small inflexible RBCs unable to pass through the spleen resulting

in an increased rate of destruction and a compensatory increase in RBC production. Complications include aplastic or megaloblastic crises, cholecystitis or cholelithiasis, splenomegally and hemolysis with jaundice. Treatments include blood transfusions and splenectomy in severe cases.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 231, “Hereditary Hemolytic Anemias,” by Jean Williams-Johnson and Eric Williams.

# **CHAPTER 137**

## **Transfusion Therapy**

T. Paul Tran

The goal of transfusion is to improve oxygen delivery to tissues, provide intravascular volume expansion, and to replace missing or depleted clotting factors in patients with clinically significant hemorrhage, anemia, thrombocytopenia or coagulopathy. Great care must be taken to ensure that the correct blood product is delivered to the correct patient.

### **WHOLE BLOOD**

There are few indications for the use of whole blood transfusion. Although whole blood can provide volume expansion and oxygen-carrying capacity, the same can usually be accomplished more efficiently by using individual blood components.

### **PACKED RED BLOOD CELLS**

Packed red blood cells (PRBC) are prepared from whole blood by removing most platelets and/or white cells. A typical unit of PRBC has 250 mL of RBCs and raises an adult's hemoglobin by 1 gram/dL (hematocrit by 3%). PRBCs increase oxygen-carrying capacity in anemic patients.

The decision to transfuse PRBC is based on individual clinical judgment, taking into account patient's hemodynamic status, underlying medical condition, tolerance for anemia, and risk of end-organ ischemic injury. Adequate oxygen delivery in healthy normovolemic patients can be maintained with hemoglobin levels as low as 7 grams/dL, although patients with comorbid conditions may require transfusion at higher levels of hemoglobin. The usual indications for PRBC transfusion include: (a) acute hemorrhage, defined as blood loss  $> 25\%$  of blood volume, (b) hemorrhagic

shock, (c) surgical blood loss greater than 2 L; and (d) symptomatic anemia or being at-risk for ischemic events, ie, patients with hemoglobin <6 grams/dL who have symptoms of end-organ ischemia,  $\geq 55$  years of age, have cardiac disease, sepsis, severe infection, or APACHE II score  $> 20$ .

Type and crossmatch assesses ABO/Rh blood type, the presence of antibodies, and patient and donor blood compatibility. It takes 15 to 30 min to perform and is ordered if the likelihood of transfusion is high. Type and screen assesses the ABO/Rh blood typing and the presence of antibodies. It takes 15 to 30 min to perform and is ordered if the likelihood of transfusion is low.

In critical situations, where there is no time to perform a complete ABO/Rh-typing, group O/Rh-negative blood (“universal donor”) can be given to patients without waiting for a complete type and crossmatch. Type O/Rh-positive blood may be used if Rh-negative blood is unavailable but is generally avoided in girls and women of childbearing age. Before transfusion, blood for baseline laboratory tests, type, and crossmatching should be obtained.

PRBC may be further treated to minimize complications in special patient populations, such as neonates, transplant and patients on transplant list, patients who have received prior transfusions, pregnant patients, immunocompromised patients, and patients with hypersensitivity to plasma. Options include leukocyte reduced, irradiated, frozen deglycerolized, washed, and CMV negative PRBC.

A unit of PRBC is usually transfused over 2 hours but can be given much faster using a pressure infusing device or over 4 hours, if needed. Micropore filters are used to filter out microaggregates of platelets, fibrin, and leukocytes. Normal saline solution is the only crystalloid compatible with PRBC. Blood warmers or concurrently administered warmed saline solution ( $39^{\circ}\text{C}$  to  $43^{\circ}\text{C}$  or  $102.2^{\circ}\text{F}$  to  $109.4^{\circ}\text{F}$ ) can be used to prevent hypothermia.

## **PLATELETS**

Platelet transfusions may be used in thrombocytopenic patients to prevent bleeding or to help stop active bleeding. Platelet transfusion is usually not helpful in cases of bleeding from platelet dysfunction (eg, uremia) or from

thrombocytopenia due to increased consumption/sequestration until the underlying disorder (eg, DIC) is corrected. General indications for platelet transfusion include platelet count  $<10,000 \text{ mm}^3$  in asymptomatic patients, platelet count  $<15,000/\text{mm}^3$  with a coagulation disorder or minor bleeding, platelet count  $<20,000/\text{mm}^3$  with major bleeding, platelet count  $<50,000/\text{mm}^3$  with an invasive procedure, general surgery or during massive transfusion, or platelet count  $<100,000/\text{mm}^3$  with neurologic or cardiac surgery.

Each unit of single donor platelets contains approximately  $3 \text{ to } 6 \times 10^{11}$  platelets in 250 to 300 mL of plasma and may increase the platelet count in an adult by up to  $50,000/\text{mm}^3$ , but less in many cases. Typical dose is 1 unit or 5 mL/kg. Typical dose of pooled donor platelet is 6 units or 5 mL/kg.

ABO- and Rh-compatible platelets are preferable. Platelets may also be washed or irradiated. The platelet count should be checked at 1 and 24 hours after transfusion. Transfused platelets survive 3 to 5 days in the absence of an ongoing platelet consumption process.

## **FRESH FROZEN PLASMA**

Fresh frozen plasma (FFP) contains all coagulation factors and fibrinogen. FFP is used to correct coagulation defects in bleeding patients with multiple coagulation deficiencies (liver disease, warfarin therapy/OD, massive transfusion) and to correct coagulation defects for which no factor is available. FFP is also administered prior to high-risk invasive procedures if  $\text{PT/INR} > 1.5 \times$  normal,  $\text{aPTT} > 1.5 \times$  top of normal range, or coagulation factor assay  $< 25\%$  normal activity. FFP is not indicated to treat a prolonged INR in the absence of active bleeding.

FFP needs to be thawed for 20 to 40 min before it can be used. Each 200 to 250 mL pack of FFP contains 1 unit/mL of each coagulation factor and 1 to 2 milligrams/mL fibrinogen. One pack will increase most coagulation factors by up to 5%. FFP should be ABO compatible. Starting dose is 15 mL/kg (approximately 4 bags). At certain medical centers, prothrombin complex concentrates or recombinant activated factor VII is used in lieu of FFP.



## **CRYOPRECIPITATE**

Cryoprecipitate is derived from FFP. One bag of cryoprecipitate contains 80 units factor VIII, 200 to 300 milligrams von Willebrand factor, 40 to 60 units fibrinogen, factor XIII, and variable amounts of fibronectin.

Cryoprecipitate is indicated for (a) active bleeding in patients with afibrinogenemia or hypofibrinogenemia (fibrinogen level <100 milligrams/dL) as a result of a pathological process (DIC, liver disease, abruptio placentae, amniotic fluid embolus, massive transfusion); (b) active bleeding in patients with von Willebrand disease when desmopressin (DDAVP) is not effective or in patients with von Willebrand disease type 2B, or if factor VIII concentrate containing von Willebrand factor is not available; and (c) hemophilia type A when virally inactivated factor VIII concentrates are not available. Cryoprecipitate can also be used as fibrin surgical adhesives in surgical patients.

Cryoprecipitate should be ABO-compatible. The volume of each unit is 20 to 50 mL. The usual dose is 1 unit/5 kg of body weight (10 to 14 units for an adult) and will raise fibrinogen concentration to 75 milligrams/dL.

## **INTRAVENOUS IMMUNOGLOBULINS**

Intravenous immunoglobulin (IVIG) is a pooled IgG product from numerous donors. Among several FDA approved and numerous off-label uses, IVIG is commonly indicated for the treatment of primary and secondary immunodeficiencies, immune-mediated thrombocytopenia; and Kawasaki syndrome. Adverse reactions include anaphylaxis, febrile reactions, headache, and renal failure.

## **ANTITHROMBIN III**

Antithrombin III (ATIII) is a serum protein that inhibits coagulation factors, thrombin, and activated factors IX, X, XI, and XII. Deficiency can be

congenital or acquired. ATIII is used mainly for prophylaxis of thrombosis or to treat thromboembolism in patients with hereditary ATIII deficiency.

## **SPECIFIC                      FACTOR                      REPLACEMENT THERAPY**

[Table 137-1](#) outlines therapy for congenital coagulation factor deficiencies.

Couglation Factor	Annual Incidence	Replacement Therapy
Factor I (fibrinogen)	Rare (1 to 2/million)	Whole blood, FFP, cryoprecipitate, fibrinogen concentrates
Factor II (prothrombin)	Rare (1 to 2/million)	FFP, factor II concentrate, prothrombin complex concentrate
Factor V (proaccelerin, labile factor)	Rare (1/million)	Whole blood, FFP
Factor VII (proconvertin, stable factor)	Rare (0.5/million)	FFP, prothrombin complex concentrate, purified factor VIIa, recombinant factor VIIa
Factor VIII (antihemophilic factor A, antihemophilic globulin, "class: hemophilic")	1 to 2/10,000 male births	Factor VIII concentrates (cryoprecipitate or FFP if not available); DDaVP for mild hemophilia
von Willebrand disease	up to 1 in 100 persons	DDaVP for mild von Willebrand disease (EXCEPT types 2B or 3); factor VIII concentrates (Aphanate, Humate-P); cryoprecipitate
Factor IX (antihemophilic factor B, plasma thromboplastin component, Christmas factor)	1/30,000 male births	Factor IX concentrates
Factor X (Stuart-Prower factor)	Rare (1 to 2/million)	Whole blood, FFP, Factor X concentrate, prothrombin complex concentrate
Factor XI (plasma thromboplastin antecedent, hemophilia C, Rosenthal syndrome)	1/10,000 in Ashkenazi Jews; 1/100,000 in general population	FFP, cryoprecipitate, Factor XI concentrate
Factor XII (Hageman factor)	Rare (1/million)	Replacement not required
Factor XIII (fibrin stabilizing factor, Laki-Lorand factor)	Rare (1/5 million)	FFP, cryoprecipitate, Factor XIII concentrate

Key: FFP = fresh frozen plasma; DDaVP = Desmopressin

**TABLE 137-1** Replacement Therapy for Congenital Factor Deficiencies

## MASSIVE TRANSFUSION

*Massive transfusion* is defined as replacement of a patient's total blood volume within a 24-hour period. The exact ratio of PRBC to platelets to FFP remains controversial; some experts recommend a 1:1:1 ratio. Complications of massive transfusion include coagulopathy, citrate toxicity, hypocalcaemia, hypomagnesaemia, and hypothermia. See [Table 137-2](#) for evaluation and management of associated complications.

Reaction Type	Signs and Symptoms	Evaluation	Management
Febrile nonhemolytic transfusion reactions (FNHTR)	Mild fever (<1.5°C rise), chills, urticaria	Can be difficult to distinguish from AHR Consider bacterial infection Hemolytic workup (see AHR)	Stop transfusion, check vital signs, O <sub>2</sub> saturation, verify patient ID, administer acetaminophen for fever, antihistamine for mild urticaria Usually self-limited but can be life threatening in patients with serious cardiopulmonary status
Acute intravascular hemolytic reaction (AIHR) vs bacterial infection	Fever, back pain, hematuria (hemoglobinuria), chills, "sense of doom," flushing, dyspnea, tachycardia, shock, renal failure, syncope, DIC Transfusion of blood contaminated with bacteria tends to precipitate a severe acute reaction with rapid onset of hyper- or hypotension, rigors and cardiovascular collapse	Save the blood unit, notify blood bank, retype and crossmatch, direct and indirect Coombs tests, CBC, creatinine, PT, aPTT, haptoglobin, indirect bilirubin, LDH, plasma free hemoglobin, blood cultures, UA	Stop transfusion, start IV hydration to maintain diuresis, diuretics if anuria or oliguria (<100 mL/h), start broad spectrum antibiotics if suspicious bacterial infection, treat DIC Cardiorespiratory support is indicated
Allergic reaction/Anaphylaxis	Rapid onset, urticaria, pruritus, dyspnea, nausea, vomiting, syncope, headache, bronchospasm, angioedema, abdominal pain, hypotension	Notify blood bank For mild symptoms that resolve with antihistamines, no further workup needed	Stop transfusion and assess patient. If allergic reaction is mild, treat with antihistamines; if symptoms resolve, can restart transfusion If allergic reaction is severe, treat as anaphylaxis
Fluid overload	Dyspnea, tachycardia, hypertension, headache, jugular venous distention, pulmonary rales, hypoxia, hypotension if volume overload is severe	ECG, chest x-ray, monitor CVP, urine output, blood gas as indicated by clinical situation	Stop transfusion (or decrease rate to 1 mL/kg/h if very mild symptoms), start diuretics (furosemide)
Transfusion related acute lung injury (TRALI)	Dyspnea, nonproductive cough, acute respiratory distress syndrome (ARDS), hypotension, fever/chills, monocytopenia/neutropenia	Chest x-ray (bilateral nodular infiltrates, batwing pattern)	Stop transfusion, consult hematology, treat as ARDS
Complications from massive transfusion	Bleeding, hypothermia, citrate toxicity, hypocalcaemia or hypomagnesemia	Monitor temperature, coagulation parameters, acid base balance, serum potassium and calcium	Use blood warmers for hypothermia; administer warm isotonic fluid, additional PRBC, FFP, cryoprecipitate, and platelet as clinically indicated; treat symptomatic hypocalcaemia or hypomagnesemia

Key: PT = prothrombin time, aPTT = activated partial thromboplastin time, CBC = complete blood count, CVP = central venous pressure, DIC = disseminated intravascular coagulation, FFP = fresh frozen plasma, LDH = lactate dehydrogenase

**TABLE 137-2** Selected Acute Transfusion Reactions: Recognition, Management, and Evaluation

## **COMPLICATIONS OF TRANSFUSIONS**

Acute complications of include febrile nonhemolytic transfusion reaction (most common), acute hemolytic reaction, ABO incompatibility, allergic reaction and anaphylaxis, and transfusion related acute lung injury (TRALI). [Table 137-2](#) summarizes the clinical presentation, evaluation, and management of acute transfusion reactions.

Delayed transfusion complications present days or weeks after the index transfusion and include delayed hemolytic transfusion reaction, transfusion associated graft vs host disease, posttransfusion purpura, and infectious disease transmission.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 6th ed., see Chapter 233, “Transfusion Therapy,” by Clinton J. Coil and Sally A. Santen.

## CHAPTER 138

# Anticoagulants, Antiplatelet Agents, and Fibrinolytics

Jessica L. Smith

Antithrombotic therapy is used to treat ST elevation MI, non-ST elevation MI, unstable angina, deep venous thrombosis, pulmonary embolism, transient ischemic attack, and ischemic stroke. These agents are also used to prevent occlusive vascular events in patients at risk. Detailed management strategies and dosing regimens are provided in [Chapter 18](#) “Acute Coronary Syndromes: Management of Acute Myocardial Infarction and Unstable Angina,” [Chapter 25](#) “Thromboembolism,” and [Chapter 141](#) “Stroke and Transient Ischemic Attack.” This chapter provides an overview of antithrombotic agents.

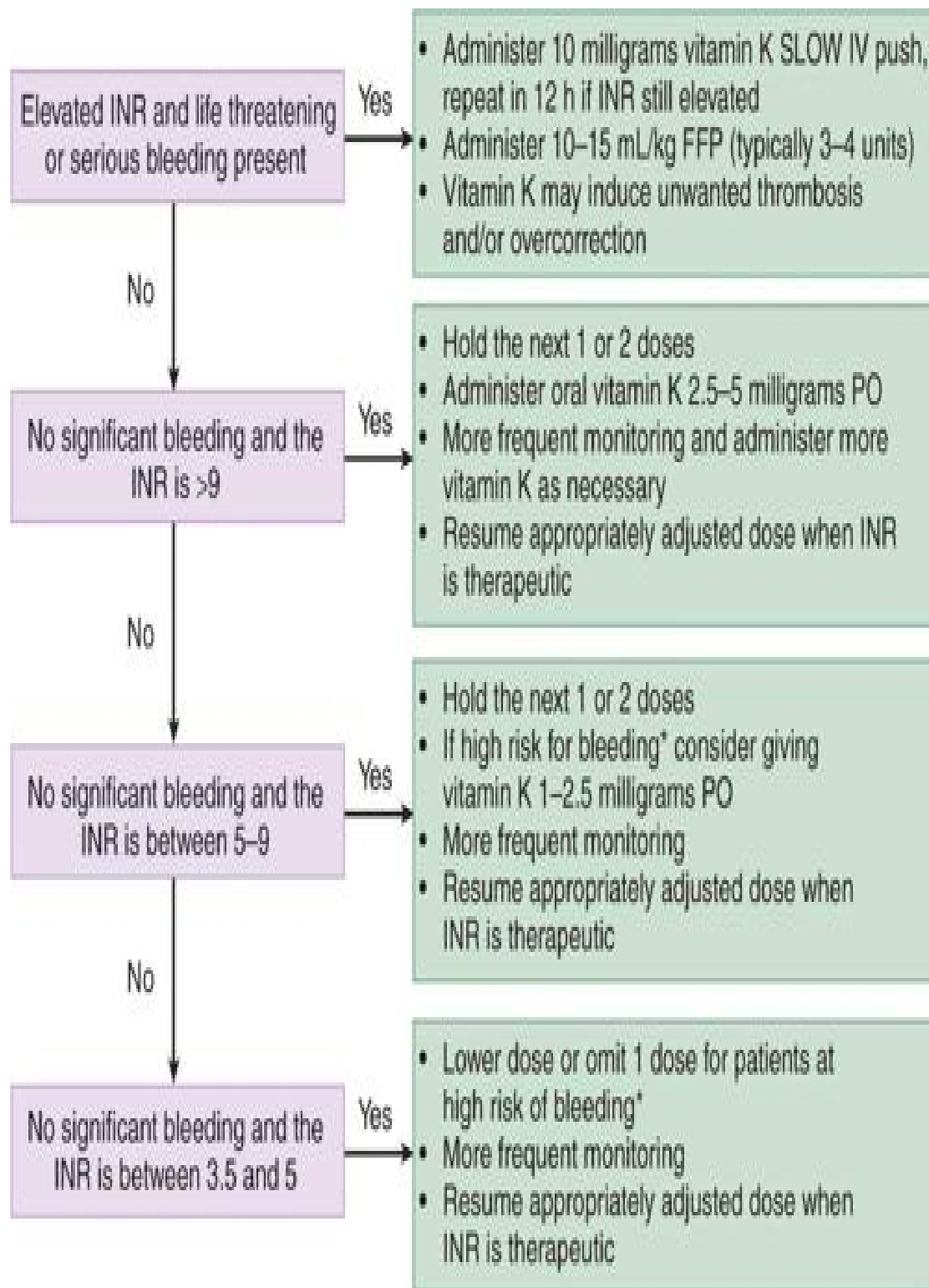
## ANTICOAGULANTS

The goals of anticoagulant therapy include (1) stop further acute thrombosis, (2) reduce the risk of embolism from a thrombus, and (3) prevent the formation of de novo thrombus in patients at risk.

### Oral Agents

**Warfarin** is the most commonly used oral anticoagulant in the United States. It works by preferentially inhibiting vitamin K dependent cofactors in the extrinsic coagulation cascade. Dosing is guided by measuring the international normalized ratio (INR), and most patients are therapeutic in a range of 2 to 3. Patients with mechanical heart valves or antiphospholipid antibody syndrome require an INR of 2.5 to 3.5. It takes about 3 to 4 days to reach full anticoagulation upon initiating treatment. A parenteral anticoagulant should be used until the INR is maintained in the desired range for 2 days as warfarin therapy causes a transient state of thrombogenesis at the start of therapy. A number of medications, foods, or disease states interfere with warfarin absorption or metabolism and cause clinically significant consequences. Warfarin use is contraindicated during pregnancy due to teratogenicity. Complications of warfarin use include bleeding from over-anticoagulation, increased bleeding risk in patients with hypertension, anemia, prior cerebrovascular disease, GI lesions, and renal disease. Skin necrosis is associated

with protein C deficiency. [Figure 138-1](#) describes the management of warfarin-induced coagulopathy.





**FIGURE 138-1.** Algorithm for management of warfarin-induced coagulopathy.

\*High risk of bleeding: age > 75 years, concurrent antiplatelet drug use, polypharmacy, liver or renal disease, alcoholism, recent surgery, or trauma. FFP = fresh frozen plasma; INR = international normalized ratio.

**Dabigatran** is currently the only available oral direct thrombin inhibitor in the United States. It requires no laboratory monitoring. No reversal agent is available and long-term safety has not been established. **Rivaroxaban**, a direct Factor Xa inhibitor, is available in Europe and Canada.

## Parenteral Agents

Parenteral agents include **unfractionated heparin (UFH)**, **low molecular weight heparin (LMWH)** (eg, **enoxaparin**, **dalteparin**), **Xa inhibitors (fondaparinux)**, and **direct thrombin inhibitors** (eg, **bivalirudin**, **lepirudin**, **argatroban**). UFH and LMWH are used to treat and prevent deep vein thrombosis, as well as pulmonary embolism (PE), unstable angina, and acute myocardial infarction. **Enoxaparin** 1 milligram/kilogram SC every 12 hours may be used in outpatient management of DVT. Dosing regimens for UFH and LMWH are weight based. UFH requires monitoring of the activated partial thromboplastin time (aPTT). Therapeutic range is 1.5 to 2.5 “normal” value. Heparin and LMWH may be used during pregnancy. Bivalirudin and argatroban are alternatives to UFH and LMWH during percutaneous intervention (PCI) for acute coronary syndrome (ACS). The 2 major complications of heparin are bleeding and heparin induced thrombocytopenia (HIT). Use hirudin, lepirudin, or argatroban for anticoagulation in patients with HIT. LMWH carries a lower bleeding risk than UFH, but a higher bleeding risk in patients with renal disease. LMWH may also cause pruritis, local skin reaction, or rarely, skin necrosis.

## ANTIPLATELET AGENTS

Oral agents include aspirin, clopidogrel, ticlopidine, and dipyridamole. Aspirin is an irreversible cyclooxygenase inhibitor; its effects last for the life of the platelet. Nonenteric-coated aspirin (162 to 325 milligrams) should be administered in the setting of ACS, and although it is contraindicated during active GI hemorrhage, it is generally considered safe to give to closely monitored patients with guaiac positive stool. Clopidogrel and ticlopidine inhibit platelet activation by rendering the fibrinogen receptor ineffective. A loading dose of **clopidogrel 600 milligram PO** results in full antiplatelet effect by 2 hours, and sustained effects for up to 48 hours.

Ticlopidine is infrequently used due to risk of neutropenia and TTP. Side effects of aspirin are mainly GI and dose related. Complications of clopidogrel include dyspepsia, rash, or diarrhea. Omeprazole reduces efficacy.

## **Parenteral Agents**

GPIIb/IIIa agents alter the common final pathway receptor in platelet aggregation. Agents used in conjunction with PCI include **abciximab** 0.25 milligram/kilogram IV bolus followed by 0.125 microgram/kilogram/ min (up to 10 micrograms) IV, **eptifibatid** 180 micrograms/kilogram IV bolus over 1 to 2 min followed by 2 micrograms/kilogram/min IV, or **tirofiban** 0.4 microgram/kilogram/min IV for 30 min followed by 0.1 microgram/ kilogram/min IV. These agents should be used in consultation with the interventional cardiologist. Patients receiving GPIIb/IIIa agents are at increased risk of bleeding complications.

## **FIBRINOLYTICS**

Fibrinolytic agents include **streptokinase, anistreplase, alteplase/tPA, reteplase, and tenecteplase**. Although mechanisms of action vary, each agent eventually converts plasminogen to plasmin, which breaks down the fibrin in a thrombus. Alteplase/tPA theoretically causes less systemic fibrinolysis, without the antigenic side effects of streptokinase and anistreplase that limit retreatment within 6 months and treatment within 12 months of a streptococcal infection. Strict adherence to established guidelines and informed consent is essential. Hemorrhagic complications, including intracranial hemorrhage, may occur. Streptokinase and anistreplase may cause hypotension or anaphylaxis. Contraindications to fibrinolytic therapy are listed in [Table 138-1](#).

Absolute
Active or recent (<14 days) internal bleeding
Ischemic stroke within the past 2 to 6 months
Any prior hemorrhagic stroke
Intracranial or intraspinal surgery or trauma within the past 2 months
Intracranial or intraspinal neoplasm, aneurysm, or arteriovenous malformation
Known severe bleeding diathesis
Current anticoagulant treatment (eg, warfarin with INR >1.7 or heparin with increased aPTT)
Uncontrolled hypertension (ie, blood pressure >185/100 mm Hg)
Suspected aortic dissection or pericarditis
Pregnancy
Relative*
Active peptic ulcer disease
Cardiopulmonary resuscitation for longer than 10 min
Hemorrhagic ophthalmic conditions
Puncture of noncompressible vessel within the past 10 days
Advanced age >75 years old
Significant trauma or major surgery within the past 2 weeks to 2 months
Advanced renal or hepatic disease

Key: aPPT = activated partial thromboplastin time; INR = international normalized ratio.

\*Concurrent menses is not a contraindication.

**TABLE 138-1** General Contraindications to Fibrinolytic Therapy

## COMPLICATIONS OF ANTITHROMBOTIC USE

Emergency treatment of bleeding complications of antithrombotic therapy are listed in [Table 138-2](#).

Agent	Management
Heparin	<p data-bbox="521 264 1062 380">Immediate cessation of heparin administration. Supratherapeutic aPTT not always present. Anticoagulation effect lasts up to 3 h from last IV dose. Observation with serial aPTT may be sufficient.</p> <p data-bbox="521 382 1062 583">Major bleeding Protamine, 1 milligram per 100 units of total amount of IV administered within the past 3 h. Protamine is given slowly IV over 1 to 3 min to a maximum of 50 milligrams over any 10-min period. Protamine has an anaphylaxis risk. Protamine does not completely reverse low-molecular-weight heparin.</p> <p data-bbox="521 585 1062 701">Enoxaparin: Protamine 1 milligram IV for every 1 milligram of enoxaparin given in the previous 8 h. If 8 to 12 h since last enoxaparin dose, give protamine 0.5 milligram IV for every 1 milligram of enoxaparin given.</p> <p data-bbox="521 703 1062 871">Dalteparin and tinzaparin: Protamine 1 milligram IV per every 100 international units of dalteparin or tinzaparin given. If aPTT (measured 2 to 4 h after the protamine infusion) remains prolonged, a second dose of protamine 0.5 milligram IV per 100 international units of dalteparin or tinzaparin.</p>

Agent	Management
Antiplatelet agents	
Aspirin	Cessation of aspirin administration. Platelet transfusion to increase count by 50,000/mm <sup>3</sup> (typically requires at least 6 units of random donor platelets). Aspirin-induced platelet inhibition may last for 7 days, so repeat platelet transfusions are sometimes required.
Other antiplatelet agents	Platelet transfusion to increase count by 50,000/mm <sup>3</sup> (typically requires at least 6 units of random donor platelets). NSAID-induced platelet inhibition typically lasts <1 day. Clopidogrel-induced platelet inhibition may last up to 7 days.
Fibrinolytics	
Minor external bleeding	Manual pressure
Significant internal bleeding	Immediate cessation of fibrinolytic agent, antiplatelet agent, and/or heparin. Reversal of heparin with protamine as above. Typed and crossmatched blood ordered with verification of aPTT, complete blood count, thrombin dotting time, and fibrinogen level. Volume replacement with crystalloid and packed red blood cells as needed.
Major bleeding or hemodynamic compromise	All measures listed for significant internal bleeding. Cryoprecipitate, 10 units IV, and recheck fibrinogen level; if fibrinogen level <100 milligrams/dL, repeat cryoprecipitate. If bleeding persists after cryoprecipitate or despite fibrinogen level >100 milligrams/dL, administer FFP, 2 units IV. If bleeding continues after FFP, administer an antifibrinolytic such as ε-aminocaproic acid, 5 grams IV over 60 min followed by 1 gram/h continuous IV infusion for 8 h or until bleeding stops, or tranexamic acid, 10 milligrams/kilogram IV every 6 to 8 h.
Intracranial hemorrhage	Consider transfusion of 10 units of random donor platelets. All measures listed for significant internal and major bleeding with hemodynamic compromise. Immediate neurosurgery consultation.

Key: aPTT = activated partial thromboplastin time; FFP = fresh frozen plasma.

**TABLE 138-2** Emergency Treatment of Bleeding Complications of Antithrombotic Therapy

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 234, “Anticoagulants, Antiplatelet Agents, and Fibrinolytics,” by David E. Slattery and Charles V. Pollack, Jr.

## **CHAPTER 139**

# **Emergency Complications of Malignancy**

Ross J. Fleischman

Oncologic emergencies arise from the underlying malignancy or as complications of radiation and chemotherapy.

## **AIRWAY OBSTRUCTION**

Patients with tumors of the upper and lower respiratory tract may experience acutely worsening airway compromise due to edema, bleeding, infection, or loss of protective mechanisms. Presenting symptoms and signs include dyspnea, tachypnea, wheezing and stridor. Imaging involves plain radiographs, CT scan, and/or endoscopic visualization. Emergency measures include supplemental humidified oxygen, maintenance of airway through optimal patient positioning, and, possibly, administration of a helium-oxygen mixture. If intubation is required, an “awake look” with a fiber optic bronchoscope with a 5-0 or 6-0 endotracheal tube is preferred. An emergency surgical airway, such as cricothyroidotomy, transtracheal jet ventilation, or tracheotomy may be needed. Consult with an oncologist or surgeon for definitive management.

## **BONE METASTASES AND SPINAL CORD COMPRESSION**

Patient with solid tumors, most commonly breast, lung, and prostate, may present with pain, pathologic fracture, or spinal cord compression caused by bony metastases. Patients with spinal cord compression may also exhibit muscular weakness, radicular pain, and bowel or bladder dysfunction. Plain radiographs are obtained initially to assess for fracture or bony involvement, followed by CAT scan or MRI to further delineate lesions.

Treatment priorities include pain control with opioid analgesia and restoration or salvage of function. Most pathologic fractures require surgical intervention. Painful bone metastases are treated with radiotherapy. The presentation, evaluation, and management of malignant spinal cord compromise are described in [Table 139-1](#).

Suspect	Patient with known cancer: especially lung, breast, prostate. Thoracic location: 70%. Progressive pain and worse when supine. Motor weakness: proximal legs. Sensory changes and bladder or bowel dysfunction: late findings.
Imaging	Plain radiographs: may detect vertebral body metastases but less sensitive and specific for malignant spinal cord compression. MRI: modality of choice, image entire vertebral column. CT myelography: used when MRI not available or accessible.
Corticosteroids	Dexamethasone, 10 milligrams IV followed by 4 milligrams PO or IV every 6 h. Consider starting in ED if imaging is delayed.
Radiotherapy	Standard approach, beneficial in approximately 70%. No specific radiotherapy regimen proven superior. Prognosis highly dependent on pretreatment neurologic function.
Surgery	Consider in highly selected cases, such as: Patient in good general condition and able to undergo extensive surgery Appropriate prognostic life expectancy Rapidly progressive symptoms Clinical worsening during radiotherapy Unstable vertebral column

**TABLE 139-1** Malignant Spinal Cord Compression

## MALIGNANT PERICARDIAL EFFUSION

Malignant pericardial effusions are usually asymptomatic but can progress to life-threatening cardiac tamponade. Symptoms depend on the rate of accumulation and distensibility of the pericardial sac (see [Chapter 24](#) “The Cardiomyopathies, Myocarditis, and Pericardial Disease”). Patients with symptomatic effusion may present with chest heaviness, dyspnea, cough, and syncope. Physical examination findings include tachycardia, narrowed

pulse pressure, hypotension, distended neck veins, muffled heart tones, and pulsus paradoxus.

Echocardiography is the test of choice as it demonstrates the size of the effusion and the presence of tamponade. Chest radiograph may demonstrate an enlarged cardiac silhouette or pleural effusion. ECG may show sinus tachycardia, low QRS amplitude, and electrical alternans. The differential diagnosis includes cardiomyopathy related to chemotherapy, such as doxorubicin and radiation therapy. Emergent ultrasound-guided pericardiocentesis may be required to relieve cardiac tamponade.

## **SUPERIOR VENA CAVA SYNDROME**

Superior vena cava syndrome most commonly occurs due to external compression by an external malignant mass such as lung cancer or lymphoma. Less common causes include thrombosis and benign masses. The most common symptoms are gradual onset of dyspnea, chest pain, cough, distended neck veins, face or arm swelling. CT of the chest with IV contrast is the diagnostic procedure of choice. CXR will usually show a mediastinal mass.

Patients with neurologic symptoms require urgent treatment including supplemental oxygen and elevation of the head and upper body. **Dexamethasone** 20 milligrams IV or **methylprednisolone** 125 to 250 milligrams IV may be beneficial in patients with increased intracranial pressure or lymphoma. In patients without neurologic symptoms, SVC syndrome usually does not cause rapid deterioration and can await consultation regarding chemotherapy, radiation, or intravascular stenting. Patients with intravascular thrombosis may require anticoagulation, fibrinolysis, or catheter removal.

## **HYPERCALCEMIA OF MALIGNANCY**

Hypercalcemia is most commonly seen with breast and lung cancer, lymphoma, and multiple myeloma. The symptoms are nonspecific and



include polydipsia, polyuria, generalized weakness, lethargy, anorexia, nausea, constipation, abdominal pain, volume depletion, and altered mentation.

Clinical signs and symptoms are related to the rate of rise and occur above 12 milligrams/dL ((ionized >5.5 milligrams/dL)). ECG may show shortened QT interval, ST depression, and atrioventricular blocks.

**Normal saline** infusion is the mainstay of treatment. Furosemide is no longer recommended. Further treatment should be discussed with the patient's oncologist. Bisphosphonates such as **zoledronic acid** 4 milligrams IV over 15 min or **pamidronate** 60 to 90 milligrams IV over 4 to 24 hours can prevent bone resorption. **Calcitonin** 4 international units/killogram SC or IM causes a more rapid decrease in calcium levels. Glucocorticoids may be helpful in lymphoma and multiple myeloma. Consider hemodialysis for patients with profound mental status changes, renal failure, or those who cannot tolerate a normal saline infusion (see [Chapter 4](#) "Fluids, Electrolytes and Acid Base Disorders").

## **HYPONATREMIA DUE TO SYNDROME OF INAPPROPRIATE ANTI-DIURETIC HORMONE (SIADH)**

Inappropriate ADH secretion is most commonly associated with bronchogenic lung cancer, but can also occur from chemotherapy or medications. Symptoms include anorexia, nausea, headache, altered mentation, and seizures. Mild hyponatremia ( $> 125$  mEq/L) is usually asymptomatic.

SIADH should be suspected in patients with cancer who present with normovolemic hyponatremia. Laboratory abnormalities include serum osmolality  $< 280$  mOsm/L, urine osmolality  $> 100$  mOsm/L, and urine sodium  $> 20$  mEq/L. The differential diagnosis includes hypothyroidism, renal failure, cirrhosis, adrenal crisis and hypo/hypervolemia.

Mild hyponatremia  $> 125$  mEq/L is treated with a **water restriction** of 500 mL/d and close follow-up. More severe hyponatremia is treated with **furosemide**, 0.5 to 1 milligram/killogram PO with normal saline infusion to

maintain normovolemia. **Demeclocycline** 300 to 600 milligrams PO twice daily may increase water excretion. **Three percent hypertonic saline** is reserved for severe hyponatremia <120 mEq/L with seizures or coma. An infusion of 25 to 100 mL/h should be titrated to a correction of 0.5 to 1 mEq/h with a maximum of 12 mEq/L/d (see [Chapter 4](#) “Fluids, Electrolytes and Acid Base Disorders”).

## ADRENAL CRISIS

Adrenal crisis is most commonly caused by acute physiologic stress in the face of exogenous steroid-induced adrenal suppression or malignant infiltration of adrenal tissue. Symptoms include weakness or nausea, and hypotension unresponsive to fluids. Laboratory abnormalities may include hypoglycemia, hyponatremia, and hyperkalemia. A serum cortisol level is ideally drawn before giving steroids. Treat acute adrenal insufficiency empirically with stress-dose **hydrocortisone** 100 to 150 milligrams IV, **methylprednisolone** 20 to 30 milligrams IV, or **dexamethasone** 4 milligrams IV, IV crystalloids and supportive care (see [Chapter 132](#) “Adrenal Insufficiency and Adrenal Crisis”).

## TUMOR LYSIS SYNDROME

Tumor lysis syndrome occurs when massive quantities of potassium, phosphate, and uric acid are released into the systemic circulation. It usually occurs 1 to 3 days after chemotherapy for acute leukemia or lymphoma. The resulting hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia may cause uric acid precipitates and calcium phosphate deposits in the kidney with renal failure, life-threatening arrhythmias, tetany, and seizures. Laboratory evaluation should include 12-lead ECG, basic electrolyte levels, complete blood count, uric acid, and phosphorus.

Preventative measures reduce the incidence of tumor lysis syndrome. Hyperkalemia is the most immediate life threat. Treatment of hyperkalemia is with **insulin**, **glucose**, **bicarbonate** (if acidotic), **kayexalate**, and

**albuterol** (see [Chapter 4](#) “Fluids, Electrolytes and Acid Base Disorders”). **Calcium** administration is avoided unless ventricular arrhythmias or widened QRS complexes are seen, as it may worsen calcium phosphate precipitation in the kidney. Aggressive **infusion of isotonic fluids** reverses volume depletion and helps to prevent renal deposition of uric acid and calcium phosphate crystals. Hyperuricemia may be treated with **rasburicase** 0.2 milligram/ killogram IV. Hyperphosphatemia is managed with IV **insulin** and **glucose**. Phosphate binders have a limited effect. Consider hemodialysis for potassium levels above 6.0 mEq/L, uric acid levels above 10.0 milligrams/dL, phosphate levels above 10 milligrams/dL, creatinine levels above 10 milligrams/ dL, symptomatic hypocalcemia, or volume overload. Patients with tumor lysis syndrome should be admitted to an intensive care unit.

## **FEBRILE NEUTROPENIA**

Febrile neutropenia is defined by temperatures above 38°C for an hour or a single temperature above 38.3°C with an absolute neutrophil count (ANC) below 1000 cells/mm<sup>3</sup>. Neutrophil counts typically reach a nadir 10 to 15 days after chemotherapy and rebound 5 days later.

Febrile neutropenic patients often lack localizing signs and symptoms because of an attenuated immune response. Meticulous attention must be paid to all skin surfaces, mucosal areas, and vascular access sites in which the patient may have an occult infection. Digital rectal examination is often withheld until after initial antibiotic administration because of the fear of inducing bacteremia.

Laboratory evaluation includes complete blood count with differential, blood cultures obtained through all lumens of indwelling catheters as well as a peripheral site, urinalysis, urine culture, and CXR, electrolytes, renal, and liver function tests. Additional studies based on symptoms may include stool culture (diarrhea), sputum culture (cough), lumbar puncture (headache, stiff neck, altered mental status), wound culture (drainage), and CT or ultrasound (abdominal pain).

The decision for empiric antibiotics and admission should be made with the patient's oncologist. **Empiric antibiotics** ([Table 139-2](#)) are generally indicated for an ANC <500/mm<sup>3</sup>. For neutrophils counts between 500 and 1000, the decision is based on the patient's presentation. Add **vancomycin** for severe mucositis, catheter site infection, recent use of fluoroquinolone prophylaxis, hypotension, residence in an institution with hospital-associated methicillin-resistant *Staphylococcus aureus*, or known colonization with resistant gram-positive organisms.

Circumstance	Drug and Adult Dosage	Comments
Outpatient	Ciprofloxacin, 500 milligrams PO every 8 h <i>and</i> Amoxicillin/clavulanate, 500 milligrams PO every 8 h	Useful for low-risk patients with daily assessments by a medical provider for the initial 3 days.
Monotherapy	Cefepime, 2 grams IV every 8 h <i>or</i> Ceftazidime, 2 grams IV every 8 h <i>or</i> Imipenem/cilastatin, 1 gram IV every 8 h <i>or</i> Meropenem, 1 gram IV every 8 h <i>or</i> Piperacillin/tazobactam, 4.5 grams IV every 6 h	Monotherapy with these broad-spectrum agents appears to be as good as dual-drug therapy in most circumstances.
Dual therapy	One of the monotherapy agents <i>plus</i> Gentamicin, 1.7 milligrams/killogram IV every 8 h <i>or</i> Tobramycin, 1.7 milligrams/killogram IV every 8 h <i>or</i> Amikacin, 5 milligrams/killogram IV every 8 h	Potential advances include synergistic effects against some gram-negative bacteria and reduced emergence of drug resistance. Increased risk for adverse effects, including nephrotoxicity, ototoxicity, and hypokalemia.
Risk factors for severe gram-positive infection (see text)	Vancomycin, 1 gram IV every 12 h <i>plus</i> Cefepime, 2 grams IV every 8 h <i>or</i> Ceftazidime, 2 grams IV every 8 h <i>or</i> Imipenem/cilastatin, 1 gram IV every 8 h <i>or</i> Meropenem, 1 gram IV every 8 h	Vancomycin is not usually necessary for initial empiric antibiotic therapy if it is available for subsequent treatment modifications. Vancomycin may be incorporated into initial therapeutic regimens of high-risk patients in institutions with increased gram-positive infection rates.

**TABLE 139-2** Suggestions for Initial Empiric Antibiotic Therapy in Febrile Neutropenia

## **HYPERVISCOSITY SYNDROME**

Hyperviscosity syndrome refers to impaired blood flow due to abnormal elevations of paraproteins or cellular blood components. It most often occurs in patients with dysproteinemia, acute leukemia, or polycythemia. Hematocrits above 60% and WBC counts above  $> 100,000/\text{mm}^3$  often cause hyperviscosity syndromes.

Initial symptoms include fatigue, abdominal pain, headache, blurry vision, dyspnea, fever, or altered mental status. Thrombosis or bleeding may occur. Physical exam findings may include retinal hemorrhages, exudates, and “sausage-linked” vessels. Elevated serum viscosity ( $> 5$ ), rouleaux formation (red cells stacked like coins), or abnormal protein electrophoresis (IgM  $> 4$  grams/dL) support the diagnosis.

Initial therapy consists of intravenous **isotonic fluids** and **plasmapheresis or leukopheresis** in consultation with a hematologist. A temporizing measure in patients with coma is 1000 mL **phlebotomy** with simultaneous infusion of 2 to 3 L normal. Red cell transfusion is not recommended, as it may increase blood viscosity.

## **THROMBOEMBOLISM**

Thromboembolism is the second leading cause of death in cancer patients. See [Chapter 25](#) for discussion of the diagnosis and management of deep vein thrombosis and pulmonary embolism. Cancer patients, even those with brain metastases, do not appear at increased risk for anticoagulant-related bleeding complications.

## **NAUSEA AND VOMITING**

Chemotherapy commonly causes nausea and vomiting. Other causes of nausea and vomiting include radiation enteritis, bowel obstruction, infection or tumor infiltration, and increased intracranial pressure. Treatment consists of rehydration, administration of antiemetics, and correction of electrolyte derangements ([Table 139-3](#)).

Class and Agent	Adult Dose	Comments
Dopamine receptor antagonists		
Metoclopramide	10 milligrams IV or IM	Dose-related extrapyramidal side effects IV use common but not approved by FDA
Promethazine	25 milligrams IV or IM	
Serotonin antagonists		
Dolasetron	100 milligrams (or 1.8 milligrams/kilogram) IV over 5 min	Constipation, headaches (all)
Granisetron	10 micrograms/kilogram IV over 5 min	
Ondansetron	32 milligrams (or 0.15 milligram/kilogram) IV over 15 min	
Corticosteroids		
Dexamethasone	20 milligrams IV	Mechanism unknown, no immunosuppression
Benzodiazepines		
Lorazepam	1 to 2 milligrams IV	Sedation, anxiolysis
Histamine receptor antagonists		
Diphenhydramine	50 milligrams IV or IM	Minor therapeutic effect

Key: FDA = U.S. Food and Drug Administration.

**TABLE 139-3** Antiemetic Agents for Chemotherapy-Induced Vomiting

## EXTRAVASATION OF CHEMOTHERAPEUTIC AGENTS

If irritation develops during infusion through a peripheral line, stop the infusion and attempt aspiration through the line. Clinical manifestations of extravasation include pain, erythema and swelling, usually within hours of the infusion. Consult with the patient's oncologist to discuss the use of

antidotes for extravasation of anthracyclines, vinca alkaloids, mitomycin, cisplatin, mechlorethamine, and paclitaxel.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 235, “Emergency Complications of Malignancy,” by Paul Blackburn.

## **SECTION 15 Neurology**



# CHAPTER 140

## Headache and Facial Pain

Steven Go

Headache is a common complaint in the ED. The emergency physician's key goal is to detect life- or organ-threatening causes of headache.

### CLINICAL FEATURES

Headaches are divided into *primary* headaches and those due to *secondary* causes. Primary headache syndromes include the various types of migraine, tension-type, and cluster headaches, while secondary headaches have a legion of causes ([Table 140-1](#)). The particular clinical features of a given headache depend on the specific etiology.

Critical Secondary Causes	Reversible Secondary Causes	Primary Headache Syndromes
Vascular conditions	Noncentral nervous system infections	Migraine
Subarachnoid hemorrhage	Focal	Tension
Intraparenchymal hemorrhage	Systemic	Cluster
Epidural hematoma	Sinus	
Subdural hematoma	Odontogenic	
Stroke	Otic	
Cavernous/venous sinus thrombosis	Drug-related causes	
Arteriovenous malformation	Long-term analgesic use (medication overuse)	
Temporal arteritis	Monosodium glutamate	
Carotid or vertebral artery dissection	Miscellaneous causes	
Central nervous system infection	Postlumbar puncture	
Meningitis	Hypertensive emergency	
Encephalitis		
Cerebral abscess		
Tumor		
Pseudotumor cerebri		
Ophthalmic conditions		
Glaucoma		
Iritis		
Optic neuritis		
Drug-related causes		
Nitrates and nitrites		
Monoamine oxidase inhibitors		
Alcohol withdrawal		
Toxicity		
Carbon monoxide poisoning		
Endocrine conditions		
Pheochromocytoma		
Metabolic conditions		
Hypoxia		
Hypoglycemia		
Hypercapnia		
High-altitude cerebral edema		
Preeclampsia		

**TABLE 140-1** Etiology of Headache

## DIAGNOSIS AND DIFFERENTIAL

A careful history should be taken to elicit the headache pattern (constant, waxing, waning, different from previous headaches), onset (especially sudden onset, which is often a harbinger of a dangerous etiology), location, associated symptoms (syncope, altered level of consciousness, neck pain/

stiffness, persistent visual changes, fever, seizure), medications, toxic exposures (eg, carbon monoxide), relevant comorbidities (HIV, malignancy, coagulopathy, hypercoagulable state, hypertension), and family history (migraine, subarachnoid hemorrhage [SAH]).

Physical examination should be tailored to the differential. Relevant parts of the exam may include vital signs (fever), HEENT examination (sinuses, temporal arteries, slit lamp examination, funduscopy, tonometry, meningismus testing), and neurological examination (mental status, cranial nerves, motor and sensory function, reflexes, cerebellar exam, gait, and station).

The primary imaging modality for headache in the ED remains the noncontrast head CT, which usually excludes causes requiring emergent intervention, with the notable exception of subarachnoid hemorrhage. In 2008, the American College of Emergency Physicians (ACEP) made the following recommendations:

1. Patients presenting to the ED with headache and new abnormal findings in a neurologic examination (eg, focal deficit, altered mental status, altered cognitive function) should undergo emergent noncontrast head CT (Level B recommendation).
2. Patients presenting with new sudden-onset severe headache should undergo an emergent head CT (Level B recommendation).
3. HIV-positive patients with a new type of headache should be considered for an emergent neuroimaging study (Level B recommendation).
4. Patients who are older than 50 years and presenting with new type of headache but with a normal neurologic examination should be considered for an urgent (arranged prior to ED discharge) neuroimaging study (Level C recommendation).

Depending on the most likely diagnosis, other modalities, such as MRI (tumors, isodense subdural hemorrhages, cerebral venous thrombosis) and CT angiogram (strong suspicion of SAH with normal CT and lumbar puncture (LP) not possible, cervical artery dissection), may be of value in the ED.

LP is indicated to exclude meningitis or SAH (if the CT is normal). CT should precede LP if increased intracranial pressure is suspected.

**Subarachnoid hemorrhage** (also see [Chapter 141](#)) classically presents as the severe, sudden onset of “the worst headache of my life.” However, this presentation is not absolute. It is sometimes precipitated by exertion, but may also occur spontaneously. While neurological findings can occur, nearly 50% of neurological examinations in SAH patients are normal.

**Meningitis** (also see [Chapter 148](#)) can cause a headache of sudden onset, generally associated with fever and meningismus, and photophobia; however, the meningismus is often more subtle than many clinicians appreciate. Immunocompromised patients can experience a more insidious onset with opportunistic meningitis.

**Intraparenchymal hemorrhage** and **stroke** (also see [Chapter 141](#)) can present with headaches, approximately 50% and <25% of the time, respectively. Other neurological signs and symptoms are often present. Subdural hematoma (also see [Chapter 160](#)) headaches occur with remote trauma, usually in at risk patients (alcoholics, elders, and those on anticoagulants).

**Brain tumor**-associated headaches may be bilateral, unilateral, constant, or intermittent. The headache may be worse in the morning, associated with nausea and vomiting, and positional. Only 8% have neurological examination abnormalities.

**Cerebral venous thrombosis** presents with headache, vomiting, and seizures in patients with a hypercoagulable state (oral contraceptives, postpartum, perioperative, various clotting factor deficiencies, mutations, or polycythemia). Papilledema can be present, and neurological findings can wax and wane.

**Temporal arteritis** patients (also see [Chapter 149](#) and [Table 140-2](#)) present most commonly with headache (60% to 90%), which is most often unilateral, frontotemporal (can be bilateral), severe, and throbbing. Associated symptoms may include jaw claudication, polymyalgia rheumatica, URI symptoms, and vision changes. The involved temporal artery can be tender, nonpulsatile, or have a diminished pulse, but can also be normal.

Age >50 years
New-onset localized headache
Temporal artery tenderness or decreased pulse
Erythrocyte sedimentation rate >50 mm/h
Abnormal arterial biopsy findings

\*Three of the 5 criteria must be met.

**TABLE 140-2** Criteria for Diagnosis of Temporal Arteritis\*

**Ophthalmic disorders** (also see [Chapter 149](#)), such as acute glaucoma, can present with severe headache and are commonly associated with nausea and vomiting. These conditions can be diagnosed with a thorough eye examination, including measurement of intraocular pressures.

**Migraine headaches** generally have a gradual onset, last 4 to 72 hours, and are typically unilateral, pulsating, and worsened by physical activity. Nausea and vomiting, photophobia, and phonophobia are frequently present. Other neurological symptoms such as visual auras, hemiparesthesias, hemiparesis, and aphasia can be present, but if these cases are of new onset, migraine is a diagnosis of exclusion. Any change from the patient's typical migraine should raise the suspicion for other causes of headache.

**Cluster headaches** are rare (0.4% of population of the United States) and present with severe, unilateral orbital, supraorbital, or temporal pain lasting 15 to 180 min, frequently associated with lacrimation, nasal congestion, rhinorrhea, and conjunctival injection. These symptoms tend to occur daily for weeks, only to remit for weeks to years.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. In SAH, the chances of rebleeding can be reduced by maintaining the patient's prebleed blood pressure (or MAP <130 mm Hg if baseline blood pressure is unknown). This is best done by administering a titratable IV antihypertensive such as **labetalol** (typical adult starting dose is 10 to 20 milligrams over 1 to 2 min; continuous infusion dosage generally starts at 2 milligrams/min, titrated to effect). **Nimodipine** (60 milligrams PO every 4 hours) may produce modest improvements in outcome by

decreasing vasospasm. Emergent **neurosurgical consultation** is indicated.

2. Suspected meningitis patients should receive prompt empiric antibiotic therapy. Antibiotic therapy should not be delayed for LP or imaging.
3. Medications effective in the treatment of the patient with migraine headache include **dihydroergotamine (DHE)**, **sumatriptan**, and **dopamine-antagonist antiemetics (metoclopramide, chlorpromazine, prochlorperazine)**. Doses and considerations in the use of these agents are listed in [Table 140-3](#). Current guidelines recommend opiates only if migraine-specific therapy fails.

Drug	Dosing and Adjuncts	Contraindications (CI), Precautions (PC), and Notes
Dihydroergotamine	1 milligram IV over 3 min; pretreat with metoclopramide or chlorpromazine or prochlorperazine to reduce nausea and vomiting	CI: pregnancy, hypertension (uncontrolled), coronary artery disease, recent sumatriptan use, hemiplegic or basilar migraine PC: may cause nausea, vomiting, diarrhea, abdominal pain
Sumatriptan	6 milligrams SC	CI: pregnancy, hypertension (uncontrolled), coronary artery disease, ergot use in past 24 hours, monoamine oxidase inhibitor use, hemiplegic or basilar migraine PC: minor adverse effects; rarely, coronary artery spasm, myocardial infarction, dysrhythmias
Ketorolac	30 milligrams IV or 60 milligrams IM	CI: history of peptic ulcer disease (especially in the elderly) PC: pregnancy class B drug, avoid in third trimester
Chlorpromazine	Pretreat with normal saline bolus to minimize hypotension; 7.5 milligrams IV	PC: pregnancy class C drug, may cause hypotension, drowsiness, dystonic reactions <i>Note:</i> effective antiemetic

Prochlorperazine	5 to 10 milligrams IV or PR	PC: pregnancy class C drug, may cause drowsiness, dystonic reactions <i>Note:</i> effective antiemetic
Metoclopramide	10 milligrams IV	PC: pregnancy class B drug, may cause drowsiness, dystonic reactions <i>Note:</i> effective antiemetic
Droperidol	2.5 milligrams IV slow, or 2.5 milligrams IM	PC: cases of QT-interval prolongation and/or torsades de pointes have been reported
Olanzapine	10 milligrams IM	QT-interval prolongation
Magnesium sulfate	2 grams IV over 30 min	<i>Note:</i> nonvalidated but occasionally useful therapy (effective in eclampsia)
Methylprednisolone	125 milligrams IV or IM	<i>Note:</i> nonvalidated but occasionally useful rescue therapy
Dexamethasone	10 milligrams IV	<i>Note:</i> decreased headache reoccurrence when used as adjunctive therapy

**TABLE 140-3** Treatment Options for Migraine Headache

4. Cluster headaches will resolve with the administration of **high-flow oxygen** in 70% of patients. Dihydroergotamine mesylate, NSAIDs, and sumatriptan also may be effective; however, oral medications may be ineffective because of the length of time required for absorption and the short duration of the headache.
5. Indications for admission include presence of life-threatening cause of headache or failure to achieve adequate symptom control in the ED. Have a low threshold for specialty consultation when a serious cause for headache is suspected.
6. Discharge instructions should include red flags for the patient to monitor (fever, neck stiffness, vision changes, and new neurological symptoms/signs) as well as medication precautions if the patient received or is being prescribed medications that may cause drowsiness. Appropriate time for referral depends on the working diagnosis. For example, patients with suspected temporal arteritis without acute vision changes should see an ophthalmologist within 24 hours, whereas patients with a typical migraine could be referred nonemergently to a primary care physician.

## FACIAL PAIN

Temporomandibular disorder (TMD) causes pain at the temporomandibular joint, surrounding muscles, and ligaments. Patients often will complain of joint pain and noise with movement, locking of the jaw, limited jaw movements, and bruxism. ED treatment of TMD consists of **NSAIDs** and **narcotic analgesics**. Follow-up should be made with a dentist or oral surgeon.

Trigeminal neuralgia presents as an intermittent, seconds-long, “electric shock”-like pain in a unilateral trigeminal nerve distribution. Initial treatment may include **carbamazepine** (100 milligrams PO twice a day), which has been shown to be very effective. Refer patients to a neurologist for intractable cases.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 159, “Headache and Facial Pain,” by Christopher J. Denny and Michael J. Schull.



## CHAPTER 141

# Stroke, Transient Ischemic Attack, and Subarachnoid Hemorrhage

Steven Go

*Stroke* is defined as any disease process that interrupts blood flow to the brain. Ischemic strokes (87%) are more common than hemorrhagic intracerebral (10%) and a traumatic subarachnoid hemorrhage (SAH) (3%) ([Table 141-1](#)). A transient ischemic attack (TIA) is a transient neurologic deficit that typically lasts less than 1 to 2 hours, but duration can no longer be used to discriminate between TIA and stroke; they are best thought of as similar disease processes on a continuum.

Stroke Type	Mechanism	Major Causes	Clinical Notes
<b>Ischemic</b>			
Thrombotic	Narrowing of a damaged vascular lumen by an in situ process, usually clot formation	Atherosclerosis Vasculitis Arterial dissection Polycythemia Hypercoagulable state Infection (human immunodeficiency virus infection, syphilis, trichinosis, tuberculosis, aspergillosis)	Symptoms often have gradual onset and may wax and wane. Common cause of transient ischemic attack.
Embolic	Obstruction of a normal vascular lumen by intra-vascular material from a remote source	Valvular vegetations Mural thrombi Paradoxical emboli Cardiac tumors (myxomas) Arterial-arterial emboli from proximal source Fat emboli Particulate emboli (intravenous drug use) Septic emboli	Typically sudden in onset. Account for 20% of ischemic strokes.
Hypoperfusion	Low-blood flow	Cardiac failure	Diffuse injury pattern

state leading to hypoperfusion of the brain

resulting in systemic hypotension

in watershed regions. Symptoms may wax and wane with hemodynamic factors.

## Hemorrhagic

Intracerebral

Intraparenchymal hemorrhage from previously weakened arterioles

Hypertension  
Amyloidosis  
iatrogenic anticoagulation  
Vascular malformations  
Cocaine use

Intracranial pressure rise causes local neuronal damage. Secondary vasoconstriction mediated by blood breakdown products or neuronal mechanisms (diaschisis) can cause remote perfusion changes. Risks include advanced age, history of stroke, tobacco or alcohol use. More common in Asians and blacks.

Nontraumatic subarachnoid

Hemorrhage into subarachnoid space

Berry aneurysm rupture  
Vascular malformation rupture

May be preceded by a sentinel headache ("warning leak").

**TABLE 141-1** Stroke Classification

## CLINICAL FEATURES

Specific findings in stroke patients depend on regions of the brain that are compromised and the severity of the insult ([Table 141-2](#)). It is important to remember that stroke presentation can vary considerably from classically described syndromes.

Traditional symptoms	Sudden numbness or weakness of face, arm, or leg—especially unilateral Sudden confusion or aphasia Sudden memory deficit or spatial orientation or perception difficulties Sudden visual deficit or diplopia Sudden dizziness, gait disturbance, or ataxia Sudden severe headache with no known cause
Nontraditional symptoms	Loss of consciousness or syncope Shortness of breath Sudden pain in the face, chest, arms, or legs Seizure Falls or accidents Sudden hiccups Sudden nausea Sudden fatigue Sudden palpitations Altered mental status

**TABLE 141-2** Symptoms of Stroke

If the anterior cerebral artery is involved, the typical symptoms include contralateral leg weakness and sensory changes. A classic middle cerebral artery stroke presents with hemiparesis (arm > leg), facial plegia, and sensory loss. Weakness in the lower half of the face (variable) and ipsilateral gaze preference may occur. Aphasia (receptive and/or expressive) is often present if the dominant hemisphere (usually left) is affected while contralateral hemineglect suggests nondominant hemisphere involvement.

A posterior circulation stroke can present very subtly. Findings such as unilateral headache, visual field defects, dizziness, vertigo, diplopia, dysphagia, ataxia, cranial nerve deficits, or bilateral limb weakness can occur alone or in various combinations. Occlusion of the basilar artery causes severe quadriplegia, coma, and the locked-in syndrome. Cerebellar

strokes present similarly to other posterior stroke syndromes, but can deteriorate quickly if a hematoma or edema is present.

Cervical artery dissection can involve both the anterior and posterior arterial systems and can therefore present rather variably, but symptoms with an internal carotid dissection may include unilateral head pain (50% to 67%), neck pain (25%), or face pain (10%). Vertebral artery dissections may present with headache (69%) and posterior neck pain (46%), which can be unilateral or bilateral.

Intracranial hemorrhages may be clinically indistinguishable from cerebral infarction and may present with any of the anatomic syndromes discussed previously. SAH classically presents with sudden onset of a headache at its most severe. It occurs 20% of the time during activity associated with elevated blood pressures, such as sexual intercourse, weight lifting, defecation, or coughing. Vomiting, photophobia, nuchal irritation, low-grade fever, and altered mental status all may occur. Focal findings can occur depending on the location of the aneurysm. A recent history suggestive of a warning leak may be obtained in many patients. Symptoms may resolve spontaneously as blood diffuses in the SAH space; therefore, the clinician should not be misled by an improving clinical condition when the history is strongly suggestive of SAH.

## **DIAGNOSIS AND DIFFERENTIAL**

Stroke presents variably and sometimes subtly. Assess for stroke risk factors: elders, atrial fibrillation, hypertension, diabetes, smoking, coronary atherosclerotic disease, valvular replacement, and recent myocardial infarction. Causative mechanisms (eg, stroke symptoms or headaches after chiropractic manipulation) should be elicited. Because of the plethora of possible presentations, stroke mimics abound ([Table 141-3](#)) and reasonable efforts should be made to exclude them. Most importantly, an accurate determination of the time the patient was last known to be at their neurological baseline is essential. Finally, determine eligibility for thrombolytic therapy if stroke is the primary working diagnosis ([Tables 141-4](#) and [141-5](#)).

Stroke Mimic	Distinguishing Clinical Features
Seizures/postictal paralysis (Todd paralysis)	Transient paralysis following a seizure, which typically disappears quickly; can be confused with transient ischemic attack. Seizures can be secondary to a cerebrovascular accident.
Syncope	No persistent or associated neurologic symptoms.
Brain neoplasm or abscess	Focal neurologic findings, signs of infection, detectable by imaging.
Epidural/subdural hematoma	History of trauma, alcoholism, anticoagulant use, bleeding disorder; detectable by imaging.
Subarachnoid hemorrhage	Sudden onset of severe headache.
Hypoglycemia	Can be detected by bedside glucose measurement, history of diabetes mellitus.
Hyponatremia	History of diuretic use, neoplasm, excessive free water intake.
Hypertensive encephalopathy	Gradual onset; global cerebral dysfunction, headache, delirium, hypertension, cerebral edema.
Meningitis/encephalitis	Fever, immunocompromise may be present, meningismus, detectable on lumbar puncture.
Hyperosmotic coma	Extremely high glucose levels, history of diabetes mellitus.

Wernicke encephalopathy	History of alcoholism or malnutrition; triad of ataxia, ophthalmoplegia, and confusion.
Labyrinthitis	Predominantly vestibular symptoms; patient should have no other focal findings; can be confused with cerebellar stroke.
Drug toxicity (lithium, phenytoin, carbamazepine)	Can be detected by particular toxidromes and elevated blood levels. Phenytoin and carbamazepine toxicity may present with ataxia, vertigo, nausea, and abnormal reflexes.
Bell palsy	Neurologic deficit confined to isolated peripheral seventh nerve palsy; often associated with younger age.
Complicated migraine	History of similar episodes, preceding aura, headache.
Ménière disease	History of recurrent episodes dominated by vertigo symptoms, tinnitus, deafness.
Demyelinating disease (multiple sclerosis)	Gradual onset. Patient may have a history of multiple episodes of neurologic findings in multifocal anatomic distributions.
Conversion disorder	No cranial nerve findings, nonanatomic distribution of findings (eg, midline sensory loss), inconsistent history or examination findings.

**TABLE 141-3** Differential Diagnoses of Consequence for Acute Stroke Symptoms

## Indications

Measurable diagnosis of acute ischemic stroke

Use of NIHSS recommended. Stroke symptoms should not be clearing, minor, or isolated. Caution is advised before giving rt-PA to persons with severe stroke (NIHSS score of >22), because they have increased risk of intracerebral hemorrhage; however, they are at high risk of death, regardless.

Age  $\geq$  18 y

No clear upper age limit.

Time of symptom onset  $\leq$  3 h

Must be *well established* (2009 AHA/ASA Scientific Advisory suggests time window may be extended to 3 to 4.5 h if ECASS criteria are met).

## Exclusion Criteria

Symptoms consistent with subarachnoid hemorrhage

Seizure with postictal residual neurologic impairments

Previous head trauma or stroke within preceding 3 months

Previous myocardial infarction within preceding 3 months\*

Previous GI or urinary tract hemorrhage within preceding 21 days

Major surgery within preceding 14 days



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Prior intracranial hemorrhage

---

Pretreatment systolic blood pressure > 185 mm Hg *or* diastolic blood pressure > 110 mm Hg despite therapy (Table 161-8)

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Evidence of active bleeding or acute major fracture

---

Blood glucose level < 50 milligrams/dL (2.7 mmol/L)

---

International normalized ratio > 1.7 (oral anticoagulant use in and of itself is not a contraindication to rt-PA)

---

Use of heparin within preceding 48 h *and* a prolonged activated partial thromboplastin time  
Platelet count < 100 000/mm<sup>3</sup>

---

Head CT shows multilobar infarction (hypodensity of more than one-third cerebral hemisphere) or hemorrhage or tumor

---

Failure of the patient or responsible party to understand the risks and benefits of, and alternatives to, the proposed treatment after a full discussion

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Key: NIHSS = National Institutes of Health Stroke Scale.

\*Rationale for this criterion was a statement indicating that myocardial rupture can result if rt-PA is given within a few days of acute myocardial infarction.

**TABLE 141-4** American Heart Association/American Stroke Association 2007 Criteria for IV Recombinant Tissue Plasminogen Activator (rt-PA) in Acute Ischemic Stroke

Age >80 years  
Severe stroke as assessed clinically (NIHSS score >25)  
Combination of previous stroke and diabetes mellitus  
Blood glucose <50 milligrams/dL or >400 milligrams/dL  
Oral anticoagulant treatment

**TABLE 141-5** Additional Exclusion Criteria for IV Recombinant Tissue Plasminogen Activator (rT-PA) in Acute Ischemic Stroke when Given 3 to 4.5 h after Symptom Onset

Focus the physical exam on the neurological examination, with particular emphasis on detecting meningismus, signs of emboli, papilledema, or preretinal hemorrhage. Calculate a National Institutes of Health Stroke Scale (NIHSS) score upon presentation ([Table 141-6](#)).

## Instructions

## Scale Definition

### 1a. Level of consciousness (LOC)

The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, or orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation.

0 = Alert, keenly responsive

1 = Not alert, but arousable by minor stimulation to obey, answer, or respond.

2 = Not alert; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped).

3 = Responds only with reflex motor or autonomic effects or is totally unresponsive, flaccid, and areflexic.

### 1b. LOC questions

The patient is asked the month and his or her age. The answer must be correct—there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions are given a score of 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a score of 1. It is important that only the initial answer be graded and that the examiner not “help” the patient with verbal or nonverbal cues.

0 = Answers both questions correctly.

1 = Answers one question correctly.

2 = Answers neither question correctly.

### 1c. LOC commands

The patient is asked to open and close the eyes and then to grip and release the nonparetic hand. Substitute another one-step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime) and the result scored (i.e., follows no, one, or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.

0 = Performs both tasks correctly.

1 = Performs one task correctly.

2 = Performs neither task correctly.

## 2. Best gaze

Only horizontal eye movements are tested. Voluntary or reflexive (oculocephalic) eye movements are scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score is 1. If a patient has an isolated peripheral nerve palsy (cranial nerve II, IV, or VI), the score is 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, preexisting blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.

0 = Normal.

1 = Partial gaze palsy; gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present.

2 = Forced deviation, or total gaze paresis not overcome by the oculocephalic maneuver.

## 3. Visual

Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If the patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a score of 1, and the results are used to respond to item 11.

0 = No vision loss.

1 = Partial hemianopia.

2 = Complete hemianopia.

3 = Bilateral hemianopia (blind including cortical blindness).

## 4. Facial palsy

Ask, or use pantomime to encourage, the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or noncomprehending patient. If facial trauma/bandages, orotracheal tube, tape, or other physical barriers obscure the face, these should be removed to the extent possible.

0 = Normal symmetric movements.

1 = Minor paralysis (flattened nasolabial fold, asymmetry on smiling).

2 = Partial paralysis (total or near-total paralysis of lower face).

3 = Complete paralysis of one or both sides (absence of facial movement in the upper and lower face).

### 5. Motor arm

The limb is placed in the appropriate position: extend the arms (palms down) 90° (if sitting) or 45° (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the nonparetic arm. Only in the case of amputation or joint fusion at the shoulder, the examiner should record the score as unstable (UN) and clearly write the explanation for this choice.

5a. Left arm

5b. Right arm

0 = No drift; limb holds 90° (or 45°) for full 10 seconds.

1 = Drift; limb holds 90° (or 45°), but drifts down before full 10 seconds; does not hit bed or other support.

2 = Some effort against gravity; limb cannot get to or maintain (if cued) 90° (or 45°), drifts down to bed, but has some effort against gravity.

3 = No effort against gravity; limb falls.

4 = No movement.

### 6. Motor leg

The limb is placed in the appropriate position: hold the leg at 30° (the patient is always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the nonparetic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as unstable (UN) and clearly write the explanation for this choice.

6a. Left leg

6b. Right leg

0 = No drift; leg holds 30° position for full 5 seconds.

1 = Drift; leg falls by the end of the 5-second period but does not hit bed.

2 = Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity.

3 = No effort against gravity; leg falls to bed immediately.

4 = No movement.

### 7. Limb ataxia

This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with the patient's eyes open. In case of visual defect, ensure that testing is done in the intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as unstable (UN) and clearly write the explanation for this choice. In case of blindness, test by having the patient touch the nose from an extended arm position.

0 = Absent.

1 = Present in 1 limb.

2 = Present in 2 limbs.

## 8. Sensory

Sensation of grimace to pinprick when tested, or withdrawal from a noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal, and the examiner should test as many body areas (arms (not hands), legs, trunk, face) as needed to check accurately for hemisensory loss. A score of 2, "severe or total sensory loss," should be given only when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will therefore probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2. Patients in a coma (item 1a score = 5) are automatically given a 2 on this item.

0 = Normal, no sensory loss.

1 = Mild-to-moderate sensory loss; patient feels pinprick is less sharp or is dull on affected side, or there is loss of superficial pain with pinprick, but patient is aware of being touched.

2 = Severe to total sensory loss; patient is not aware of being touched on face, arm, and leg.

## 9. Best language

A great deal of information about comprehension is obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the test picture, to name the items on the test naming sheet, and to read from the test list of sentences. Comprehension is judged from responses here as well as responses to all of the commands in the preceding general neurologic examination. If vision loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a score = 5) automatically scores 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands.

0 = No aphasia, normal.

1 = Mild-to-moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. However, reduction of speech and/or comprehension makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response.

2 = Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient's response.

3 = Mute; global aphasia; no usable speech or auditory comprehension.

### 10. Dysarthria

If the patient is thought to be normal, an adequate sample of speech must be obtained by asking the patient to read or repeat words from the test list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN) and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.

0 = Normal

1 = Mild-to-moderate dysarthria; patient says at least some words and, at worst, can be understood with some difficulty.

2 = Severe dysarthria; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/aphasic.

### 11. Extinction and inattention

Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe vision loss preventing visual double simultaneous stimulation and the responses to cutaneous stimuli are normal, the score is 0. If the patient has aphasia but does appear to attend to both sides, the score is 0. The presence of visual spatial neglect or anosognosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never scored as UN.

0 = No abnormality.

1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.

2 = Profound hemi-inattention or extinction in more than one modality; patient does not recognize own hand or orients to only 1 side of space.

\*Item deleted from modified NIHSS.

Scale for item 11 is compressed to two elements (0 = Normal; 1 = Abnormal) for modified NIHSS.

Sources: Stroke Scales and Related Information, National Institute of Neurological Disorders and Stroke. Available at: [http://www.ninds.nih.gov/ctors/NIH\\_Stroke\\_Scale.pdf](http://www.ninds.nih.gov/ctors/NIH_Stroke_Scale.pdf). Accessed March 24, 2008, and Meyer BC, Herrmen TM, Jackson CM, Lyden PT. Modified National Institutes of Health Stroke Scale for use in stroke clinical trials: prospective reliability and validity. *Stroke* 33(5):1261, 2002.

## TABLE 141-6 National Institutes of Health Stroke Scale (NIHSS)

Because stroke treatment is time sensitive, the history or physical examination should not be unduly prolonged.

An emergent noncontrast CT scan (best interpreted by a neuroradiologist) is essential to determine whether hemorrhage or a stroke mimic is present. Most acute ischemic strokes will not be visualized in the early hours of a stroke.

The differential diagnosis for SAH is broad ([Table 141-7](#)). Modern CT scanners are 98% sensitive to detect SAH within 12 hours of symptom onset. If SAH is suspected and the CT is negative, most authorities agree that a lumbar puncture is indicated. CSF xanthochromia does not develop until 12 hours after symptom onset and the threshold number of RBCs needed in the CSF to be considered diagnostic of SAH is still unclear. A normal head CT, no xanthochromia, and zero or few RBCs ( $<5 \times 10^6$  RBCs/L) is generally considered to exclude SAH. A growing body of literature is exploring the combination of CT/CT angiography to exclude SAH, but no definitive data on the sensitivity of this combination have been found to date.

Other intracranial hemorrhage
Drug toxicity
Ischemic stroke
Meningitis
Encephalitis
Intracranial tumor
Intracranial hypotension
Metabolic derangements
Venous thrombosis
Primary headache syndromes (benign thunderclap headache, migraine, cluster headache)

**TABLE 141-7** Differential Diagnosis of Subarachnoid Hemorrhage

Other diagnostic tests that may be useful in certain patients to exclude stroke mimics or concurrent conditions include a complete blood count, ECG, pulse oximetry, electrolyte and coagulation studies, cardiac enzyme levels, toxicology screen, blood alcohol level, echocardiogram, carotid duplex scanning. MRI, MRA, and CT angiogram may be of value in detecting particular disease entities (eg, cervical artery dissection, tumor, SAH).

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Rapidly assess and stabilize any airway, breathing, and circulation abnormalities. Keep oxygen saturation  $\geq 92\%$ .



2. Establish IV access while the patient is placed on a cardiac monitor.
3. Obtain a rapid bedside glucose and normalize any hypoglycemia.
4. Keep the patient npo.
5. Once the patient's condition is stabilized, immediately send the patient for a noncontrast head CT.
6. Hypertension management in acute ischemic stroke is an area in flux. In general, if a patient is not a candidate for thrombolysis, then permissive hypertension is in order (no intervention unless systolic blood pressure (SBP) > 220 mm Hg or diastolic blood pressure (DBP) > 120 mm Hg). If blood pressure control is needed, use a titratable IV antihypertensive, such as **labetalol** (typical starting dose is 10 to 20 milligrams over 1 to 2 min; continuous infusion dosage generally starts at 2 milligrams/min, titrated to effect) with a target MAP reduction of 10% to 25%. Take extreme caution to avoid overcorrection.
7. If a patient is a candidate for thrombolytics, then the target blood pressures are SBP ≤ 185 mm Hg and DBP ≤ 110 mm Hg. For management of hypertensive patients who are potentially thrombolytic candidates, see [Table 141-8](#).

If the patient is a candidate for rt-PA therapy, the target arterial blood pressures are: **systolic blood pressure  $\leq$  185 mm Hg and diastolic blood pressure  $\leq$  110 mm Hg**

Drug	Comments
Labetalol, 10 to 20 milligrams IV over 1 to 2 min, may repeat $\times$ 1	Use with caution in patients with severe asthma, severe chronic obstructive pulmonary disease, congestive heart failure, diabetes mellitus, myasthenia gravis, concurrent calcium channel blocker use, hepatic insufficiency. May cause dizziness and nausea. Pregnancy category C (D in second and third trimesters).
<i>or</i> Nitroglycerin paste, 1 to 2 inch to skin	Contraindicated in patients with hypersensitivity to organic nitrates, concurrent use of phosphodiesterase 5 inhibitors (sildenafil, tadalafil, or vardenafil), or angle-closure glaucoma. Increases intracranial pressure. Commonly causes headache. Pregnancy category C.
<i>or</i> Nicardipine infusion, 5 milligrams/h, titrate up by 2.5 milligrams/h at 5 to 15 min intervals; maximum dose, 15 milligrams/h; when desired blood pressure attained, reduce to 3 milligrams/h	Use with caution in patients with myocardial ischemia, concurrent use of fentanyl (hypotension), congestive heart failure, hypertrophic cardiomyopathy, portal hypertension, renal insufficiency, hepatic insufficiency (may need to adjust starting dose). Contraindicated in patients with severe aortic stenosis. Can cause headache, flushing, dizziness, nausea, reflex tachycardia. Pregnancy category C.

If the target arterial blood pressures for rt-PA administration cannot be reached with these initial measures, then *the patient is no longer a candidate for rt-PA therapy.*

**TABLE 141-8** Approach to Management of Arterial Hypertension before Potential Administration of Recombinant Tissue Plasminogen Activator (rt-PA)

- The US Food and Drug Administration has approved the use of IV recombinant tissue plasminogen activator (**rt-PA**) for acute ischemic stroke  $\leq$  3 hours of symptom onset. Based on the European Cooperative Acute Stroke Study III, FDA approval for expansion of the rt-PA treatment window to 4.5 hours is under consideration at the time of this writing. A careful review of rt-PA inclusion and exclusion criteria must be meticulously performed prior to administration of rt-PA ([Table 141-4](#)). If the therapeutic window is to be extended to 3 to 4.5 hours, then use the additional ECASS III exclusion criteria ([Table 141-5](#)). Determine the

precise time when the patient was last known to be at their neurological baseline.

9. Obtain informed consent from the patient or their designee prior to thrombolytic therapy. Although thrombolytic treatment of ischemic stroke is associated with improved outcomes, the risk of symptomatic intracerebral hemorrhage (SIH) is 6.5% (45% mortality) when rt-PA is given within  $\leq 3$  hours of symptom onset, and 7.9% (NINDS definition) between 3 and 4.5 hours.
10. **The total dose of rt-PA is 0.9 milligram/kilogram IV, with a maximum dose of 90 milligrams; 10% of the dose is administered as a bolus, with the remaining amount infused over 60 min.** No aspirin or heparin should be administered in the initial 24 hours after treatment. Intracerebral bleeding should be suspected as the cause of any neurologic worsening.
11. Closely monitor blood pressures for patients who receive rt-PA and treat as necessary (see [Table 141-9](#)).

Blood Pressure Monitoring Frequencies	
Time after start of rt-PA infusion	Frequency of blood pressure monitoring
0 to 3 h	Every 15 min
3 to 9 h	Every 30 min
9 to 24 h	Every 60 min
Drug Treatment of Hypertension during and after Administration of rt-PA	
If systolic blood pressure is 180 to 230 mm Hg <i>or</i> Diastolic blood pressure is 105 to 120 mm Hg	Labetalol, 10 milligrams IV over 1 to 2 min. The dose may be repeated every 10 to 20 min up to a total dose of 300 milligrams. <i>or</i> Labetalol, 10 milligrams IV followed by infusion at 2 to 8 milligrams/min.
If systolic blood pressure is >230 mm Hg <i>or</i> Diastolic blood pressure is 121 to 140 mm Hg	Labetalol, 10 milligrams IV over 1 to 2 min. The dose may be repeated every 10 to 20 min up to a total dose of 300 milligrams. <i>or</i> Labetalol, 10 milligrams IV followed by infusion at 2 to 8 milligrams/min. <i>or</i> Nicardipine infusion, 5 milligrams/h, titrate up by 2.5 milligrams/h at 5 to 15 min intervals; maximum dose 15 milligrams/h.
If blood pressure is not controlled by above measures	Consider sodium nitroprusside infusion (0.5 to 10 micrograms/kilogram/min). Continuous arterial monitoring advised; use with caution in patients with hepatic or renal insufficiency. Increases intracranial pressure. Pregnancy category C.

**TABLE 141-9** Management of Blood Pressure during and after Administration of Recombinant Tissue Plasminogen Activator (rt-PA)

12. For TIA patients, **aspirin** (325 milligrams PO) plus **dipyridamole** (400 milligrams PO) is recommended. However, for stroke patients, aspirin (325 milligrams PO) is recommended within 24 to 48 hours. Aspirin does not interfere with subsequent consideration for thrombolytic therapy. Antiplatelet therapy is contraindicated for hemorrhagic stroke.
13. There is currently no role for heparin or warfarin in the acute treatment of TIA or stroke in the ED, even in the presence of atrial fibrillation. A possible exception exists for a weight-based heparin protocol in cervical artery dissection, but this remains controversial and neurology consultation is advisable for these cases.
14. If an ischemic stroke patient presents outside the rt-PA therapeutic time window, then provide aggressive supportive care in the ED (aspiration

prevention, normalization of glucose level, fall precautions, treatment for comorbidities).

15. For patients with evidence of increased intracranial pressure (ICP), head elevation to 30°, analgesia, and sedation is needed. If more aggressive ICP reduction is indicated, mannitol (0.25 to 1.0 gram/ kilogram IV), intubation with neuromuscular blockade with mild hyperventilation, and invasive monitoring of ICP may be required.
16. In SAH, the chances of rebleeding can be reduced by maintaining the patient's prebleed blood pressure (or MAP <130 mm Hg if baseline blood pressure is unknown). This is best done by administering an IV titratable antihypertensive such as **labetalol** (typical adult starting dose is 10 to 20 milligrams over 1 to 2 min; continuous infusion dosage generally starts at 2 milligrams/min, titrated to effect). **Nimodipine** (60 milligrams PO every 4 hours) may produce modest improvements in outcome by decreasing vasospasm. Emergent neurosurgical consultation is indicated. Administer medications and antiemetics as needed. Seizure prophylaxis is controversial and should be discussed with the specialist who will manage the patient after they leave the ED.
17. Management of blood pressure for spontaneous intracerebral hemorrhage remains controversial.
18. Emergent neurology consultation may be helpful in difficult stroke cases where thrombolytics are a consideration; however, therapy should not be unduly delayed while waiting for a response. Early neurosurgical consultation is indicated for patients with intracerebral hemorrhage with evidence of increased ICP or in other conditions where surgical intervention may be indicated. For example, cerebellar stroke mandates a neurosurgical consultation because swelling with compression of the brainstem may lead to rapid deterioration; posterior fossa decompression may be life saving.
19. Admit all patients with acute ischemic stroke or intracerebral hemorrhage, even if they are not candidates for interventional therapy. Admission to specialized stroke units is associated with improved outcomes for stroke patients; therefore, transfer to a designated stroke center may be indicated if the patient presents to a nonstroke center.
20. The **ABCD<sup>2</sup> scoring system** may be used to predict stroke risk in TIA patients ([Table 141-10](#)). Using this system, the 2-day risks of subsequent

stroke are: 1% (ABCD<sup>2</sup> score 0 to 3); 4.1% (4 to 5); and 8.1% (6 to 7)). Because of the proven efficacy of early carotid endarterectomy, many stroke experts recommend admission for most TIA patients for inpatient evaluation and observation. In select low-risk, asymptomatic patients, next day follow-up and evaluation with a specialist may be appropriate, but responsible adults to observe the patient in a favorable social situation must be available and very strong return precautions given.

Criteria	Points
Age ≥ 60 y	0 = Absent 1 = Present
Blood pressure ≥ 140/90 mm Hg	0 = Absent 1 = Present
Clinical features	0 = Absent 1 = Speech impairment without unilateral weakness 2 = Unilateral weakness (with or without speech impairment)
Duration	0 = Absent 1 = 10 to 59 min 2 = ≥ 60 min
Diabetes	0 = Absent 1 = Present

Adapted with permission from Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al: Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. *Lancet* 369(9558): 283, 2007.

**TABLE 141-10** ABCD<sup>2</sup> Score to Predict Very Early Stroke Risk after Transient Ischemic Attack

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 160, “Spontaneous Subarachnoid and Intracerebral Hemorrhage,” by Jeffrey L. Hackman, Melissa D. Johnson, and O. John Ma; and Chapter 161, “Stroke, Transient Ischemic Attack, and Cervical Artery Dissection,” by Steven Go and Daniel J. Worman.

# CHAPTER 142

## Altered Mental Status and Coma

C. Crawford Mechem

Mental status is the clinical state of emotional and intellectual functioning of the individual. Presentations of altered mental status in the ED include delirium, dementia, and coma.

### DELIRIUM

#### Clinical Features

Delirium is a transient disorder characterized by impaired attention, perception, memory, and cognition. Sleep-wake cycles may be disrupted, with increased somnolence during the day and agitation at night (“sundowning”). Alertness is reduced. Activity levels may fluctuate rapidly. Different care-givers may witness completely different behaviors within a brief time span. Tremor, asterixis, tachycardia, sweating, hypertension, emotional outbursts, and hallucinations may be present. Features of delirium, dementia, and psychiatric causes are listed in [Table 142-1](#).

Characteristic	Delirium	Dementia	Psychiatric Disorder
Onset	Over days	Insidious	Sudden
Course over 24 h	Fluctuating	Stable	Stable
Consciousness	Reduced or hyperalert	Alert	Alert
Attention	Disordered	Normal	May be disordered
Cognition	Disordered	Impaired	May be impaired
Orientation	Impaired	Often impaired	May be impaired
Hallucinations	Visual and/or auditory	Often absent	Usually auditory
Delusions	Transient, poorly organized	Usually absent	Sustained
Movements	Asterixis, tremor may be present	Often absent	Absent

**TABLE 142-1** Features of Delirium, Dementia, and Psychiatric Disorder

## Diagnosis and Differential

The acute onset of attention deficits and cognitive abnormalities fluctuating throughout the day and worsening at night is virtually diagnostic. A detailed medication history should be obtained. ED evaluation is directed at identifying an underlying process, such as infection. Ancillary tests include basic metabolic panel, hepatic studies, urinalysis, complete blood count, and chest radiograph. Cranial CT should be performed if a mass lesion is suspected, followed by lumbar puncture if meningitis or subarachnoid hemorrhage is a consideration. The possible causes of delirium in the elderly are listed in [Table 142-2](#).

Infectious	Pneumonia Urinary tract infection Meningitis or encephalitis Sepsis
Metabolic/toxic	Hypoglycemia Alcohol ingestion Electrolyte abnormalities Hepatic encephalopathy Thyroid disorders Alcohol or drug withdrawal
Neurologic	Stroke or transient ischemic attack Seizure or postictal state Subarachnoid hemorrhage Intracranial hemorrhage Central nervous system mass lesion Subdural hematoma
Cardiopulmonary	Congestive heart failure Myocardial infarction Pulmonary embolism Hypoxia or CO <sub>2</sub> narcosis
Drug-related	Anticholinergic drugs Alcohol or drug withdrawal Sedatives-hypnotics Narcotic analgesics Polypharmacy

**TABLE 142-2** Important Medical Causes of Delirium in Elderly Patients



## **Emergency Department Care and Disposition**

1. Direct treatment at the underlying cause. Protect the patient while the workup proceeds. Consider restraining the patient, as needed. Environmental manipulation such as adequate lighting and emotional support may put the patient at ease.
2. Treat agitation with **haloperidol**, 5 to 10 milligrams PO, IM, or IV, with reduced dosing of 1 to 2 milligrams in the elderly. **Lorazepam**, 0.5 to 2 milligrams PO, IM, or IV, may be used in combination with haloperidol in doses of 1 to 2 milligrams.

Admit patients for further care unless a readily reversible cause for the acute mental status change is discovered, treatment is initiated, and improvement is seen.

## **DEMENTIA**

### **Clinical Features**

Dementia implies a loss of mental capacity. Psychosocial level and cognitive abilities deteriorate and behavioral problems develop. The largest categories of dementia are Alzheimer disease and vascular dementia. Onset is insidious. Hallucinations, delusions, repetitive behaviors, and depression are common, as is impairment of memory, particularly recent memory. Other features of dementia include naming problems, forgetting items, loss of reading and direction, disorientation, inability to perform self-care tasks, and personality changes. Anxiety and speech difficulties may be observed. Patients with vascular dementia may be noted to have exaggerated or asymmetric deep tendon reflexes, gait abnormalities, or extremity weakness.

### **Diagnosis and Differential**

Alzheimer disease usually develops slowly. Abrupt worsening suggests vascular dementia. Physical examination may identify a precipitant or underlying cause. Focal neurologic signs suggest vascular dementia or mass lesion. A fluctuating, stepped course also suggests vascular dementia. Increased motor tone and other extrapyramidal signs suggest Parkinson disease. Diagnostic studies may include a complete blood count, basic metabolic profile, urinalysis, thyroid profile, serum vitamin B<sub>12</sub> level, testing for syphilis, erythrocyte sedimentation rate, serum folate level, human immunodeficiency virus testing, and chest radiography. Consider head CT or MRI as well as lumbar puncture if the diagnosis is not readily apparent. The differential diagnosis includes delirium, depression, and other treatable causes.

## **Emergency Department Care and Disposition**

Identify any treatable causes or precipitating disease processes.

1. Use antipsychotic drugs to manage persistent psychosis or severely disruptive or dangerous behavior. Their use is associated with adverse reactions.
2. Aim treatment of vascular dementia at addressing risk factors, such as hypertension.
3. Consider normal pressure hydrocephalus if urinary incontinence and gait disturbance are noted. This is further suggested by excessively large ventricles on head CT. Consider lumbar puncture with cerebrospinal fluid drainage.

Most patients with newly diagnosed dementia require admission for further evaluation and treatment. Discharge those who have longstanding and stable symptoms, consistent caregivers, and reliable follow-up for outpatient evaluation after life-threatening conditions have been excluded.

## **COMA**

## Clinical Features

Coma is a state of reduced alertness and responsiveness from which the patient cannot be aroused. Severity can be quantified using the Glasgow Coma Scale ([Table 142-3](#)). Pupillary findings, other cranial nerve evaluation, hemiparesis, and response to stimulation can assign the cause into a probable general category: diffuse (toxic-metabolic coma) or focal (structural coma) CNS dysfunction. Structural coma is divided into hemispheric (supratentorial) or posterior fossa (infratentorial) coma. Toxic-metabolic coma is characterized by lack of focal physical examination findings. The pupils are typically small and reactive, but may be large in severe sedative poisoning as from barbiturates. Coma from supratentorial lesions or masses may present with progressive hemiparesis or asymmetric muscle tone and reflexes. Coma without lateralizing signs may result from decreased cerebral perfusion from increased ICP. Reflex changes in blood pressure and heart rate may be observed, such as the Cushing reflex (hypertension and bradycardia) from increased ICP. Coma from posterior fossa or infratentorial lesions may cause abrupt coma, abnormal extensor posturing, and loss of pupillary reflexes and extraocular movements. Brainstem compression with loss of brainstem reflexes may develop rapidly. Pontine hemorrhage, another infratentorial cause of coma, may present with pinpoint pupils. Pseudocomma or psychogenic coma is a diagnostic challenge. History taking and observation of responses to stimulation reveal findings that differ from typical syndromes. Pupillary responses, extraocular movements, muscle tone, and reflexes are intact. Valuable tests include responses to manual eye opening (there should be little or no resistance in the truly unresponsive patient) and extraocular movements. If avoidance of gaze is consistently seen with the patient always looking away from the examiner, or if nystagmus is demonstrated with caloric vestibular testing, this is strong evidence for nonphysiologic or feigned unresponsiveness.

Component	Score	Adult	Child <5 years	Child >5 years
Motor	6	Follows commands	Normal spontaneous movements	Follows commands
	5	Localizes pain	Localizes to supraocular pain (>9 months)	
	4	Withdraws to pain	Withdraws from nail bed pressure	
	3	Flexion	Flexion to supraocular pain	
	2	Extension	Extension to supraocular pain	
	1	None	None	
Verbal	5	Oriented	Age-appropriate speech/vocalizations	Oriented
	4	Confused speech	Less than usual ability; irritable cry	Confused
	3	Inappropriate words	Cries to pain	Inappropriate words
	2	Incomprehensible	Moans to pain	Incomprehensible
	1	None	No response to pain	
Eye opening	4	Spontaneous	Spontaneous	
	3	To command	To voice	
	2	To pain	To pain	
	1	None	None	

**TABLE 142-3** Glasgow Coma Scale

## Diagnosis and Differential

History, examination, laboratory studies, and neuroimaging will usually identify the cause. Abrupt coma suggests stroke or seizure. Gradual onset suggests a metabolic process or progressive lesion such as a tumor or bleed. Examination may reveal signs of trauma or suggest other possibilities, such as toxidromes. Fine neurologic testing is not feasible, but asymmetric findings on pupillary examination, assessment of corneal reflexes, and testing of oculovestibular reflexes may suggest focal lesions. Asymmetric muscle tone or reflexes also suggest a focal lesion. Extensor or flexor posturing suggests profound CNS dysfunction. A head CT should be obtained, followed by lumbar puncture if the scan is unremarkable and a bleed or infection is suspected. Basilar artery thrombosis is a concern in a comatose patient with a “normal” head CT; MRI or cerebral angiography is needed to make the diagnosis. Patients who have had seizures and remain

unresponsive may be having electrical seizures without motor activity. Perform an EEG if this is suspected. Consider toxic ingestions, infections, and nonaccidental trauma in comatose children. The differential diagnosis of coma includes generalized disease processes that also affect the brain and primary CNS disorders ([Table 142-4](#)).

<p><b>Coma from causes affecting the brain diffusely</b></p> <ul style="list-style-type: none"> <li>Encephalopathies <ul style="list-style-type: none"> <li>Hypoxic encephalopathy</li> <li>Metabolic encephalopathy <ul style="list-style-type: none"> <li>Hypoglycemia</li> <li>Hyperosmolar state (eg, hyperglycemia)</li> <li>Electrolyte abnormalities (eg, hyponatremia or hyponatremia, hypercalcemia)</li> </ul> </li> <li>Organ system failure <ul style="list-style-type: none"> <li>Hepatic encephalopathy</li> <li>Uremia/renal failure</li> <li>Endocrine (eg, Addison disease, hypothyroidism, etc.)</li> </ul> </li> <li>Hypoxia <ul style="list-style-type: none"> <li>CO<sub>2</sub> narcosis</li> </ul> </li> <li>Hypertensive encephalopathy</li> </ul> </li> <li>Toxins <ul style="list-style-type: none"> <li>Drug reactions (eg, neuroleptic malignant syndrome)</li> <li>Environmental causes—hypothermia, hyperthermia</li> <li>Deficiency state—Wernicke encephalopathy</li> </ul> </li> <li>Sepsis</li> </ul>
<p><b>Coma from primary CNS disease or trauma</b></p> <ul style="list-style-type: none"> <li>Direct CNS trauma <ul style="list-style-type: none"> <li>Diffuse axonal injury</li> <li>Subdural hematoma</li> <li>Epidural hematoma</li> </ul> </li> <li>Vascular disease <ul style="list-style-type: none"> <li>Intraparenchymal hemorrhage (hemispheric, basal ganglia, brainstem, cerebellar)</li> <li>Subarachnoid hemorrhage</li> </ul> </li> <li>Infarction <ul style="list-style-type: none"> <li>Hemispheric, brainstem</li> </ul> </li> <li>CNS infections</li> <li>Neoplasms</li> <li>Seizures <ul style="list-style-type: none"> <li>Nonconvulsive status epilepticus</li> <li>Postictal state</li> </ul> </li> </ul>

Key: CNS = central nervous system.

**TABLE 142-4** Differential Diagnosis of Coma

## Emergency Department Care and Disposition

Treatment of coma involves supportive care and identification of the cause.

1. Stabilize the airway, ventilation, and circulation.
2. Identify and treat reversible causes, such as hypoglycemia and opioid toxicity. Consider empiric **naloxone**. Administer **thiamine** before **glucose** in hypoglycemic patients with a history of alcohol abuse or malnutrition.
3. If elevated ICP is suspected, elevate the head to 30° and keep at midline. **Mannitol** (0.25 to 1.0 gram/kilogram) will help reduce ICP.

Discharge patients with readily reversible causes of coma if home care and follow-up care are adequate and a clear cause of the episode is found. Admit all other patients for further evaluation and management.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 162, “Altered Mental Status and Coma,” by J. Stephen Huff.

# **CHAPTER 143**

## **Ataxia and Gait Disturbances**

Ross J. Fleischman

Ataxia is the inability to produce smooth, intentional movements. Ataxia and gait disturbances are not disease entities themselves, but are manifestations of either systemic or nervous system conditions.

### **CLINICAL FEATURES**

Key symptoms suggesting disease processes beyond the sensory and motor systems include headache, nausea, fever, and decreased level of alertness. The physical examination may also show abnormalities outside the nervous system. Orthostatic vital sign changes point to systemic illness.

Once the physician has determined that ataxia is the principal problem and not a manifestation of broader illness, the examination should attempt to differentiate between sensory and motor causes of ataxia. While cerebellar lesions may cause ataxia, isolated cerebellar lesions are not the most common cause. Dysmetria (undershoot or overshoot of movements) may be elicited by finger to nose testing. Dysdiadochokinesia (clumsy rapid alternating movements) may be seen when the patient alternately flips their palms and backs of their hands on their thighs. Both of these are suggestive of cerebellar causes. Having the patient slide one heel down the opposite shin is useful for distinguishing between cerebellar and sensory causes. Overshoot of the knee or ankle signifies cerebellar disease, while a wavering course down the shin suggests a deficit of proprioception.

Vibration and position sense in the toes test the posterior columns, which degenerate in tabes dorsalis (neurosyphilis) and vitamin B<sub>12</sub> deficiency. Nystagmus suggests an intracranial cause. In the Romberg test, the patient is asked to stand with arms at sides. Significantly worsening instability with eye closure (a positive Romberg sign) suggests that the patient is relying on visual input for balance caused by a sensory ataxia including posterior

column disease or vestibular dysfunction. Instability with the eyes still open suggests a cerebellar lesion.

Observing the patient rise from a chair and walk on heels and toes may expose subtle proximal or distal weakness. Tandem walking (heel to toe) may elicit subtle ataxia. A motor ataxic gait is characterized by broadbased, unsteady steps. Sensory ataxia with loss of proprioception may be notable for abrupt movements and slapping of the feet with each impact. A senile gait that is slow, broad based, and with a shortened stride may be seen with aging, but also with neurodegenerative disease such as Parkinson disease and normal pressure hydrocephalus. Parkinson disease may also show a festinating gait that is narrow-based, with small shuffling steps that become more rapid. Peroneal muscle weakness causes foot drop, known as an equine gait.

## **DIAGNOSIS AND DIFFERENTIAL**

[Table 143-1](#) shows common causes of acute ataxia and gait disturbances.



Systemic conditions
Intoxications with diminished alertness
Ethanol
Sedative-hypnotics
Intoxications with relatively preserved alertness (diminished alertness at higher levels)
Phenytoin
Carbamazepine
Valproic acid
Heavy metals-lead, organic mercurials
Other metabolic disorders
Hyponatremia
Inborn errors of metabolism
Wernicke disease
Disorders predominantly of the nervous system
Conditions affecting predominantly one region of the central nervous system
Cerebellum
Hemorrhage
Infarction
Degenerative changes
Abscess
Cortex
Frontal tumor, hemorrhage, or trauma
Hydrocephalus
Subcortical
Thalamic infarction or hemorrhage
Parkinson disease
Normal pressure hydrocephalus
Spinal cord
Cervical spondylosis
Posterior column disorders
Conditions affecting predominantly the peripheral nervous system
Peripheral neuropathy
Vestibulopathy

**TABLE 143-1** Common Etiologies of Acute Ataxia and Gait Disturbances

The extent of ED evaluation will depend on the acuity and severity of symptoms, with patients who have become unable to walk over hours to days requiring extensive evaluation. CT is less sensitive than MR for lesions of the posterior fossa, and is insensitive for acute ischemia. Perform a lumbar puncture if infection is suspected.

Consider vitamin B<sub>12</sub> deficiency in patients with loss of position sense in the second toe and a positive Romberg test. A serum cyanocobalamin level and complete blood count are the initial steps in evaluation, although neurologic manifestations often precede macrocytic anemia. Neurosyphilis

will cause similar symptoms of posterior column disease and can be screened for by the VDRL or RPR tests.

Suspect normal pressure hydrocephalus in an elderly patient with a broad-based, shuffling gait, urinary incontinence, and dementia. CT will show ventricular dilatation out of proportion to sulcal atrophy.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Administer **thiamine** 100 milligrams IV to alcoholics and other malnourished individuals who might have Wernicke disease, which is suggested by findings of ataxia, altered mental status, and ophthalmoplegia.
2. Admit patients with an acute inability to walk for further evaluation and diagnostic testing.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 163, “Ataxia and Gait Disturbances,” by J. Stephen Huff.

# CHAPTER 144

## Vertigo and Dizziness

Steven Go

Vertigo results from the mismatch of the perception of movement by the visual, vestibular, and proprioceptive symptoms when none actually exists.

### CLINICAL FEATURES

Vertigo is classically described as “the room is spinning,” but can also include atypical sensations of other types of movement. Vertigo is classified as peripheral or central ([Table 144-1](#)). Peripheral vertigo (involving vestibular apparatus and eighth cranial nerve) usually has a sudden onset and intense symptoms. Central vertigo (involving brainstem and cerebellum) can present abruptly or gradually, but usually has more ill-defined, less severe symptoms. Attempt to discriminate between the 2 in the ED, whilst recognizing significant overlap exists.

	Peripheral	Central
Onset	Sudden	Sudden or slow
Severity of vertigo	Intense spinning	Ill defined, less intense
Pattern	Paroxysmal, intermittent	Constant
Aggravated by position/movement	Yes	Variable
Associated nausea/diaphoresis	Frequent	Variable
Nystagmus	Rotatory-vertical, horizontal	Vertical
Fatigue of symptoms/signs	Yes	No
Hearing loss/tinnitus	May occur	Does not occur
Abnormal tympanic membrane	May occur	Does not occur
Central nervous system symptoms/signs	Absent	Usually present

**TABLE 144-1** Differentiating Peripheral from Central Vertigo

### DIAGNOSIS AND DIFFERENTIAL

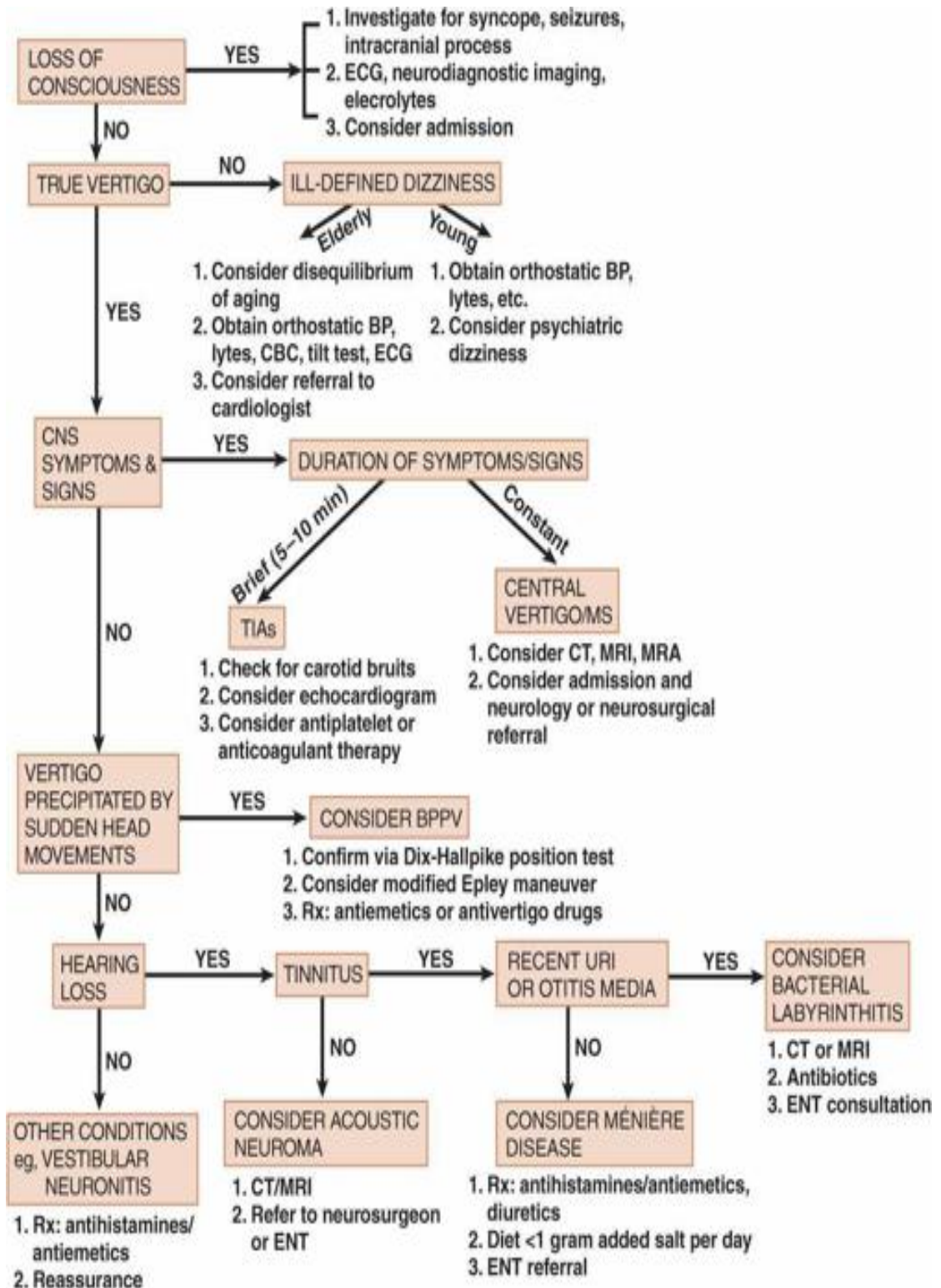
The differential diagnosis for vertigo ([Table 144-2](#)) is extensive and certain key findings should be sought on history and physical examination. The initial episode should be described in detail by the patient including speed of onset, severity,

associated symptoms (especially involving cranial nerves, loss of consciousness), and temporal pattern. Risk factors for stroke (age, hypertension, cardiovascular disease) and coagulopathy should be investigated. Physical examination should include eye (ie, nystagmus), ear, neurologic, and vestibular examinations, with particular focus on the cranial nerve and cerebellar examinations. If benign paroxysmal positional vertigo (BPPV) is suspected, a Dix-Hallpike position test may be useful (sensitivity 50% to 80%).

Vestibular/otologic	Benign paroxysmal positional vertigo Traumatic: following head injury Infection: labyrinthitis, vestibular neuronitis, Ramsay-Hunt syndrome
Syndrome	Ménière syndrome Neoplastic Vascular Otosclerosis Paget disease Toxic or drug-induced: aminoglycosides
Neurologic	Vertebrobasilar insufficiency Lateral Wallenberg syndrome Anterior inferior cerebellar artery syndrome Neoplastic: cerebellopontine angle tumors Cerebellar disorders: hemorrhage, degeneration Basal ganglion diseases Multiple sclerosis Infections: neurosyphilis, tuberculosis Epilepsy Migraine headaches Cerebrovascular disease
General	Hematologic: anemia, polycythemia, hyperviscosity syndrome Toxic: alcohol Chronic renal failure Metabolic: thyroid disease, hypoglycemia

**TABLE 144-2** An Etiologic Classification of Vertigo

In general, laboratory investigations are not indicated in vertiginous patients unless a specific cause for central vertigo is being investigated. With regards to imaging, the question of whether to obtain an emergent CT or MRI should be driven by the specific differential for a particular patient. However, an emergent noncontrast head CT should be obtained in elders, patients who have signs/symptoms of central vertigo (especially cranial nerve or cerebellar findings), hypertension, cardiovascular disease, other stroke risks, coagulopathy (eg, taking warfarin), headache, or intractable or persistent (> 72 hours) symptoms. If vertebrobasilar insufficiency (VBI) is a consideration, MRI with MRA (or CT Angiogram) and duplex US of the carotids are indicated. [Figure 144-1](#) illustrates a recommended approach to patients with vertigo.



**FIGURE 144-1.** Guideline approach to vertigo.

*Key:* BP = blood pressure; BPPV = benign paroxysmal positional vertigo; CBC = complete blood count; CNS = central nervous system; ENT = ear, nose, and throat;

MRA = magnetic resonance angiography; MS = multiple sclerosis; Rx = treatment; TIAs = transient ischemic attacks; URI = upper respiratory infection.

**BPPV** is thought to be caused by loose otoliths that enter the posterior semicircular canal and cause the inappropriate sensation of motion. Findings suggestive of BPPV are listed in [Table 144-3](#). The Dix-Hallpike position test can confirm the diagnosis. In this test, the patient begins seated with the head turned 45° to the right. The patient is then rapidly lowered to a supine position with the head hanging over the edge of the bed an additional 30° to 45°. Patients with BPPV will exhibit a latent and shortlived nystagmus with the rapid component toward the affected ear. The patient is then returned to the sitting position (the nystagmus often reverses upon resuming the upright position) and the left side tested. The side that is symptomatic serves as the starting point for the curative Epley maneuver (see below).

Latency period of <30 seconds between the provocative head position and onset of nystagmus.  
The intensity of nystagmus increases to a peak before slowly resolving.  
Duration of vertigo and nystagmus ranges from 5 to 40 seconds.  
If nystagmus is produced in one direction by placing the head down, then the nystagmus reverses direction when the head is returned to the sitting position.  
Repeated head positioning causes both the vertigo and accompanying nystagmus to fatigue and subside.

**TABLE 144-3** Supportive Findings in Benign Paroxysmal Positional Vertigo

**Ménière disease** is characterized by recurrent bouts of usually unilateral roaring tinnitus and a sense of fullness and diminished hearing in the affected ear. Because the diagnosis requires multiple episodes of attacks with progressive hearing loss, Ménière syndrome cannot be diagnosed on the first presentation of vertigo.

A **perilymphatic fistula** presents with sudden onset of vertigo during activities that can cause barotrauma such as flying, scuba diving, heavy lifting, and coughing. Infection can also cause a perilymph fistula, and the diagnosis is confirmed by nystagmus elicited by pneumatic otoscopy (Hennebert sign).

**Vestibular neuronitis** is characterized by the sudden onset of severe vertigo sometimes associated with unilateral tinnitus and hearing loss. It is thought to be viral in nature, lasts several days to weeks, and does not reoccur. **Vestibular ganglionitis** causes vertigo when a neurotrophic virus such as varicella zoster reactivates. The most famous variant is Ramsay-Hunt syndrome (deafness, vertigo, and facial nerve palsy), associated with vesicles inside the external auditory canal. **Labyrinthitis**, although commonly viral, also can be due to bacterial infection from otitis media, meningitis, and mastoiditis, and presents with sudden vertigo with hearing loss and middle ear findings.

**Ototoxicity** may induce vertigo and hearing loss. Common offenders causing peripheral toxicity include salicylates, aminoglycosides, and cytotoxic agents. Anticonvulsants, antidepressants, neuroleptics, hydrocarbons, alcohol, and phencyclidine may cause centrally mediated vertigo.

**Tumors of the eighth cranial nerve and cerebellopontine angle**, such as meningioma, acoustic neuroma, and acoustic schwannoma, also may present as vertigo with hearing loss. These tumors may be associated with ipsilateral facial weakness, impaired corneal reflexes, and cerebellar signs.

Vertigo may occur after **closed head injury** (eg, basilar skull fracture) and tends to resolve over weeks.

**Cerebellar hemorrhage or infarction** are central causes of vertigo. The vertigo is moderate and can be associated with nausea and vomiting. Cerebellar findings such as truncal ataxia are usually present.

**Lateral medullary infarction of the brainstem** (Wallenberg syndrome) causes vertigo and ipsilateral facial numbness, loss of the corneal reflex, Horner syndrome, and pharyngeal and laryngeal paralysis. Contralateral loss of pain and temperature sensation in the extremities also occurs.

**Vertebrobasilar insufficiency** (VBI) may result in sudden vertigo due to brainstem TIA that typically lasts min to up to 24 hours. Associated focal brainstem signs and syncope may also be present. Unlike other causes of central vertigo, VBI may be induced by movement of the head due to decreased vertebral artery blood flow.

**Vertebral artery dissection** (VAD) can be caused by sudden rotation of the head (motor vehicle crash, chiropractic adjustments, sneezing) and presents with central vertigo. Associated symptoms may include vertigo, headache, and unilateral Horner syndrome.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. In peripheral vertigo, first line therapies include the antihistamines ([Table 144-4](#)). **Diphenhydramine** 25 to 50 milligrams IM, IV, or PO and **meclizine** 25 milligrams PO are often effective in providing symptomatic relief. Second-line drugs for treatment failures include antidopaminergic agents such as **metoclopramide** 10 to 20 milligrams IV or PO and **promethazine** 25 to 50 milligrams IM or PR. Transdermal **scopolamine** 0.5 milligram is considered a drug of choice for treatment of vertigo; however, it is not useful acutely due to its prolonged onset of action (4 to 8 hours). It may be used as a discharge medication. **Benzodiazepines**

prevent the process of vestibular rehabilitation and should be used sparingly. Remember that antivertigo medications can have undesirable anticholinergic side effects such as drowsiness and urinary retention. In patients without true vertigo, these medications may exacerbate the dizziness experienced by the patient so using these drugs in combination should be avoided.

Category	Drug	Dosage	Indications
Anticholinergics	Scopolamine	0.5 milligram transdermal patch (behind ear) 3 to 4 times a day	Vertigo, nausea
Antihistamines	Dimenhydrinate	50 to 100 milligrams IM, IV, or PO every 4 h	Vertigo, nausea
	Diphenhydramine	25 to 50 milligrams IM, IV, or PO every 4 h	Vertigo, nausea
	Meclizine	25 milligrams PO 2 to 4 times a day	Vertigo, nausea
Antiemetics	Hydroxyzine	25 to 50 milligrams PO 4 times a day	Vertigo, nausea
	Metoclopramide	10 to 20 milligrams IV, PO 3 times a day	Vertigo, nausea
	Promethazine	25 milligrams IM, PO, or PR 3 to 4 times a day	Vertigo, nausea
Benzodiazepines	Diazepam	2 to 5 milligrams PO 2 to 4 times a day	Central vertigo, anxiety related to peripheral vertigo
	Clonazepam	0.5 milligram PO 2 times a day	Central vertigo, anxiety related to peripheral vertigo
Calcium antagonists	Cinnarizine	25 milligrams PO 2 to 3 times a day	Peripheral vertigo, migraine
	Nimodipine	30 milligrams PO 2 times a day	Peripheral vertigo
	Flunarizine	20 milligrams PO 2 times a day	Ménière
Vasodilators	Betahistine	8 to 16 milligrams PO 2 times a day	Peripheral vertigo

**TABLE 144-4** Pharmacotherapy of Vertigo and Dizziness

2. Treat patients with BPPV with the **Epley maneuver** to move the otoliths out of the semicircular canal. With the patient seated, turn the head 45° toward the affected ear. (The affected ear is determined by the direction in which the Dix-Hallpike position test is positive.) Slowly bring the patient to the recumbent position with the head hanging 30° to 45° below the examining table. Gently rotate the head 45° to the midline. Then rotate the head another 45° to the unaffected side. The patient rolls onto the shoulder of the unaffected side while rotating the head another 45°. Return the patient to the sitting position and the head to the midline. If the Epley maneuver is not completely successful, instruct the patient in vestibular rehabilitation exercises.



3. Treat vestibular ganglionitis with antivirals and bacterial labyrinthitis with appropriate antibiotics.
4. Most patients with peripheral vertigo may be discharged home with follow-up. Referral to an ENT specialist is indicated in patients with perilymph fistula or labyrinthitis of suspected bacterial etiology. Consult neurosurgery for patients diagnosed with a tumor. Admit patients with intractable symptoms.
5. Patients with central vertigo require imaging studies and specialty referral. Consult neurosurgery for patients with posterior fossa hemorrhage. Emergent causes of central vertigo such as ischemic cerebrovascular incidents and VAD require neurologic consultation in the ED. Refer urgent causes such as suspected multiple sclerosis for outpatient neurologic consultation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 164, "Vertigo and Dizziness," by Brian Goldman.

## CHAPTER 145

# Seizures and Status Epilepticus in Adults

C. Crawford Mechem

A seizure is an episode of abnormal neurologic function caused by inappropriate electrical discharge of brain neurons. Primary seizures are those without a clear cause. Secondary seizures result from another identifiable neurologic condition, such as a mass or stroke ([Table 145-1](#)).

Trauma (recent or remote)
Intracranial hemorrhage (subdural, epidural, subarachnoid, intraparenchymal)
Structural CNS abnormalities
Vascular lesion (aneurysm, arteriovenous malformation)
Mass lesions (primary or metastatic neoplasms)
Degenerative neurologic diseases
Congenital brain abnormalities
Infection (meningitis, encephalitis, abscess)
Metabolic disturbances
Hypoglycemia or hyperglycemia
Hyponatremia or hypernatremia
Hyperosmolar states
Uremia
Hepatic failure
Hypocalcemia, hypomagnesemia (rare)
Toxins and drugs (many)
Cocaine, lidocaine
Antidepressants
Theophylline
Alcohol withdrawal
Drug withdrawal
Eclampsia of pregnancy (may occur up to 8 weeks postpartum)
Hypertensive encephalopathy
Anoxic-ischemic injury (cardiac arrest, severe hypoxemia)

**TABLE 145-1** Common Causes of Secondary Seizures

## CLINICAL FEATURES

Seizures are classified as *generalized* and *partial*. Generalized seizures are caused by nearly simultaneous activation of the entire cerebral cortex. The attacks begin with abrupt loss of consciousness. Generalized tonic-clonic seizures (*grand mal*) are the most familiar example. The patient suddenly becomes rigid, trunk and extremities are extended, and the patient falls to the ground. The rigid (tonic) phase is followed by a symmetric, rhythmic (clonic) jerking of the trunk and extremities, commonly

associated with incontinence. After the attack, the patient is flaccid and unconscious. A typical episode lasts from 60 to 90 seconds, with gradual return of consciousness. Postictal confusion may persist for hours. *Absence* (petit mal) seizures are a subclass of generalized seizures. Typically seen in school-aged children, they usually last only a few seconds. Patients suddenly lose consciousness without losing postural tone. They appear confused, detached or withdrawn, and current activity ceases. The attack ends abruptly.

Partial seizures are due to electrical discharges beginning in a localized region of the cerebral cortex, often a brain lesion. The discharges may remain local or spread to other regions. Partial seizures may be *simple*, in which consciousness is not affected, or *complex*, in which consciousness is altered. Complex partial seizures are often due to discharges in the temporal lobe (also termed *temporal lobe seizures*). Symptoms include automatisms, visceral complaints, hallucinations, memory disturbances, distorted perception, and affective disorders.

*Status epilepticus* is continuous or intermittent seizures for more than 5 min without recovery of consciousness. *Nonconvulsive status epilepticus* is associated with minimal or imperceptible convulsive activity and is confirmed by EEG.

*Eclampsia* refers to the combination of seizures, hypertension, edema, and proteinuria in pregnant women beyond 20 weeks gestation or up to 3 weeks postpartum.

## DIAGNOSIS AND DIFFERENTIAL

Obtain a detailed history, addressing the presence of preceding aura, abrupt or gradual onset, progression of motor activity, incontinence, whether the activity was local or generalized and symmetric or not, duration, and the presence of postictal confusion or lethargy. If the patient has a known seizure disorder, ascertain previous seizure pattern, common precipitants, and any change in antiepileptic regimen. If there is no previous history of seizures, a more detailed inquiry is needed. Ask the patient about recent or remote head injury. Persistent, severe, or sudden headache suggests intracranial pathology. Pregnancy or recent delivery raises the possibility of eclampsia. A history of metabolic or electrolyte abnormalities, hypoxia, systemic illness (especially cancer), coagulopathy or anticoagulation, drug ingestion or withdrawal, and alcohol use may pinpoint predisposing factors. Seizures are a common manifestation of central nervous system (CNS) disease in patients with the human immunodeficiency virus, although their causes differ somewhat from immunocompetent patients ([Table 145-2](#)).

Mass lesion
Toxoplasmosis
Lymphoma
Meningitis/encephalitis
Cryptococcal
Bacterial/aseptic
Herpes zoster
Cytomegalovirus
Human immunodeficiency virus encephalopathy/acquired immunodeficiency syndrome dementia complex
Progressive multifocal leukoencephalopathy
CNS tuberculosis
Cysticercosis
Neurosyphilis

**TABLE 145-2** Causes of Seizures in the Human Immunodeficiency Virus Patient

Physical examination should include checking for injuries. Perform a directed neurologic exam and follow closely the level of consciousness. Progressive deterioration requires prompt intervention. A transient focal deficit following a simple or complex focal seizure is referred to as *Todd paralysis* and should resolve within 48 hours. If the patient’s symptoms cannot be readily attributed to a benign cause, further urgent evaluation is warranted.

Laboratory testing should be individualized. In a patient with a known seizure disorder who has had a routine seizure, a glucose level and an anticonvulsant level may suffice. In an adult with a first seizure, more extensive studies are usually needed, including serum glucose, electrolytes, renal functions, calcium, magnesium, a pregnancy test, and toxicology studies. Lumbar puncture is indicated if the patient is febrile or immunocompromised, or if subarachnoid hemorrhage is suspected. Obtain a noncontrast head CT for the patient with a first seizure or a change in seizure pattern to identify a structural lesion or an acute intracranial process. A follow-up contrast CT or MRI may be indicated. Other radiographic testing will be case-specific.

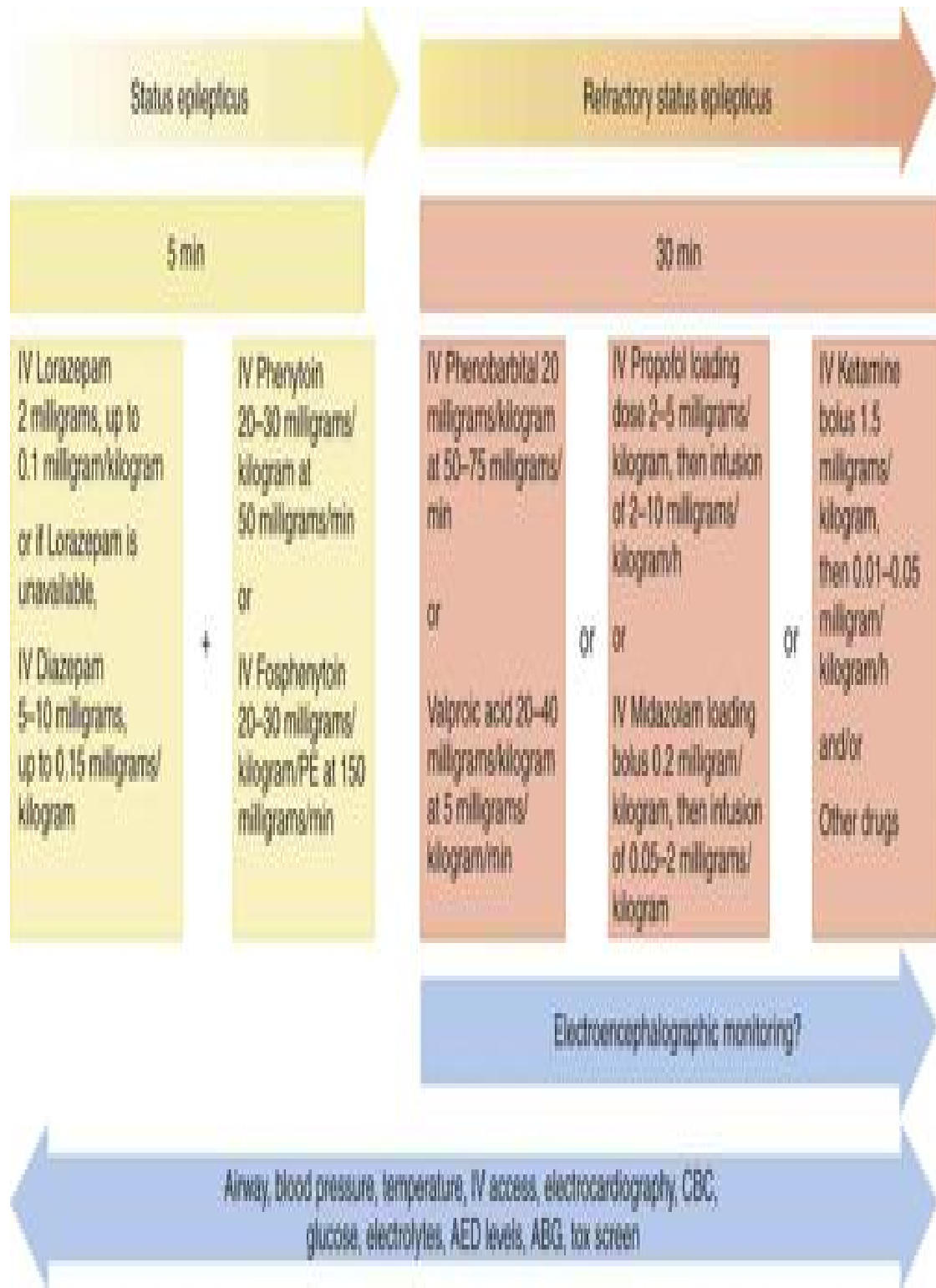
The differential diagnosis of seizures includes syncope, pseudoseizures, hyperventilation syndrome, movement disorders, migraines, and narcolepsy.

**EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Most commonly, little is required during the course of a seizure other than to protect the patient from injury. IV anticonvulsants are not indicated during an uncomplicated seizure.
2. In patients with a known seizure disorder whose anticonvulsant levels are low, supplemental doses may be appropriate. Oral loading of **phenytoin** (18

milligrams/kilogram PO as a single dose or divided into 3 doses given every 2 hours) will achieve therapeutic concentrations in 2 to 24 hours. Alternatively, 10 to 20 milligrams/kilogram of IV phenytoin at a rate of 25 milligrams/min achieves anticonvulsant effects in 1 to 2 hours. The dose of **fosphenytoin** is 10 to 20 milligrams phenytoin equivalent at a maximum IV rate of 150 milligrams/min. Loading doses of other antiepileptics may be guided by the patient's neurologist.

3. Administer **magnesium sulfate**, 4 to 6 grams IV, to eclamptic patients followed by an infusion of 1 to 2 grams/h. Consult an obstetrician early in the patient's care.
4. Patients in status epilepticus require large-bore IV access, cardiac monitoring, and pulse-oximetry. Endotracheal intubation is recommended. If a paralytic agent is used, a short-acting agent is preferred so seizure activity can be monitored. **Lorazepam** is considered the initial anticonvulsant of choice, followed by **phenytoin** or **fosphenytoin** ([Figure 145-1](#)). In refractory cases, consider IV **valproate**, **phenobarbital**, or **pentobarbital**. If seizures persist, induce anesthesia with IV infusions of **midazolam** or **propofol**. **Neuromuscular blocking agents** may be necessary, in which case EEG monitoring is mandatory.



**FIGURE 145-1.** Guidelines for management of status epilepticus.

*Key:* ABG = arterial blood gases; AED = antiepileptic drug; CBC = complete blood count; PE = phenytoin equivalent.

Patients with a first seizure who have a normal neurologic examination, no acute or chronic medical comorbidities, normal diagnostic testing including neuroimaging, and a normal mental status can be discharged from the ED. Initiation of antiepileptic medication and further testing may be deferred to the outpatient setting. Consider admission for patients who do not meet the above criteria. Instruct discharged patients to take precautions to minimize the risks for injury from further seizures. Swimming, working with hazardous tools or machines, and working at heights should be prohibited. Driving is prohibited until cleared by the neurologist or primary care physician, and driving privileges should conform to state laws.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 165, “Seizures and Status Epilepticus in Adults,” by Derrick D. Lung, Christina L. Catlett, and Judith E. Tintinalli.

# CHAPTER 146

## Acute Peripheral Neurologic Lesions

Jeffrey L. Hackman

A systematic approach to evaluating neurologic symptoms includes localizing the problem anatomically and distinguishing peripheral from central etiology. Peripheral nerve disorders may affect sensory, motor, and autonomic functions ([Table 146-1](#)).

	Central	Peripheral
History	Cognitive changes Sudden weakness Nausea, vomiting Headache	Weakness confined to 1 limb Weakness with pain associated Posture- or movement-dependent pain Weakness after prolonged period in 1 position
Physical examination		
Reflexes	Brisk reflexes (hyperreflexia) Babinski sign Hoffman sign	Hypoactive reflexes Areflexia
Motor	Asymmetric weakness of ipsilateral upper and lower extremity Facial droop Slurred speech	Symmetric proximal weakness
Sensory	Asymmetric sensory loss in ipsilateral upper and lower extremity	Reproduction of symptoms with movement (compressive neuropathy) All sensory modalities involved
Coordination	Discoordination without weakness	Loss of proprioception

**TABLE 146-1** Differentiating Central from Peripheral Nervous System Disorders

## NEUROMUSCULAR JUNCTION DISORDERS

Botulism is caused by *Clostridium botulinum* toxin and occurs in 3 forms: foodborne, wound, and infantile. Foodborne botulism typically comes from improperly preserved canned foods. In infantile botulism, organisms arise



from ingested spores, often in honey, and produce a systemically absorbed toxin. Clinical features appear 6 to 48 hours after ingestion and may be preceded by nausea, vomiting, and diarrhea. Wound botulism should be considered in patients with a wound or a history of intravenous drug use. Early complaints involve the eye or bulbar musculature and progress to descending weakness and respiratory insufficiency. Absent light reflex is a diagnostic clue, and mentation is normal. Infants may present with constipation, poor feeding, lethargy, and weak cry. Treatment includes respiratory support, trivalent botulinum antitoxin 10 mL IV, and admission.

## ACUTE PERIPHERAL NEUROPATHIES

Guillain-Barré syndrome (GBS) affects all ages and usually follows a viral or febrile illness, *Campylobacter jejuni* infection, or vaccination. Although numerous variants exist, the typical presentation includes ascending symmetric weakness or paralysis and loss of deep tendon reflexes. Respiratory failure and lethal autonomic fluctuations may occur. Cerebrospinal fluid (CSF) analysis typically shows high protein and a normal cell count ([Table 146-2](#)). Initial treatment includes respiratory support, admission to a monitored setting, and neurologic consultation.

Required
Progressive weakness of more than 1 limb
Areflexia
Suggestive
Progression over days to weeks
Recovery beginning 2 to 4 weeks after cessation of progression
Relative symmetry of symptoms
Mild sensory signs and symptoms
Cranial nerve involvement (Bell's palsy, dysphagia, dysarthria, ophthalmoplegia)
Autonomic dysfunction (tachycardia, bradycardia, dysrhythmias, wide variations in blood pressure, postural hypotension, urinary retention, constipation, facial flushing, anhidrosis, hypersalivation)
Absence of fever at onset
Cytoalbuminologic dissociation of cerebrospinal fluid (high protein and low white cell count)
Typical findings on electromyogram and nerve conduction studies

**TABLE 146-2** Diagnostic Criteria for Classic Guillain-Barré Syndrome

## **FOCAL NEUROPATHIES**

Carpal tunnel syndrome, resulting from compression of the median nerve at the wrist, classically causes pain, paresthesias, and numbness in the distribution of the median nerve. Tinel sign (light percussion over median nerve at wrist results in electric shock sensation shooting into hand) and Phalen maneuver (holding wrists in flexion for 60 seconds, worsens symptoms) may be helpful to confirm the diagnosis. ED treatment consists of recommending avoiding aggravating factors and splinting the wrist in neutral position. Cubital tunnel syndrome, resulting from compression of the ulnar nerve at the elbow, causes tingling in the fifth and lateral fourth fingers that may progress to paralysis and wasting of the intrinsic hand muscles. ED treatment consists of anti-inflammatory medications and splinting with a long arm posterior splint or a sling. Other common focal neuropathies include deep peroneal entrapment (causing foot drop and numbness between the first and second toes), meralgia paresthetica (entrapment of the lateral femoral cutaneous nerve causing numbness and pain of the anterolateral thigh), and mononeuritis multiplex (dysfunction of multiple peripheral nerves separated temporally and anatomically). Direct treatment at the underlying cause.

## **PLEXOPATHIES**

Brachial plexopathy causes weakness in the arm or shoulder girdle followed by pain and paresthesias. Patients have weakness in various distributions of the brachial plexus. ED evaluation is directed at identifying acutely reversible causes (eg, direct trauma, shoulder reduction) and referral for other causes (eg, neoplasm, radiation). Lumbosacral plexopathy due to radiation or diabetic amyotrophy, or compression from aortic aneurysm, retroperitoneal hemorrhage, or arteriovenous malformations, will cause weakness, decreased sensation, and possibly decreased reflexes in the areas innervated by the affected portions of the plexus. Plain radiographs, MRI,

and abdominal CT may be useful in determining the etiology. Direct treatment at the underlying cause.

## **HIV-ASSOCIATED PERIPHERAL NEUROLOGIC DISEASE**

HIV infection, its complications, and treatments cause a variety of peripheral neurologic disorders. Antiretroviral drug-induced and HIV neuropathy are chronic and do not cause acute symptoms. Patients with HIV have a high rate of mononeuritis multiplex and a myopathy resembling polymyositis. In early infection, they are more prone to GBS. In the later stages of AIDS, patients may develop cytomegalovirus (CMV) radiculitis, with acute weakness and decreased sensation of the lower extremities, hyporeflexia, and varying bowel and bladder dysfunction. MRI shows swelling and clumping of the cauda equina. Treatment for CMV radiculitis, which may precede definitive diagnosis, consists of IV ganciclovir 5 milligrams/ kilogram every 12 hours for 3 to 6 weeks.

## **BELL'S PALSY**

Bell's palsy causes seventh cranial nerve dysfunction, and patients may complain of facial weakness, articulation problems, difficulty keeping an eye closed, or inability to keep food in the mouth on one side. Physical examination findings demonstrate weakness on one side of the face, including the forehead, and no other focal neurologic findings. The differential diagnosis includes stroke, Lyme disease, GBS, parotid tumors, middle ear lesions, cerebellopontine angle tumors, eighth cranial nerve lesions, HIV, and vascular disease. The ear should be inspected for ulcerations caused by cranial herpes zoster activation (Ramsey-Hunt syndrome), which should be treated with oral acyclovir. If muscle strength is retained in the forehead, the lesion most likely is central (ie, in the brainstem or above); this would exclude Bell's palsy, and CT of the head is indicated. Treatment with prednisone (60 milligrams/d for 7 days) is

recommended by most studies. Treatment with acyclovir (200 milligrams 5 times a day for 10 days) is controversial, and its added benefit is unclear. Patients should apply lacrilube to prevent corneal drying at night. Close follow-up with a neurologist or ENT specialist is indicated.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 166, “Acute Peripheral Neurologic Lesions,” by Phillip Andrus and Andy Jagoda.

# **CHAPTER 147**

## **Chronic Neurologic Disorders**

Sarah Andrus Gaines

Acute ED management of these disorders centers on the care of acute respiratory failure as the most notable complication.

### **AMYOTROPHIC LATERAL SCLEROSIS**

#### **Clinical Features**

Amyotrophic lateral sclerosis (ALS) causes progressive muscle atrophy and weakness. Upper motor neuron dysfunction causes limb spasticity, hyperreflexia, and emotional lability. Lower neuron dysfunction causes limb muscle weakness, atrophy, fasciculations, dysarthria, dysphagia, and difficulty in mastication. Symptoms are asymmetric. Patients may appear to have an acute compressive radiculopathy. Respiratory muscle weakness causes progressive respiratory depression.

#### **Diagnosis and Differential**

The diagnosis is suggested by upper and lower motor neuron dysfunction without other central nervous system dysfunction. The differential includes myasthenia gravis, diabetes, thyroid dysfunction, vitamin B<sub>12</sub> deficiency, lead toxicity, vasculitis, and CNS or spinal cord tumors.

#### **Emergency Department Care and Disposition**

Respiratory failure, pneumonia, choking, and trauma are the common ED presentations. Optimize pulmonary function with nebulizer treatments,

steroids, antibiotics, and intubation, as indicated. Admit patients with pneumonia or inability to handle secretions.

## **MYASTHENIA GRAVIS**

### **Clinical Features**

Myasthenia gravis (MG) is characterized by muscle weakness and fatigue. Most MG patients have weakness of the proximal extremity muscles, neck extensors, and facial or bulbar muscles. Ptosis and diplopia are common presenting symptoms; 10% of patients have ocular muscle weakness only. Symptoms worsen as the day progresses and improve with rest. Severe respiratory muscle weakness causing respiratory failure is seen in myasthenic crisis.

### **Diagnosis and Differential**

The differential for MG includes Lambert-Eaton, thyroid disorders, and stroke. MG is confirmed through administration of edrophonium, electromyogram, and serum testing for acetylcholine receptor antibodies. The edrophonium (Tensilon) test can differentiate a myasthenic crisis (inadequate treatment) from a cholinergic crisis (overmedication). Edrophonium has a rapid onset and short duration of action. First, a test dose of 1 to 2 milligrams IV is given and, if symptoms such as muscle weakness or respiratory depression worsen (cholinergic crisis), then the test is stopped. Emergent intubation may be necessary. Otherwise, the test is considered positive, indicating myasthenic crisis. Edrophonium can cause bradycardia, which responds to atropine.

### **Emergency Department Care and Disposition**

MG is treated with airway management, avoidance of drugs that worsen MG, and administration of acetylcholinesterase inhibitors, high-dose

steroids, plasmapheresis, or IV immunoglobulins.

1. Administer supplemental **oxygen**. Etomidate can be used if rapid sequence intubation is considered. Avoid depolarizing or nondepolarizing paralytic agents.
2. If the Tensilon test is positive, **neostigmine** should be given (0.5 to 2 milligrams IM, IV, SC or increments of 15 milligrams PO). MG patients treated for other concerns should receive their usual cholinergic inhibitors.
3. Many drugs can exacerbate MG. Check carefully for drug interactions. Consult with a neurologist to aid in decision-making ([Table 147-1](#)).

Steroids	Adrenocorticotrophic hormone,* methylprednisolone,* prednisone*
Anticonvulsants	Phenytoin, ethosuximide, trimethadione, paraldehyde, magnesium sulfate, barbiturates; lithium
Antimalarials	Chloroquine,* quinine*
IV fluids	Na lactate solution
Antibiotics	Aminoglycosides, fluoroquinolones,* neomycin,* streptomycin,* kanamycin,* gentamicin, tobramycin, dihydrostreptomycin,* amikacin, polymyxin A, polymyxin B, sulfonamides, viomycin, colistimethate,* lincomycin, clindamycin, tetracycline, oxytetracycline, rolitetracycline, macrolides, metronidazole
Psychotropics	Chlorpromazine,* lithium carbonate,* amitriptyline, droperidol, haloperidol, imipramine
Antirheumatics	D-Penicillamine, colchicine, chloroquine
Cardiovascular	Quinidine,* procainamide,* $\beta$ -blockers (propranolol, oxprenolol,



	practolol, pindolol, sotalol), lidocaine, trimethaphan; magnesium; calcium channel blockers (verapamil)
Local anesthetics	Lidocaine,* procaine*
Analgesics	Narcotics (morphine, hydromorphone, codeine, Pantopon, meperidine)
Endocrine	Thyroid replacement*
Eyedrops	Timolol,* echothiophate
Others	Amantadine, diphenhydramine, emetine, diuretics, muscle relaxants, central nervous system depressants, respiratory depressants, sedatives, procaine,* phenothiazines
Neuromuscular blocking agents	Tubocurarine, pancuronium, gallamine, dimethyl tubocurarine, succinylcholine, decamethonium

Note: See also discussion on eMedicine from WebMD by Aashit K. Shah, MD, available at: <http://emedicine.medscape.com/article/793136-treatment>.

\*Case reports implicate drugs in exacerbations of myasthenia gravis.

**TABLE 147-1** Drugs That Should Be Avoided in Myasthenia Gravis

## **MULTIPLE SCLEROSIS**

### **Clinical Features**

Multifocal areas of CNS demyelination causing motor, sensory, visual, and cerebellar dysfunction are seen in multiple sclerosis (MS). Types include relapsing and remitting (90%), relapsing and progressive, and chronically progressive. Lhermitte sign is described as an electric shock sensation going down into the arms or legs from neck flexion. Physical examination shows decreased strength, increased tone, hyperreflexia, clonus, decrease in vibratory sense and joint proprioception, and reduced pain and temperature sense. Increases in body temperature may worsen symptoms. Optic neuritis is the presenting symptom in 30% of cases and may cause an afferent papillary defect (Marcus-Gunn pupil). Acute or sub-acute central vision loss occurs over days and is usually unilateral. Retrobulbar or extraocular pain usually precedes vision loss. Internuclear ophthalmoplegia causes abnormal adduction and horizontal nystagmus, often bilaterally and is strongly suggestive of MS. Cognitive and emotional problems are common. Rarely, acute transverse myelitis occurs.

## **Diagnosis and Differential**

The diagnosis is suggested by 2 or more episodes of neurologic dysfunction. MRI shows lesions including in the supratentorial white matter or periventricular areas. CSF protein and  $\gamma$ -globulin levels are often elevated. The differential includes SLE, Lyme disease, neurosyphilis, and HIV disease.

## **Emergency Department Care and Disposition**

Treatment is directed at the complications of acute MS exacerbation. Consult a neurologist for care of a preexisting diagnosis and refer to a neurologist for new onset symptoms.

1. High-dose **methylprednisolone** has been shown to shorten duration of exacerbations.
2. Reduce fever to minimize symptoms and investigate for sources of infection. Evaluate for acute UTI or pyelonephritis; postvoid residuals >

100 mL require intermittent catheterization.

3. Admit toxic-appearing patients with exacerbations requiring steroids or antibiotics.

## **LAMBERT-EATON SYNDROME**

## **MYASTHENIC**

Lambert-Eaton syndrome causes fluctuating proximal limb muscle weakness and fatigue and is seen mainly in older men with lung cancer. Strength improves with activity. Patients complain of myalgias, stiffness, paresthesias, metallic tastes, and autonomic symptoms. Eye movements are unaffected. Electromyography is abnormal, and serum tests are specific for antibodies to voltage-gated calcium channels. Treatment of the underlying neoplasm improves symptoms. Pyridostigmine and immunosuppressive drugs may reduce symptom severity.

## **PARKINSON'S DISEASE**

### **Clinical Features**

Parkinson's disease (PD) presents with 4 classic signs: resting tremor, cogwheel rigidity, bradykinesia or akinesia, and impaired posture and equilibrium. Initially, most patients complain of a unilateral resting arm tremor, described as "pill rolling," which improves with intentional movement.

### **Diagnosis and Differential**

The diagnosis is clinical and based on the presence of the 4 classic signs. Parkinsonism can result from street drugs, toxins, neuroleptic drugs, hydrocephalus, head trauma, and other rare neurologic disorders. Drug-

induced PD most commonly presents with akinesia. No neuroimaging or laboratory study is pathognomonic.

## **Emergency Department Care and Disposition**

Most patients are on medications that either increase central dopamine, are anticholinergics, or act as dopamine receptor agonists. Medication toxicity includes psychiatric or sleep disturbances, cardiac dysrhythmias, orthostatic hypotension, dyskinesias, and dystonia. With significant motor or psychiatric disturbances or decreased drug efficacy, a “drug holiday” for 1 week should be initiated in consultation with a neurologist.

# **POLIOMYELITIS                      AND                      POSTPOLIO SYNDROME**

## **Clinical Features**

Poliomyelitis is caused by an enterovirus that induces paralysis by motor neuron destruction and muscle denervation and atrophy. Most patients have a mild viral syndrome and no paralysis. Major illness most commonly involves the spinal cord, resulting in asymmetric proximal limb weakness and flaccidity, absent tendon reflexes, and fasciculations. Maximal paralysis occurs within 5 days and is followed by muscle wasting. Autonomic dysfunction is common. Paralysis will resolve within the first year in nearly all patients. Other sequelae include bulbar polio (speech and swallowing dysfunction) and encephalitis. Polio is still endemic in Nigeria, Pakistan, Afghanistan, and India.

Postpolio syndrome is the recurrence of motor symptoms after a latent period of several decades. Symptoms may include muscle fatigue, joint pain, or weakness of new and previously affected muscle groups. Patients may have new bulbar, respiratory, or sleep difficulties.

## **Diagnosis and Differential**

Polio should be considered in a patient with an acute febrile illness, aseptic meningitis, and asymmetric flaccid paralysis with loss of deep tendon reflexes and normal sensation. CSF shows a pleocytosis and positive cultures for poliovirus. Throat and rectal swabs are higher yield tests.

The differential diagnosis includes Guillain-Barré syndrome, peripheral neuropathies (eg, mononucleosis, Lyme disease, or porphyria), abnormal electrolyte levels, toxins, inflammatory myopathies, and other viruses (eg, Coxsackie, mumps, echo, and various enteroviruses).

### **Emergency Department Care and Disposition**

Acute care is supportive. Lamotrigine may improve quality of life. Disposition should be made in consultation with a neurologist.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 167, “Chronic Neurologic Disorders” by Edward P. Sloan, Daniel A. Handel, and Sarah Andrus Gaines.

## **CHAPTER 148**

# **Central Nervous System and Spinal Infections**

O. John Ma

## **MENINGITIS**

### **Clinical Features**

In classic and fulminant cases of bacterial meningitis, the patient presents with fever, headache, neck stiffness, and altered mental status. Seizures may occur in up to 25% of cases. The presenting picture, however, may be more nonspecific, particularly in the very young and elderly. It is important to inquire about recent antibiotic use, which may cloud the clinical picture in a less florid case. Other key historical data include living conditions (eg, Army barracks, college dormitories), trauma, immunocompetence, immunization status, and recent neurosurgical procedures.

Physical examination should include assessment for meningeal irritation with resistance to passive neck flexion, Brudzinski sign (flexion of hips and knees in response to passive neck flexion), and Kernig sign (contraction of hamstrings in response to knee extension while hip is flexed). Examine the skin for the purpuric rash characteristic of meningococemia or streptococemia. Percuss the paranasal sinuses and examine the ears for evidence of primary infection in those sites. Assess for focal neurologic deficits and check the fundi for papilledema, which indicate increased intracranial pressure.

### **Diagnosis and Differential**

When the diagnosis of bacterial meningitis is entertained, treatment should precede diagnostic testing (see ED Care and Disposition Section).

Performing a lumbar puncture (LP) is mandatory when bacterial meningitis is suspected. At a minimum, analyze cerebrospinal fluid (CSF) for gram stain and culture, cell count, protein, and glucose. Typical CSF results for meningeal processes are listed in [Table 148-1](#). Additional studies to be considered are latex agglutination or counterimmune electrophoresis for bacterial antigens in potentially partially treated bacterial cases, India ink or serum cryptococcal antigen in immunocompromised patients, acid-fast stain and culture for mycobacteria in tuberculous meningitis, *Borrelia* antibodies for possible Lyme disease, and viral cultures in suspected viral meningitis. Other laboratory tests should include a complete blood count, blood cultures, coagulation studies, and basic metabolic panel.

Parameter (Normal)	Bacterial	Viral	Neoplastic	Fungal
Opening pressure (< 170 mm cerebrospinal fluid)	> 300 mm	< 300 mm	200 mm	300 mm
White blood cell count (< 5 mononuclear)	> 1000/mm <sup>3</sup>	< 1000/mm <sup>3</sup>	< 500/mm <sup>3</sup>	< 500/mm <sup>3</sup>
% Polymorphonuclear cells (0)	> 80%	1% to 50%	1% to 50%	1% to 50%
Glucose (> 40 milligrams/dL)	< 40 milligrams/dL	> 40 milligrams/dL	< 40 milligrams/dL	< 40 milligrams/dL
Protein (< 50 milligrams/dL)	> 200 milligrams/dL	< 200 milligrams/dL	> 200 milligrams/dL	> 200 milligrams/dL
Gram stain (-)	+	-	-	-
Cytology (-)	-	-	+	+

**TABLE 148-1** Typical Spinal Fluid Results for Meningeal Processes

[Table 148-2](#) lists suggested criteria for obtaining head CT prior to LP. In these cases, administer empiric antibiotic therapy before patient transport to CT.

Altered mental status or deteriorating level of consciousness
Focal neurologic deficit
Seizure
Papilledema
Immunocompromised state
Malignancy
History of focal central nervous system disease (stroke, focal infection, tumor)
Concern for mass central nervous system lesion
Age > 60 years

**TABLE 148-2** Some Suggested Criteria for Obtaining Head CT before Lumbar Puncture for Suspected Meningitis

The differential diagnosis includes subarachnoid hemorrhage, meningeal neoplasm, brain abscess, viral encephalitis, cerebral toxoplasmosis, and other infectious meningitides.

**Emergency Department Care and Disposition**

1. Emergent respiratory and hemodynamic support is given top priority.
2. Upon presentation of the patient with suspected bacterial meningitis, always administer empiric antibiotic therapy as soon as possible and never delay it for neuroimaging or LP. Empiric treatment for bacterial meningitis is based on the likelihood of certain pathogens and risk factors ([Table 148-3](#)).



Patient Category	Potential Pathogens	Empirical Therapy
<b>Age</b>		
18 to 50 years	<i>Streptococcus pneumoniae</i> , <i>Neisseria meningitidis</i>	Ceftriaxone, 2 grams IV every 12 h, plus vancomycin, 15 milligrams/ kilogram IV every 8 to 12 h (or rifampin if <i>S pneumoniae</i> resistance possible)
>50 years	<i>S pneumoniae</i> , <i>N meningitidis</i> , <i>Listeria monocytogenes</i> , aerobic gram-negative bacilli	Ceftriaxone, 2 grams IV every 12 h, plus ampicillin, 2 grams IV every 4 h, plus vancomycin, 15 milligrams/ kilogram IV every 8 to 12 h (or rifampin if <i>S pneumoniae</i> resistance possible)
<b>Special Circumstances</b>		
CSF leak with history of closed head trauma	<i>S pneumoniae</i> , <i>Haemophilus</i> <i>influenzae</i> , group B streptococcus	Ceftriaxone, 2 grams IV every 12 h, plus vancomycin, 15 milligrams/ kilogram IV every 8 to 12 h

History of recent penetrating head injury, neurosurgery, CSF shunt	<i>Staphylococcus aureus</i> , <i>S epidermidis</i> , diphtheroids, aerobic gram-negative bacilli	Vancomycin, 25 milligrams/kilogram IV load (maximum infusion rate, 500 milligrams/h), plus ceftazidime, 2 grams IV every 8 h, or ceftazidime or meropenem
Penicillin or cephalosporin allergy	<i>S pneumoniae</i> , <i>N meningitidis</i> , <i>L monocytogenes</i> , aerobic gram-negative bacilli	Vancomycin 30 to 60 milligrams/kilogram IV per day in 2 to 3 divided doses, plus moxifloxacin 400 milligrams IV once daily, plus trimethoprim-sulfamethoxazole 10 to 20 milligrams/kilogram (of the trimethoprim component) IV per day divided every 6 to 12 h

Note: See also <http://www.hopkins-abxguide.org>, accessed March 3, 2009.

Key: CSF = cerebrospinal fluid.

\*For pediatric meningitis treatment, see Chapter 113, Fever and Serious Bacterial Illness in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed.

**TABLE 148-3** Guidelines for Empirical Treatment of Bacterial Meningitis in Adults\* or with No Organisms on Gram Stain

- Dexamethasone** (10 milligrams IV in adults, 0.15 milligram/kilogram IV in children) before or at the time of first antibiotic administration has been shown to be beneficial in improving outcomes in bacterial (*Streptococcus Pneumoniae*) meningitis.

4. Avoid hypotonic fluids. Monitor serum sodium levels in order to detect the syndrome of inappropriate antidiuretic hormone or cerebral salt-wasting.
5. Manage potential complications of meningitis, seizures, hyperpyrexia, increased intracranial pressure, coagulopathy, and systemic consequences of a comatose state, with standard methods.
6. Manage viral meningitis, without evidence of encephalitis, on an outpatient basis as long as the patient is nontoxic in appearance, can tolerate oral fluids, and has reliable follow-up within 24 hours. However, it remains a diagnosis of exclusion; unless the diagnosis of viral meningitis is obvious, admission is warranted.

## **ENCEPHALITIS**

### **Clinical Features**

Viral encephalitis is a viral infection of brain parenchyma producing an inflammatory response. It is distinct from, although often coexists with, viral meningitis. In North America, viruses that cause encephalitis are the arboviruses (including the West Nile virus), herpes simplex virus (HSV), herpes zoster virus (HZV), Epstein-Barr virus, cytomegalovirus (CMV), and rabies.

Encephalitis should be considered in patients presenting with any or all of the following features: new psychiatric symptoms, cognitive deficits (aphasia, amnesic syndrome, acute confusional state), seizures, and movement disorders. Headache, photophobia, fever, and meningeal irritation may be present. Assessment for neurologic findings and cognitive deficits is crucial. Motor and sensory deficits are not typical. Encephalitides may show special regional tropism. HSV involves limbic structures of the temporal and frontal lobes, with prominent psychiatric features, memory disturbance, and aphasia. Some arboviruses predominantly affect the basal ganglia, causing chorea-athetosis and Parkinsonism. Involvement of the brainstem nuclei leads to hydrophobic choking characteristic of rabies encephalitis.

Symptoms of West Nile virus infection include fever, headache, muscle weakness, and lymphadenopathy. Most infections are mild and last only a

few days. More severe symptoms and signs consist of high fever, neck stiffness, altered mental status, tremors, and seizures. In rare cases (mostly involving the elderly), the infection can lead to encephalitis and death.

## **Diagnosis and Differential**

Findings on CT or magnetic resonance imaging (MRI) and LP aid in the ED diagnosis of encephalitis. Neuroimaging, particularly MRI, not only excludes other potential lesions, such as brain abscess, but may display findings highly suggestive of HSV encephalitis if the medial temporal and inferior frontal gray matter is involved. Findings of aseptic meningitis are typically found on CSF examination. For the West Nile virus, the most widely used screening test is the IgM ELISA assay for detecting acute antibody.

The differential diagnosis includes brain abscess; Lyme disease; sub-acute subarachnoid hemorrhage; bacterial, tuberculous, fungal, or neoplastic meningitis; bacterial endocarditis; postinfectious encephalomyelitis; toxic or metabolic encephalopathies; and primary psychiatric disorders.

## **Emergency Department Care and Disposition**

1. Admit the patient suspected of suffering from viral encephalitis. Treat patients with suspected HSV or HZV encephalitis with **acyclovir** 10 milligrams/kilogram IV every 8 hours. Treat patients with suspected CMV encephalitis with **ganciclovir** 5 milligrams/kilogram IV every 12 hours.
2. Manage potential complications of encephalitis—seizures, disorders of sodium metabolism, increased intracranial pressure, and systemic consequences of a comatose state—with standard methods.
3. There is no specific treatment for the West Nile virus infection. In more severe cases, intensive supportive therapy is indicated. The primary prevention step is advocating the use of insect repellent containing DEET when people go outdoors during dawn or dusk.

## **BRAIN ABSCESS**

## **Clinical Features**

A brain abscess, which is a focal pyogenic infection, is composed of a central pus-filled cavity, ringed by a layer of granulation tissue and an outer fibrous capsule. Since patients typically are not acutely toxic, the presenting features of brain abscess are nonspecific. Presenting signs and symptoms include headache, neck stiffness, fever, vomiting, confusion, or obtundation. The presentation may be dominated by the origin of the infection (eg, ear or sinus pain). Meningeal signs and focal neurologic findings, such as hemiparesis, seizures, and papilledema, are present in less than half the cases.

## **Diagnosis and Differential**

Brain abscess can be diagnosed by a CT scan of the head with contrast, which demonstrates one or several thin, smoothly contoured rings of enhancement surrounding a low-density center and in turn surrounded by white matter edema. LP is contraindicated if brain abscess is suspected or after the diagnosis has been established. Routine laboratory studies are usually nonspecific. Blood cultures should be obtained.

The differential diagnosis includes cerebrovascular disease, meningitis, brain neoplasm, sub-acute cerebral hemorrhage, and other focal brain infections, such as toxoplasmosis.

## **Emergency Department Care and Disposition**

1. Decisions on antibiotic therapy for brain abscess are dependent on the likely source of the infection ([Table 148-4](#)).

Presumed Source	Primary Empiric Therapy	Alternative Therapy
Otogenic	Cefotaxime, 2 grams IV every 6 h, plus metronidazole, 500 milligrams IV every 6 h	Cefepime, 2 grams IV every 8 h, plus metronidazole, 500 milligrams IV every 6 h
Sinogenic or odontogenic	Cefotaxime, 2 grams IV every 6 h, plus metronidazole, 500 milligrams IV every 6 h	Clindamycin plus metronidazole
Penetrating trauma or neurosurgical procedures	Vancomycin, 15 milligrams/kilogram IV every 12 h (maximum, 1 gram/dose; monitor serum levels), plus ceftazidime, 2 grams IV every 8 h	Nafcillin, 2 grams IV every 4 h, plus ceftazidime, 2 grams IV every 8 h
Hematogenous	Cefotaxime, 2 grams IV every 6 h, plus metronidazole, 500 milligrams IV every 6 h	No recommendation
No obvious source	Cefotaxime, 2 grams IV every 6 h, plus metronidazole, 500 milligrams IV every 6 h	No recommendation

Note: See also <http://www.hopkins-abxguide.org>, accessed May 2010.

**TABLE 148-4** Guidelines for Empiric Treatment of Brain Abscess Based on Presumed Source

2. Neurosurgical consultation and admission are warranted since many cases will require surgery for diagnosis, bacteriology and, definitive treatment.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 168, “Central Nervous System and Spinal Infections,” by Keith E. Loring and Judith E. Tintinalli.

# **SECTION 16 Eye, Ear, Nose, Throat, and Oral Emergencies**

# CHAPTER 149

## Ocular Emergencies

Steven Go

### INFECTIONS

#### Preseptal and Postseptal Cellulitis

Preseptal cellulitis is an infection of the periorbital tissues, whereas postseptal cellulitis involves the orbit. These disease entities occur mostly in patients <10 years of age. They may present nearly identically with typical symptoms of erythema, warmth, tenderness to palpation. In preseptal cellulitis, the visual acuity and pupillary responses are normal, and there is the complete absence of any pain with extraocular movements, proptosis, and diplopia. If any of these characteristics are present, or if there is concern about postseptal involvement, then a **CT scan** (or MRI) of the orbit (axial and coronal views, with and without contrast) should be obtained to rule out orbital involvement. Preseptal cellulitis in nontoxic patients may be treated as an outpatient with **amoxicillin/clavulanic acid** (20 milligrams/kilogram PO divided every 12 hours; 500 milligrams PO tid in adults) with 24 hours follow-up with an ophthalmologist. In preseptal cellulitis in children <5 years or adults with significant comorbidities or in cases of postseptal cellulitis, an emergent ophthalmology consultation for admission should be obtained. Empiric intravenous therapy should begin with **cefuroxime** (50 milligrams/kilogram IV every 8 hours) or **ceftriaxone** (50 milligrams/kilogram every 12 hours), or **ampicillin-sulbactam** (50 milligrams/kilogram IV every 6 hours), with IV vancomycin added if MRSA is suspected.

#### Stye (External Hordeolum) and Chalazion



A stye or external hordeolum, is an acute infection of an oil gland at the lash line that appears as a pustule at the lid margin. A chalazion is an acute or chronic noninfectious inflammation of the eyelid secondary to meibomian gland blockage in the tarsal plate. When it is acute, a chalazion may be painful, but is usually painless when chronic. A stye or acute chalazion is treated with warm, wet compresses 4 times daily and with **erythromycin** 0.5% ophthalmic ointment twice daily for 7 to 10 days. Persistent or recurrent lesions should be referred to an ophthalmologist for further evaluation and treatment.

## **Bacterial Conjunctivitis**

Bacterial conjunctivitis presents as eyelash matting, mild to moderate mucopurulent discharge, and conjunctival inflammation. Fluorescein staining of the cornea should be performed in patients with suspected conjunctivitis to avoid missing abrasions, ulcers, and dendritic lesions. Topical antibiotics ([Table 149-1](#)) are appropriate. In children, *Haemophilus influenzae* and *Moraxella catarrhalis* are considerations; therefore, if erythromycin ointment is ineffective, a change in antibiotics should be initiated. Contact lens wearers should receive topical antibiotic coverage for *Pseudomonas*, such as **ciprofloxacin** or **tobramycin**. The lens should be discarded and not replaced until the infection has completely resolved. In patients younger than 2 months, **sulfacetamide** 10% solution 1 drop every 2 to 3 hours for 5 to 7 days may be used. Gentamicin has fallen out of favor due to the high incidence of ocular irritation.

Drug	Indication	Dose
Cyclopentolate*	Short-term mydriasis and cycloplegia for examination	0.5% in children, 1 drop; 1% in adults, 1 drop; onset 30 min, duration $\leq$ 24 h
Tropicamide*	Short-term mydriasis and cycloplegia for examination	1 to 2 drops of 0.5% or 1% solution, onset 20 min; duration of action 6 h
Homatropine*	Intermediate-term pupil dilation, cycloplegia, treatment of iritis	1 to 2 drops of 2% solution; onset 30 min; duration of action 2 to 4 days; for iritis 1 to 2 drops twice a day
Naphazoline and* pheniramine	Conjunctival congestion/itching	1 drop 3 to 4 times a day
Olopatadine	Allergic conjunctivitis	0.1% solution, 1 drop twice daily, onset of action 30 to 60 min, duration 12 h
Tetracaine ophthalmic solution	Anesthetic for eye examination, foreign body removal	0.5% solution, 1 to 2 drops; onset of action 1 min, duration 30 min

Proparacaine ophthalmic solution	Anesthetic for eye examination foreign body removal	0.5% solution, 1 to 2 drops; onset of action 20 s, duration 15 min
Erythromycin ophthalmic ointment	Conjunctivitis. Do not use for corneal abrasion if a contact lens wearer	One-half inch applied to lower eyelid 2 to 4 times/day
Ciprofloxacin	Conjunctivitis, corneal abrasion if a contact lens wearer	Solution: 1 to 2 drops when awake every 2 h for 2 days; ointment, half inch applied to lower eyelid 3 times a day for 2 days
Tobramycin	Conjunctivitis, corneal abrasions if a contact lens wearer	0.3% solution, 1 to 2 drops every 4 h; 0.3% ointment, half inch applied to lower lid 2 to 3 times/day
Gentamicin	Conjunctivitis, corneal abrasion if a contact lens wearer	0.3% solution, instill 1 to 2 drops every 4 h; 0.3% ointment, half inch applied to lower lid 2 to 3 times/day

\*Agents that affect pupillary dilation, or serve as a conjunctival decongestant should be avoided in patients with glaucoma.

**TABLE 149-1** Common Ophthalmic Medications Used in the ED

A severe purulent discharge with a hyperacute onset (within 12 to 24 hours) should prompt an emergent ophthalmology consultation for an aggressive workup for possible gonococcal conjunctivitis. *Neisseria gonorrhoea* infections may be confirmed by Gram stain (gram-negative intracellular diplococci). Emergency department (ED) care for *N gonorrhoea* infections include culture, antibiotics (**ceftriaxone** 1 gram IM) and **saline solution irrigation** to remove the discharge; for corneal involvement, use **ceftriaxone** 1 gram IV q12h, **tobramycin** 1 drop q1h, and **doxycycline** 100 milligrams PO bid (for possible chlamydial coinfection), and **saline solution irrigation** 4 times daily.

If bacterial conjunctivitis is present in a neonate, STDs are of primary concern. These are sight-threatening conditions which can be difficult to diagnose, and an emergent ophthalmology consult in the ED is warranted. If gonorrhoea is suspected, an inpatient workup for disseminated disease is indicated. If chlamydia, then associated pneumonia must be ruled out prior to discharge. Herpes is also of concern. (See Herpes Simplex below).

## **VIRAL CONJUNCTIVITIS**

Viral conjunctivitis presents as watery discharge, chemosis, and conjunctival inflammation. It is often associated with viral respiratory symptoms and occasionally preauricular lymphadenopathy. Fluorescein staining should be done as in bacterial conjunctivitis, specifically in this case to rule out dendritic lesions. Treatment consists of cool compresses 4 times daily, **naphazoline/pheniramine** 0.025%/0.3% 1 drop 3 times daily, as needed, for conjunctival congestion or itching, **artificial tears** 5 or 6 times a day, and ophthalmology follow-up in 7 to 14 days. If a clear distinction between viral and bacterial etiologies cannot be made, consideration should be made to add topical antibiotics ([Table 149-1](#)) until reexamination by an ophthalmologist; however, routine use of antibiotics is discouraged. All cases of viral conjunctivitis are extremely contagious, and appropriate transmission precautions must be taken.

## Allergic Conjunctivitis

Allergic conjunctivitis presents as pruritus, watery discharge, and chemosis with a history of allergies. There should be no lesions with fluorescein staining and preauricular nodes should be absent. Conjunctival papillae are seen on slit lamp examination. Treatment consists of elimination of the inciting agent, cool compresses 4 times daily, **artificial tears** 5 or 6 times daily, and **naphazoline/pheniramine** 0.025%/0.3% 1 drop 4 times daily. Severe cases may require a mild topical steroid such as **fluorometholone** 0.1% 1 drop 4 times daily for 7 to 14 weeks, but should be only administered in consultation with an ophthalmologist.

## Herpes Simplex Virus

Herpes simplex virus (HSV) infection may involve the eyelids, conjunctiva, or cornea. The hallmark dendrite of herpes keratitis appears as a linear branching, epithelial defect with terminal bulbs that stain brightly with fluorescein dye during slit lamp examination. It is essential that HSV infection not be confused with conjunctivitis; hence, the necessity of a slit lamp fluorescein examination in these patients. If the outbreak involves only the eyelids, **acyclovir** 800 milligrams PO 5 times daily for 7 to 10 days should be prescribed. If the conjunctiva is involved, **trifluorothymidine** 1% drops or **vidarabine** 3% ointment 5 times daily should be prescribed. In addition, **erythromycin** ophthalmic 0.5% ointment twice daily and warm soaks 3 times daily to skin lesions can help prevent secondary bacterial infections. If corneal involvement is present, then the **trifluorothymidine** 1% drops *9 times* daily or **vidarabine** 3% ointment 5 times daily is used. If an anterior-chamber reaction is present, a cycloplegic agent such as **scopolamine** 0.25% 1 drop 3 times daily can be used. **Acyclovir** 800 milligrams PO 5 times daily or **famciclovir** 500 milligrams 3 times daily for 7 to 10 days may be considered if compliance with topic medications is questionable. Topical steroids are to be strictly avoided. All treatment should be performed in consultation with an ophthalmologist, and follow-up within 1 to 2 days should be scheduled.

When conjunctivitis is present in the neonate, herpes also should be suspected, even in the absence of maternal infection. As with other causes of neonatal conjunctivitis, an emergent ophthalmology consult in the ED is warranted. If herpes is diagnosed or strongly suspected, IV **acyclovir** (20 milligrams/kilogram IV every 8 hours), **trifluorothymidine** 1% drops 9 times daily or **vidarabine** 3% ointment 5 times daily, and a workup for sepsis is indicated.

## **Herpes Zoster Ophthalmicus**

Shingles in a trigeminal distribution with ocular involvement is termed *herpes zoster ophthalmicus* (HZO). The presence or eventual development of HZO should be suspected in any patient whose shingles involve the tip of the nose (Hutchinson sign). Photophobia and pain secondary to iritis are often present. Slit lamp examination may show a “pseudodendrite,” a poorly staining mucus plaque without epithelial erosion. **Acyclovir** 800 milligrams PO 5 times a day, **famciclovir** 500 milligrams 3 times daily, or **valacyclovir** 1000 milligrams 3 times daily for 7 to 10 days should be prescribed if the skin lesions are younger than 7 days. In addition, **erythromycin** 2% **ointment** and warm compresses should be applied to skin lesions. Ocular involvement requires **erythromycin** 0.5% ophthalmic ointment to the eye twice daily. For comfort, oral opioid analgesia, cycloplegic agents (**cyclopentolate** 1% 1 drop 3 times daily), and cool compresses are helpful. If iritis is present, **prednisolone acetate** 1% 1 drop every 1 to 6 hours is effective. However, because topical steroid use in patients with herpes simplex keratoconjunctivitis may be catastrophic, it is imperative that there be no corneal lesions present on slit lamp examination before topical steroids are used. In severe cases, admission and **acyclovir** IV may be required. For this reason, all cases of suspected HZO require an ophthalmology consultation. All patients younger than 40 years with HZO should undergo an outpatient medical evaluation for a possible immunocompromised state.

## **Corneal Ulcer**

A corneal ulcer is a serious infection of the corneal stroma caused by bacteria including *Pseudomonas aeruginosa*, viruses including Herpes simplex and Varicella zoster, and fungi. Immunocompromised patients are at risk for fungal or viral etiologies. It is commonly associated with trauma, especially in patients who use extended-wear contact lenses and those who wear lenses while sleeping. Ulcers cause pain, redness, tearing, and photophobia. Slit lamp examination shows a staining corneal defect with a surrounding white hazy infiltrate, and associated iritis and sometimes a hypopyon. Topical **ofloxacin** 0.3% or **ciprofloxacin** 0.3% ophthalmic solution should be administered, 1 drop in the affected eye each hour. Antifungal or antiviral antibiotics may be given to immunocompromised patients in consultation with an ophthalmologist. Topical cycloplegics, such as **cyclopentolate** 1% 1 drop 3 times daily, aid in pain relief. Eye patching is strictly contraindicated because of the risk of worsening a potential *Pseudomonas* infection. It would be ideal for the patient to be seen by an ophthalmologist in the ED in order to culture the ulcer prior to starting antibiotics. However, if this is not possible, they should see the patient within 12 to 24 hours.

## **Iritis**

Iritis is inflammation of the anterior uveal tract (iris and ciliary body) that has many causes ([Table 149-2](#)). It presents with red eye, photophobia, and decreased vision. A hallmark physical examination sign is *consensual pain* (pain in the affected eye when light is shined in the nonaffected eye). Slit lamp examination reveals WBCs in the anterior chamber, usually with associated flare, and a hypopyon can eventually occur. Fluorescein exam should be done because possibly etiologies may be detected (corneal abrasion, ulcer, or dendrite). Once iritis is diagnosed, an appropriate ED workup for a systemic etiology should be undertaken. Treatment is directed toward the underlying cause, if found, and symptomatic treatment with **homatropine** or **tropicamide** ([Table 149-1](#)) is helpful. Ophthalmology consultation is appropriate with follow-up in 24 to 48 hours. Steroid drops can be of value, but should only be given if directed by the ophthalmologist.

Systemic Diseases	Malignancies
Juvenile rheumatoid arthritis	Leukemia
Ankylosing spondylitis	Lymphoma
Ulcerative colitis	Malignant melanoma
Reiter syndrome	Trauma/environmental
Behcet syndrome	Corneal foreign body
Sarcoidosis	Posttraumatic (blunt trauma)
Infectious	Ultraviolet keratitis
Tuberculosis	
Lyme disease	
Herpes simplex	
Toxoplasmosis	
Varicella zoster	
Syphilis	
Adenovirus	

**TABLE 149-2** Differential Diagnosis of Iritis

## Endophthalmitis

Endophthalmitis is an infection involving the deep structures of the globe. Patients present with pain and visual loss. It is often seen as a complication of globe violation either from trauma or ocular surgery. Hematologic spread is possible. Pathogens include *Staphylococcus*, *Streptococcus*, *Haemophilus* and *Bacillus*. Emergency ophthalmology consultation and admission are warranted. Treatment includes intraocular and systemic antibiotics. Initial empiric treatment is **vancomycin** 1 milligram in 0.1 mL normal saline (N/S) and **ceftazidime** 2.25 milligrams in 0.1 mL N/S intravitreally (note not IV). Systemic antibiotics are added if bacteremia is suspected. Considerations include **clindamycin** 600 to 900 milligrams IV q8h, **ceftazidime** 2 grams IV q8h, and **amikacin** 7.5 milligrams/kilogram IV once, then 6 milligrams/kilogram q12h. Steroids intravitreally or orally may be used by the ophthalmologist.

## TRAUMA

### Subconjunctival Hemorrhage



This injury is a disruption of conjunctival blood vessels, typically secondary to trauma, sneezing or the Valsalva maneuver. It requires no treatment and usually resolves within 2 weeks. Its primary clinical importance rests in the fact that it can be a sign of significant eye injury when due to trauma, and recurrent episodes should prompt an evaluation for a coagulopathy.

## **Corneal Abrasion and Ultraviolet (UV) Keratitis**

Traumatic abrasions may cause superficial or deep epithelial defects resulting in tearing, photophobia, blepharospasm, and pain. Administration of a topical anesthetic often will facilitate the examination. **Proparacaine** 0.5% is preferred over tetracaine because it causes less pain upon administration and provides comparable anesthesia. A corneal abrasion will glow green during a fluorescein stain examination when using the cobalt blue light on the slit lamp. A careful search for an ocular foreign body (including upper lid eversion) must be done in the presence of an abrasion, especially when they are multiple and linear. Once the diagnosis of a simple abrasion is made, **opioid analgesia** should be considered for severe pain. In addition, a **cycloplegic** (eg, **cyclopentolate** 1% 1 drop 3 times a day) is traditionally recommended for pain relief, although solid evidence of efficacy is lacking. Simple abrasions are treated with topical antibiotics ([Table 149-1](#)). Most patients are treated with erythromycin ointment; however, abrasions associated with contact lens wear should be treated with ciprofloxacin, ofloxacin, or tobramycin. **Tetanus** status should be updated. Patching corneal abrasions traditionally has been recommended; however, excellent patient comfort can be achieved without it. More importantly, patching does not hasten the healing of abrasions and can greatly harm the patient if the lesions prone to infection (eg, contact lens abrasions or corneal ulcers) are patched. Prescribing topical anesthetics is absolutely contraindicated because repeated use may cause catastrophic corneal damage. All abrasions should be reexamined in 24 hours by an ophthalmologist.

Exposure to UV light from welding (“arc welder’s keratitis”), tanning beds, or prolonged sun exposure (especially during eclipses) can cause a

diffuse burn to the cornea which appears as diffuse punctate corneal abrasions with edema. Severe pain and photophobia develop 6 to 12 hours after exposure. Treatment is similar to corneal abrasions, but more aggressive pain control is sometimes necessary.

## **Corneal Foreign Bodies**

Superficial foreign bodies of the cornea are removed under slit lamp microscopy with a 25-gauge needle, an eye spud, or an ophthalmic burr. Topical anesthesia (eg, **proparacaine 0.5%**) is used (also instilled in the unaffected eye to depress reflex blinking). For obvious reasons, this procedure should be attempted only in a sober, cooperative patient. Any corneal foreign body deep within the corneal stroma or in the central visual axis should be removed by an ophthalmologist. Metallic foreign bodies often leave an epithelial “rust ring” that may be removed immediately with an **eye burr**; however, it is often easier to remove in 24 to 48 hours. A corneal abrasion will result from foreign body removal and is treated in the standard manner (**cycloplegics, antibiotics**). All patients should be referred to an ophthalmologist within 24 hours.

## **Lid Lacerations**

Many small superficial lacerations to the eye lids can be repaired by emergency physicians (see also [Chapter 11](#)), however, eyelid lacerations that involve the lid margin, those within 6 to 8 mm of the medial canthus or involving the lacrimal duct or sac, those involving the inner surface of the lid, wounds associated with ptosis, and those involving the tarsal plate or levator palpebrae muscle need repair by an ophthalmologist or plastic surgeon. Lid margin lacerations > 1 mm require closure under magnification by an ophthalmologist, whereas those <1 mm can heal spontaneously. For medial lid lacerations, injury to the lacrimal canaliculi and puncta must be excluded. Fluorescein instilled into the tear layer that appears in an adjacent laceration confirms the injury. Upper lid lacerations that involve the levator mechanism and all through-and-through lid lacerations must be repaired in

the operating room. If an ophthalmologist is not immediately available to evaluate a high-risk lid laceration, it is not unreasonable to prescribe **cephalexin** 500 milligrams PO 4 times daily, **erythromycin** 2% ointment 4 times daily, and gentle cold compresses with referral for ophthalmic evaluation within 24 hours, as long as any sight-threatening lesions have been excluded.

## **Blunt Eye Trauma**

An eye speculum (or 2 bent paper clips) are useful in visualization of the bluntly injured eye, but care should be taken to avoid any pressure on the globe. Once the eye is visualized, the integrity of the globe and visual acuity must be assessed immediately. Signs such as an abnormal anterior chamber depth, an irregular pupil, or blindness indicate a ruptured globe until proven otherwise, and an emergent ophthalmology referral is indicated. An **eye shield** should be placed as soon as a globe injury is suspected. If the globe and vision are preserved, signs of hyphema and blowout fracture should be sought. A complete slit lamp examination and a funduscopy examination is facilitated by dilation with **tropicamide** 1% 1 drop (**phenylephrine** 2.5% 1 drop in noncaucasians with brown eyes). Particular attention should be paid to check for abrasions, lacerations, foreign bodies, hyphema, pupillary injury, iritis, and lens dislocation. A **CT of the orbit** is the ED test of choice to confirm the presence of ruptured globe and orbital fractures, but sensitivity can be as low as 56% for occult globe injury; therefore, the involvement of an **ophthalmologist** is mandatory for possible surgical exploration in high-risk or suspicious cases. Traumatic iritis in the absence of a corneal injury can be treated with **prednisolone acetate** 1% 1 drop every 6 hours and **cyclopentolate** 1% 1 drop every 8 hours. The care of the blunt trauma eye patient should be discussed with an ophthalmologist, and the patient should follow-up with the ophthalmologist within 48 hours even if no significant injuries are initially found.

## **Hyphema**

A hyphema is the presence of blood in the anterior chamber and often is a sign of significant trauma. It also can occur spontaneously in sickle cell patients and in patients with coagulopathies. Sight-threatening increases in IOP can occur. In all hyphemas, emergent evaluation by an ophthalmologist is indicated. **The patient should be placed either upright or HOB to 30° to 45°** to allow the blood to settle inferiorly, which allows faster improvement of vision and facilitates assessment of the hyphema size and posterior pole. A **protective eye shield** should be in place, except during examination and medication administration. After ruptured globe is excluded, the patient should be evaluated for other eye injuries and treated appropriately. After ruptured globe is excluded, IOP should be measured. A 2001 Cochrane systematic review concluded found no evidence of improved visual acuity with traditional treatments of traumatic hyphema, including antifibrinolytic agents, corticosteroids, cycloplegics, miotics, aspirin, conjugated estrogens, patching, elevation of head, and bed rest. However, antifibrinolytic agents (aminocaproic acid or tranexamic acid) were associated with decreased incidence of rebleeding, but these agents should only be administered by an ophthalmologist. Because of the risk of rebleed in 3 to 5 days and the potential necessity of surgical intervention, any disposition decisions should be made by an ophthalmologist at the bedside, regardless of the size of the hyphema.

## **Blowout Fractures**

Orbital blowout fractures commonly involve the inferior wall and medial wall. The resultant entrapment of the inferior rectus muscle may cause restriction of movement, with a resultant diplopia on upward gaze. Other signs include paresthesia in the distribution of the infraorbital nerve and subcutaneous emphysema, particularly when sneezing or blowing the nose. Plain radiographs are of little utility in these patients; therefore, if a blowout fracture is suspected, CT of the orbit with 1.5-mm cuts should be performed, with additional studies as indicated. Because of the high incidence of associated ocular trauma (30%), an aggressive effort should be made to exclude associated injuries. Antibiotic prophylaxis (**cephalexin** 250 milligrams to 500 milligrams PO 4 times daily for 10 days) is recommended

due to sinus involvement. Isolated blowout fractures, with or without entrapment, require early referral to an ophthalmologist.

## **Penetrating Trauma or Ruptured Globe**

Globe penetration or rupture is a catastrophic injury that must be identified immediately. Suggestive findings include a severe subconjunctival hemorrhage, shallow or deep anterior chamber as compared with the other eye, hyphema, teardrop-shaped pupil, limitation of extraocular motility, extrusion of globe contents, or a significant reduction in visual acuity. A penetrating injury should be suspected when the history of a high-speed foreign body (eg, the patient was hammering or grinding without eye protection) or a penetrating injury in proximity of the orbit is present. A bright-green streaming appearance to fluorescein instilled into the tear layer (Seidel test) is pathognomonic, although it may be absent if the wound has sealed. Therefore, the presence of an abrasion with this mechanism does not rule out a penetrating injury. Once a globe injury is suspected, any further manipulation or examination of the eye must be avoided. In such cases, the patient should be placed upright and kept *npo*. A protective metallic eye shield should be put in place, and a first generation cephalosporin should be administered (**cefazolin** 1 gram IV) with an antiemetic (to prevent Valsalva). Tetanus status should be updated. Studies have shown plain x-rays are a poor screen for injury or foreign body in these cases. A CT of the orbit is the ED test of choice to confirm the presence an orbital foreign body or a ruptured globe. However, as noted above, even CT sensitivity can be low for occult globe injury; therefore, an ophthalmologist should be called immediately if a globe rupture or a penetrating injury is strongly suspected.

## **Chemical Ocular Injury**

Acid and alkali burns are managed in a similar manner. The eye should be flushed immediately at the scene and sterile **normal saline** or **Ringer lactate irrigation solution** should be continued in the ED immediately upon arrival (even before visual acuities or patient registration) until the pH is

normal (7.0 to 7.4). A **topical anesthetic** and a Morgan lens are used in this procedure. After the first 2 L of irrigation, the pH may be checked in the lower cul-de-sac with litmus paper or the pH square on a urine dipstick 5 to 10 min after ceasing irrigation (to allow time for equilibration). Required irrigation volumes to reach normal pH may exceed 8 to 10 L, depending on the caustic substance. A persistently abnormal pH should prompt removal of any crystallized particles in the fornices with a moistened cotton-tipped applicator. Once the pH is normal, the fornices should be inspected and the eyelids everted to look for any residual particles and reswept with a moistened cotton-tipped applicator to remove them and any necrotic conjunctiva. The pH should be rechecked in 10 min to make sure that no additional corrosive is leaching out from the tissues. A thorough slit lamp examination, with lid eversion, should be done to assess the amount of damage and any associated injuries. IOP should be measured because it can become elevated with significant burns. A cycloplegic (**homatropine** 5% or **cyclopentolate** 1%) 1 drop 3 times daily will alleviate ciliary spasm, and **erythromycin** 0.5% ophthalmic ointment applied every 1 to 2 hours while awake should be prescribed. Most patients will require **opioid pain medications**. Tetanus should be updated. If there are signs of a severe injury, such as a pronounced chemosis, conjunctival blanching, epithelial defect, corneal edema or opacification, or increased IOP, the patient should be seen in the ED by an ophthalmologist. Certain specialized burns, such as those due to hydrofluoric acid, lye, or concrete, also should be seen immediately. Otherwise, a telephone consult with the ophthalmologist to arrange close follow-up within 24 hours should be obtained for all burns.

## **Cyanoacrylate (Super Glue or Crazy Glue) Exposure**

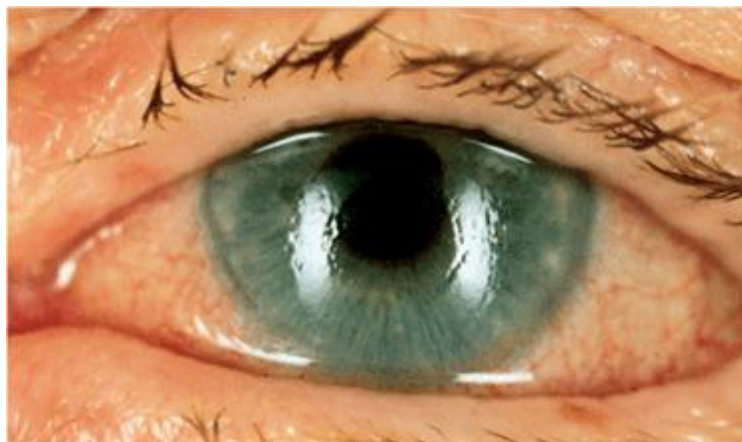
Cyanoacrylate glue easily adheres to the eyelids and corneal surface. Its primary morbidity stems from corneal injuries from the hard particles that form. Initial manual removal is facilitated by heavy application of **erythromycin** 0.5% ophthalmic ointment, with special care to avoid damaging underlying structures. After the easily removable pieces are removed, the patient should be discharged with **erythromycin** 0.5% ophthalmic ointment to be applied 5 times a day to soften the remaining

glue. Complete removal of the residual glue can be accomplished by the ophthalmologist at a follow-up visit within 24 hours. Serious injury from this exposure is rare.

## ACUTE VISUAL REDUCTION OR LOSS

### Acute Angle Closure Glaucoma

Acute angle closure glaucoma classically presents with eye pain or headache, cloudy vision, colored halos around lights, and the patient may be vomiting. Physical examination may reveal conjunctival injection, corneal clouding, (see [Fig. 149-1](#)) a fixed mid-dilated pupil, and increased IOP of 40 to 70 mm Hg (normal range, 10 to 20 mm Hg). Nausea and vomiting are also common. Sudden attacks in patients with narrow anterior chamber angles can be precipitated in movie theaters, while reading, and after illadvised use of dilatory agents or inhaled anticholinergics or cocaine. All cases require immediate ophthalmologic consultation as recent data suggest that immediate **argon laser peripheral iridoplasty (ALPI)** or **anterior chamber paracentesis** may be first-line treatments in these patients. Simultaneous to the ophthalmology consult, attempts to decrease the IOP ([Table 149-3](#)) should begin immediately. Medications to administer include: **timolol** 0.5%, **apraclonidine** 1.0%, and PO **acetazolamide** in the absence of contraindications (eg, acetazolamide is contraindicated in sickle cell and sulfa allergic patients). If IOP is greater than 50 mm Hg, if vision loss is severe, or if the patient cannot tolerate PO then **IV acetazolamide** should be considered. If IOP does not decrease and vision does not improve in 1 hour, **mannitol** should be given. **Pilocarpine** 1% to 2% 1 drop every 15 min for 2 doses in the affected eye and **pilocarpine** 0.5% 1 drop in contralateral eye may be given once the IOP is below 40 mm Hg as long as the patient has a natural lens in place. Pilocarpine is contraindicated in aphakic and pseudophakic patients or when there is mechanical closure of the angle. Some experts recommend giving pilocarpine immediately upon diagnosis. Symptoms of pain and nausea should be treated, and the IOP should be monitored hourly. Subsequent treatment decisions and disposition of the patient should be made by an ophthalmologist at the bedside.



**FIGURE 149-1.** Acute angle-closure glaucoma. The cornea is cloudy, and there is marked conjunctival injection. (Reproduced with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ: *Atlas of Emergency Medicine*, 3rd ed. © McGraw-Hill Companies, Inc. All rights reserved. Photo contributor: Kevin J. Knoop, MD, MS.)

Treatment	Effect
Topical $\beta$ -blocker (timolol 0.5%), 1 drop	Block production of aqueous humor
Topical $\beta$ -agonist (apraclonidine 1%), 1 drop	Block production of aqueous humor
Carbonic anhydrase inhibitor (acetazolamide) 500 milligrams IV or PO	Block production of aqueous humor
Mannitol, 1 to 2 grams/kilogram IV	Reduces volume of aqueous humor
Recheck IOP hourly	—
Topical pilocarpine 1% to 2%, 1 drop every 15 min for 2 doses; once IOP is below 40 mm Hg, then 4 times daily	Facilitating outflow of aqueous humor

IOP = intraocular pressure.

**TABLE 149-3** Treatment of Acute Glaucoma

## Optic Neuritis

Optic neuritis (ON) refers to inflammation at any point along the optic nerve and presents with acute vision loss, with a particular reduction in color vision. It is strongly associated with multiple sclerosis (MS). It is often



painful (> 50%), especially with extraocular movements. A decrease in color vision can be diagnosed by the “red desaturation test.” During this test, the patient looks at a dark red object with 1 eye, and then the color vision in the other eye is tested. The affected eye will see the object as pink or light red. An afferent pupillary defect (APD) often also can be detected, and visual field defects may be present. In anterior optic neuritis, the optic disc appears swollen (papillitis); there are no ophthalmoscopic findings in retrobulbar cases. An ophthalmologist should direct evaluation and treatment. IV steroids, followed by oral steroids have been shown to accelerate visual recovery and temporarily reduce the risk of developing MS, but oral steroids alone actually increase the rate of ON recurrence. A new diagnosis of optic neuritis should prompt a workup for MS.

## **Central Retinal Artery Occlusion**

Central retinal artery occlusion presents as a sudden, painless, severe monocular loss of vision, often associated with a history of amaurosis fugax. Occlusion of the central retinal artery will cause complete visual loss, whereas arterial branch obstruction will cause abrupt loss of a partial visual field. Classic signs include nearly complete or complete vision loss (94% with counting fingers to light perception only), a marked afferent pupillary defect (APD), superficial opacification or whitening of the retina in the posterior pole, and a bright red macula “cherry red spot.” Segmentation of the blood column in the arterioles (“boxcarring”) sometimes can be seen. A thorough evaluation to uncover the embolic source (commonly carotid or cardiac) is required, and giant cell arteritis must be excluded. An ophthalmologist should be contacted immediately once the diagnosis is made. In the past, digital massage, acetazolamide, and timolol have been recommended, but evidence to support these interventions is sparse. Therefore, management should be directed by an ophthalmologist per institutional protocols.

## **Central Retinal Vein Occlusion**

Central retinal vein occlusion causes acute, painless monocular vision loss. Examination shows optic disc edema, cotton wool spots, and retinal hemorrhages in all 4 quadrants. This pattern is described as “blood-and-thunder fundus.” IOP should be measured. There is no immediate treatment for central retinal vein occlusion, but predisposing drugs (eg, oral contraceptives or diuretics) should be discontinued. An ophthalmology consult should be obtained.

## **Retinal Detachment and Floaters**

Patients who experience sudden change in their vision due to retinal detachment or “floaters” in their visual field usually seek medical attention in the ED. Bilateral complaints are almost always intracranial and may be due to migraine headaches. Monocular symptoms are usually due to disorders in the symptomatic eye. Retinal detachment typically presents as a sudden dark veil or curtain-like defect in the patients visual field, affecting the symptomatic eye. Presumptive diagnosis can be made by bedside ultrasound ([Fig. 149-2](#)). Urgent ophthalmologic consultation is necessary for indirect ophthalmoscopic evaluation, and potentially laser surgery. In contrast, new “floaters” which are small particles of vitreous gel appearing to the patient as small hazy opacities, need no immediate attention, and can be followed up by the ophthalmologist in the office within 1 week.



**FIGURE 149-2.** Retinal detachment is seen as a hyperechoic membrane in the posterior aspect of the globe (arrow). (Courtesy of D. Chandwani and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.)

### **Temporal Arteritis (Giant cell arteritis)**

Temporal arteritis (TA) is a systemic vasculitis that can cause a painless ischemic optic neuropathy. Patients are typically women older than 50 years, often with a history of polymyalgia rheumatica. Associated symptoms include vision changes, headache, jaw claudication, scalp or temporal artery tenderness, fatigue, fever, sore throat, URI symptoms, and anorexia. One-third of cases have associated neurologic events such as transient ischemic attacks or stroke. An APD is frequently present, and fundoscopic examination may show flame hemorrhages. A sixth cranial nerve palsy may occur. When TA is suspected, a sedimentation rate (ESR) and C-reactive protein (CRP) should be ordered; both are elevated in TA, with the CRP the more sensitive test. Most biopsy-proven cases have an ESR in the range of

70 to 110 mm/h. If TA is not treated, bilateral vision loss can develop. Therefore, if there is strong suspicion of TA or vision loss is present, the patient should be admitted for **methylprednisolone** 250 milligrams IV every 6 hours. For less suspicious patients with no vision loss, they may be discharged with **prednisone** 80 to 100 milligrams/d PO with close follow-up. Steroids should not be delayed pending results of a biopsy. Antiulcer medications should be prescribed to be given with systemic steroids.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 236, “Eye Emergencies,” by Richard A. Walker and Srikar Adhikari; and Chapter 115, “Eye Problems in Infants and Children,” by Thomas A. Mayer, Katherine Fullerton, and Bill Bosley.

# CHAPTER 150

## Face and Jaw Emergencies

Jeffrey G. Norvell

### FACIAL INFECTIONS

#### Facial Cellulitis

Cellulitis is a soft tissue infection that involves the skin and subcutaneous tissues. Facial cellulitis is most commonly caused by *Streptococcus pyogenes* and *Staphylococcus aureus*, with an increasing predominance of methicillin-resistant *Staphylococcus aureus* (MRSA). Less commonly, cellulitis may represent an extension from a deeper facial infection. Cellulitis is characterized by erythema, edema, warmth, pain, and loss of function. Clinical features of a well-defined, palpable border are absent.

The diagnosis of cellulitis is clinical. Laboratories and blood cultures may be needed for severe illness, immunocompromise, or other significant comorbidities. Ultrasound and computed tomography (CT) may be used to evaluate for abscess. In most cases, treatment involves analgesics and oral antibiotics for 7 to 14 days. Antibiotic recommendations are listed in [Tables 150-1](#) and [150-2](#). Consider hospitalization and parenteral antibiotics for patients with signs of systemic illness, failed outpatient therapy, or significant comorbidities.

Cellulitis	<p>Oral therapy: clindamycin, dicloxacillin, or cephalosporins</p> <p>Suspected MRSA: trimethoprim-sulfamethoxazole, clindamycin, doxycycline, or minocycline</p> <p>Parenteral therapy: vancomycin, clindamycin</p> <p>Total duration 7 to 14 days</p>
Erysipelas	<p>Oral therapy: penicillin</p> <p>Methicillin-sensitive <i>Staphylococcus aureus</i> suspected: amoxicillin/clavulanate, cephalexin, dicloxacillin</p> <p>Bullous erysipelas: trimethoprim-sulfamethoxazole, clindamycin, doxycycline, or minocycline</p> <p>Parenteral therapy: vancomycin, nafcillin, clindamycin</p> <p>Total duration 7 to 14 days</p>
Impetigo	<p>Topical: mupirocin or retapamulin ointment alone or with oral therapy</p> <p>Oral therapy: dicloxacillin, amoxicillin/clavulanate, cephalexin, MRSA suspected: clindamycin or trimethoprim-sulfamethoxazole</p> <p>Total duration 7 days</p>
Suppurative parotitis	<p>Oral therapy: amoxicillin-clavulanate, clindamycin, or cephalexin and metronidazole.</p> <p>Parenteral therapy: nafcillin, ampicillin-sulbactam, or vancomycin with flagyl</p> <p>Hospital acquired or nursing home patients: include vancomycin</p> <p>Total duration: 10 to 14 days</p>
Masticator space infection	<p>Parenteral therapy: IV clindamycin is recommended; alternatives include ampicillin-sulbactam, cefoxitin, or the combination of penicillin with metronidazole</p> <p>Oral therapy: clindamycin or amoxicillin-clavulanate</p> <p>Total duration: 10 to 14 days</p>

Key: MRSA = methicillin-resistant *Staphylococcus aureus*.

**TABLE 150-1** Antibiotic Therapy for Facial Infections

Antibiotic	Dosage
<b>Topical Antibiotics</b>	
Mupirocin ointment	2%, apply to lesions 3 times per day
Retapamulin ointment	1%, apply to lesions 2 times per day
<b>Oral Antibiotics</b>	
Amoxicillin/clavulanate	875/125 milligrams 2 times per day
Cephalexin	500 milligrams 4 times per day
Clindamycin	300 to 450 milligrams 2 times per day
Didoxacillin	500 milligrams 4 times per day
Doxycycline	100 milligrams 2 times per day
Metronidazole	500 milligrams every 8 h
Minocycline	100 milligrams 2 times per day
Penicillin V	500 milligrams 4 times per day
Trimethoprim-sulfamethoxazole	1 to 2 double-strength tablets 2 times per day
<b>Parenteral Antibiotics</b>	
Ampicillin-sulbactam	1.5 to 3.0 g every 6 h
Clindamycin	600 milligrams every 8 h
Cefazolin	1 gram every 8 h
Metronidazole	1 gram loading dose, then 500 milligrams every 8 h
Nafcillin	1 to 2 grams every 4 h
Penicillin G	2 to 3 million units every 6 h
Vancomycin	1 gram every 12 h

**TABLE 150-2** Antibiotic Doses for Facial Infections

## Erysipelas

Erysipelas is a superficial form of cellulitis, involving the epidermis, upper levels of the dermis, and the lymphatic system. Most cases are caused by *S pyogenes*; *S aureus* is a rare etiology. Clinical features include a red, raised, puffy appearance with a sharply defined, palpable border. The diagnosis is clinical. Most patients are treated with oral antibiotics, but hospitalization and parenteral antibiotics should be considered for failed outpatient therapy, immunocompromise, or evidence of systemic illness. Antibiotic recommendations are listed in [Tables 150-1](#) and [150-2](#).

## Impetigo

Impetigo is a superficial epidermal infection that can be divided into bullous and nonbullous presentations. Bullous impetigo is caused by *S aureus* and nonbullous impetigo is caused by *S aureus* and *S pyogenes*. Clinical features of nonbullous impetigo include an erythematous rash with vesicles that break and form the characteristic honey crusts. Bullous impetigo presents as vesicles that enlarge to form bullae with clear yellow fluid. Topical therapy with mupirocin is appropriate for simple, nonbullous disease. Oral antibiotics are prescribed for more extensive or bullous lesions. Antibiotic recommendations are listed in [Tables 150-1](#) and [150-2](#).

## **SALIVARY GLAND DISORDERS**

### **Viral Parotitis (Mumps)**

Viral parotitis is an infection that can present with unilateral or bilateral swelling of the parotid glands. Clinical features may include a prodrome of fever, malaise, myalgias, and headache followed by parotid gland swelling. The gland is tense and painful, but lacks erythema, warmth, and pus cannot be expressed from Stensen duct.

The diagnosis is clinical. Viral serology can be ordered but does not affect acute management. Treatment is supportive. Swelling may persist for 5 days and the patient is contagious for approximately 9 days after the onset of parotid swelling. Extrasalivary gland involvement includes epididymo-orchitis in 20% to 30% of males and oophoritis in 5% of females. Other systemic complications include pancreatitis, aseptic meningitis, hearing loss, myocarditis, arthritis, hemolytic anemia, and thrombocytopenia. Consider hospitalization for those with systemic complications.

### **Suppurative Parotitis**



Suppurative parotitis is a serious bacterial infection that occurs in patients with diminished salivary flow. Retrograde transmission of bacteria leads to infection. Factors that lead to decreased salivary flow include recent anesthesia, dehydration, prematurity, advanced age, medications (eg, diuretics,  $\beta$ -blockers, antihistamines, phenothiazines, and tricyclic antidepressants), and certain disorders (Sjögren syndrome, diabetes, hypothyroidism, and human immunodeficiency virus). Clinical features may include fever, trismus, erythema, and pain over the parotid gland. Pus may be expressed from Stensen duct.

The diagnosis is clinical. Ultrasound or CT may be ordered if an abscess is suspected. Treatment should optimize salivary flow by using sialogogues such as lemon drops and stopping any medications that cause dry mouth. Oral antibiotics (see [Tables 150-1](#) and [150-2](#)) are appropriate for those tolerating oral intake, and without trismus. Hospitalization is appropriate for those with signs of systemic illness, inability to tolerate oral intake, or those that have failed outpatient therapy. Close follow-up should be arranged.

## **Sialolithiasis**

Sialolithiasis is the development of stones in a stagnant salivary duct. Eighty percent of stones occur in the submandibular duct. Sialolithiasis is typically unilateral and presents with pain, swelling, and tenderness that may be exacerbated with eating. The diagnosis is clinical and a stone may be palpated within the duct and the gland is firm. Treatment involves analgesics, massage, sialogogues such as lemon drops, and antibiotics if a concurrent infection is suspected. Palpable stones may be milked from the duct. Persistent retained calculi may be removed by an otolaryngologist.

## **Masticator Space Abscess**

The masticator space consists of potential spaces bounded by the muscles of mastication. Infection is usually polymicrobial and is commonly associated with an odontogenic source. Clinical features include facial swelling, pain, erythema, and trismus. In advanced cases, signs of sepsis may be present.

The diagnosis is made with contrast-enhanced CT scan. Because the masticator spaces ultimately communicate with tissue planes that extend into the mediastinum, early treatment is imperative. ED treatment includes stabilization, antibiotics, otolaryngology consult, and hospitalization. Antibiotic recommendations are listed in [Tables 150-1](#) and [150-2](#).

## **MANDIBLE DISORDERS**

### **Temporomandibular Joint Dysfunction**

The temporomandibular joint (TMJ) combines a hinge and a gliding action. Anatomic derangements or systemic disease can cause dysfunction of this joint. Clinical features include pain over the muscles of mastication or in the region of the TMJ and there may be a limited range of motion. The diagnosis is usually clinical. For patients with acute trauma, imaging with CT or panoramic tomography (Panorex) may be warranted. Treatment for nontraumatic conditions consists of analgesics, soft diet, and referral to dental specialist. An oral-maxillofacial surgeon manages fractures.

### **Dislocation of the Mandible**

The mandible can be dislocated in an anterior, posterior, lateral, or superior position. Anterior dislocation is the most common. Patients with an acute jaw dislocation present with pain, difficulty swallowing, and malocclusion. In anterior dislocations, a history of extreme mouth opening is typical and there is difficulty with jaw movement. Other dislocations usually require significant trauma.

The diagnosis of atraumatic anterior dislocations is clinical. Imaging with mandibular radiographs, Panorex, or CT is indicated for all other dislocations. Treatment of anterior dislocations without fracture is closed reduction and this is made easier with analgesia, muscle relaxants, or procedural sedation. Reduction of dislocated mandible technique is most commonly done in a seated patient. The thumbs are placed over the molars,

and pressure is applied downward and backward. Patients with open or nonreducible dislocations, fractures, or nerve injury should be referred emergently to an oral-maxillofacial surgeon. After reduction, patients should be instructed to not open their mouth more than 2 cm for 2 weeks.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 238, “Face and Jaw Emergencies,” by Corey R. Heitz.

# CHAPTER 151

## Ear, Nose, and Sinus Emergencies

Medley O. Gatewood

### OTOLOGIC EMERGENCIES

#### Otitis Externa

Otitis externa, or “swimmer’s ear,” is characterized by pruritus, pain, and tenderness of the external ear. Erythema and edema of the external auditory canal, otorrhea, crusting, and hearing impairment may also be present. Pain is elicited with movement of the pinna or tragus. Risk factors for development of otitis externa include swimming, trauma of the external canal, and any process that elevates the pH of the canal.

The most common organisms implicated in otitis externa are *Pseudomonas aeruginosa*, *Enterobacteriaceae* and *Proteus species*, and *Staphylococcus aureus*, with *P aeruginosa* being the most common organism causing malignant otitis externa. Otomycosis, or fungal otitis externa, is found in tropical climates and in the immunocompromised or subsequent to long-term antibiotic therapy. *Aspergillus* and *Candida* are the most common fungal pathogens.

The treatment of otitis externa includes analgesics, cleaning the external auditory canal, acidifying agents, topical antimicrobials, and occasionally topical steroid preparations. **Ofloxacin** otic 5 drops 2 times daily, **acetic acid/hydrocortisone** otic 5 drops 3 times daily (do not use with perforated TM), and **ciprofloxacin/hydrocortisone** otic 3 drops 2 times daily are commonly used for 7 days to treat otitis externa. If significant swelling of the external canal is present, a wick or piece of gauze may be inserted into the canal to allow passage of topical medications.

**Malignant otitis externa** is a potentially life-threatening infection of the external auditory canal with variable extension to the skull base. In greater

than 90% of cases, it is caused by *Pseudomonas aeruginosa*. Elderly, diabetic, and immunocompromised patients are most commonly affected. Diagnosis of malignant otitis externa requires a high index of suspicion. Computed tomography (CT) is necessary to determine the extent and stage of the disease. Emergent otolaryngologic (ENT) consultation, **tobramycin** 2 milligrams/kilogram IV and **piperacillin** 3.375 to 4.5 grams IV, or **ceftriaxone** 1 gram IV, or **ciprofloxacin** 400 milligrams IV, and admission to the hospital are mandatory.

## Otitis Media

The incidence and prevalence of otitis media (OM) peak in the preschool years and decline with advancing age. The most common bacterial pathogens in acute OM are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. The predominant organisms involved in chronic OM are *S aureus*, *P aeruginosa*, and anaerobic bacteria.

Patients with OM present with otalgia, with or without fever; occasionally, hearing loss and otorrhea are present. The tympanic membrane (TM) may be retracted or bulging and will have impaired mobility on pneumatic otoscopy. The TM may appear red as a result of inflammation or may be yellow or white due to middle ear secretions.

A 10-day course of **amoxicillin** 250 to 500 milligrams PO 3 times daily for 7 to 10 days is the preferred initial treatment for OM. Alternative agents include **azithromycin** 500 milligrams PO daily for 1 day then 250 milligrams PO daily for 4 days, or **cefuroxime** 500 milligrams PO 2 times daily for 10 days. Cefuroxime or amoxicillin/clavulanate may be given for OM unresponsive to first-line therapy after 72 hours. Antibiotic coverage should be extended to 3 weeks for patients with OM with effusion. Analgesics should be prescribed for patients with any degree of pain. Patients should follow-up with a primary care physician for reexamination and to assess the effectiveness of therapy.

Complications of OM include TM perforation, conductive hearing loss, acute serous labyrinthitis, facial nerve paralysis, acute mastoiditis, lateral sinus thrombosis, cholesteatoma, and intracranial complications. TM perforation and conductive hearing loss are most often self-limiting and

often require no specific intervention. Facial nerve paralysis is uncommon but requires emergent ENT consultation.

## **Acute Mastoiditis**

Acute mastoiditis occurs as infection spreads from the middle ear to the mastoid air cells. Patients present with otalgia, fever, and postauricular erythema, swelling, and tenderness. Protrusion of the auricle with obliteration of the postauricular crease may be present. CT will delineate the extent of bony involvement. Emergent ENT consultation, **vancomycin** 1 to 2 grams IV or **ceftriaxone** 1 gram IV, and admission to the hospital are necessary. Surgical drainage ultimately may be required.

## **Lateral Sinus Thrombosis**

This condition arises from extension of infection and inflammation into the lateral and sigmoid sinuses. Headache is common and papilledema, sixth nerve palsy, and vertigo may be present. Diagnosis may be made with CT, although magnetic resonance imaging or angiography may be necessary. Therapy consists of emergent ENT consultation, combination therapy with **nafcillin** 2 grams IV, **ceftriaxone** 1 gram IV, and **metronidazole** 500 milligrams IV, and hospital admission.

## **Bullous Myringitis**

Bullous myringitis is a painful condition of the ear characterized by bulla on the TM and deep EAC. Numerous pathogens have been implicated including viruses, *Mycoplasma pneumoniae*, and *Chlamydia psittaci*. The diagnosis is made by clinical examination. The treatment consists of pain control and warm compresses. Antibiotics can be given for concomitant OM.

## **Trauma to the Ear**

A hematoma can develop from any type of trauma to the ear. Improper treatment of ear hematomas can result in stimulation of the perichondrium and development of asymmetric cartilage formation. The resultant deformed auricle has been termed *cauliflower ear*. Immediate incision and drainage of the hematoma with a compressive dressing is necessary to prevent reaccumulation of the hematoma.

Thermal injury to the auricle may be caused by excessive heat or cold. Superficial injury of either type is treated with cleaning, topical nonsulfa-containing antibiotic ointment, and a light dressing. Frostbite is treated with rapid rewarming by using saline soaked gauze at 38°C to 40°C. The rewarming process may be very painful and analgesics will be necessary. Any second- or third-degree burn requires immediate ENT or burn center consultation.

## **Foreign Bodies in the Ear**

On examination, the foreign body is usually visualized and signs of infection or TM perforation should be sought. Live insects should be immobilized with 2% **lidocaine** solution distilled into the ear canal before removal. Foreign bodies may be removed with forceps and direct visualization or with the aid of a hooked probe or suction catheter. Irrigation is often useful for small objects; however, organic material may absorb water and swell. ENT consultation is required for cases of foreign body with TM perforation or if the object cannot be safely removed.

## **Tympanic Membrane Perforation**

TM perforations can result from middle ear infections, barotrauma, blunt/penetrating/acoustic trauma, or (rarely) lightning strikes. Acute pain and hearing loss are usually noted, with or without bloody otorrhea. Vertigo and tinnitus, when present, are usually transient. As most TM perforations heal spontaneously, antibiotics are not necessary unless there is persistent foreign material in the canal or middle ear. Patients with perforations from

isolated blunt or noise trauma can be discharged with expedited specialty referral and should be instructed not to allow water to enter the ear canal.

## **Tinnitus**

Tinnitus is the perception of sound without external stimuli. It may be constant, pulsatile, high or low pitched, hissing, clicking, or ringing in nature. Objective tinnitus can be heard by the examiner, whereas the more common subjective tinnitus cannot. Causes of tinnitus include vascular, mechanical, neurologic, Ménière disease, and other causes. Common medications resulting in tinnitus include aspirin, nonsteroidal anti-inflammatory drugs, aminoglycosides, loop diuretics, and chemotherapeutics; if the patient's condition allows, potentially offending drugs should be stopped. Accurate diagnosis usually requires referral to an otolaryngologist. Pharmacologic treatment with antidepressant medications may alleviate tinnitus in which no correctable cause can be found.

## **Hearing Loss**

Causes of sudden hearing loss are varied and may be idiopathic (most common), infectious, vascular or hematologic, metabolic, rheumatologic, or conductive. Other causes include Ménière disease, Cogan syndrome, acoustic neuroma, cochlear rupture, and ototoxic medications. Indicators of poor prognosis include severe hearing loss on presentation and the presence of vertigo. If the cause is not readily determined by history and physical examination, otolaryngologic consultation is necessary.

# **NASAL EMERGENCIES AND SINUSITIS**

## **Epistaxis**



Epistaxis is classified as anterior or posterior. Posterior epistaxis is suggested if an anterior source is not visualized, if bleeding occurs from both nares, or if blood is seen draining into the posterior pharynx after anterior sources have been controlled.

1. A quick history should determine the duration and severity of the hemorrhage and the contributing factors (trauma, anticoagulant use, infection, bleeding diathesis, etc).
2. The patient should blow their nose to dislodge any clots. Instill 0.05% **oxymetazoline** 2 sprays/nostril or 0.25% **phenylephrine** 2 sprays/nostril.
3. Inspect for anterior bleeding using a good light source, a nasal speculum and a suction catheter.
4. Direct external pressure is then applied for 15 min while leaning forward in the “sniffing” position. Reexamine. Repeat once if necessary.
5. If this approach fails, and an anterior source of bleeding is visualized, proceed to chemical cautery. If no source is identified, proceed to packing.
6. **Chemical cautery with silver nitrate** is the standard of care for emergency department (ED) cautery of anterior epistaxis. Insert cotton swabs or pledgets soaked in a 1:1 mixture of a 4% **lidocaine** and 0.05% **oxymetazoline** into the nasal cavity with bayonet forceps. After hemostasis is achieved, the mucosa is cauterized by firmly rolling the tip of a silver nitrate applicator over the area until it turns silvery-black. A small surrounding area also should be cauterized to control local arterioles. Overzealous use of cautery and bilateral septal cautery are discouraged because they may cause septal perforation and unintended local tissue necrosis.
7. **Anterior nasal packing** may be performed with thrombogenic foams and gels, commercial devices, or gauze. Dehydrated nasal sponges are available in several lengths to control anterior and posterior epistaxes. A film of antibiotic ointment is applied, then the sponge is rapidly inserted along the floor of the nasal cavity where it expands upon contact with blood or secretions. Expansion can be hastened by rehydrating the sponge with sterile water from a catheter-tipped syringe. The longer sponges used to control posterior hemorrhages have been associated with some

morbidity and should be used only for posterior epistaxis. Inflatable epistaxis tamponade balloons can also be used to control anterior and/or posterior hemorrhage, are easy to use and are generally more comfortable than nasal sponges. Thrombogenic foams and gels are bioabsorbable, do not require removal, and are generally well tolerated. All nonabsorbable nasal packs should be removed in 2 to 3 days by an ENT physician. If packing or local cautery fails to control anterior bleeding, ENT consultation is necessary.

8. **Posterior epistaxis** may be treated with a dehydrated posterior sponge pack, as outlined above, or an inflatable balloon tamponade device. The balloon devices use independently inflatable anterior and posterior balloons to quickly control refractory epistaxis at these sites; the instructions for insertion are included in the balloon kit. To protect against potentially serious complications, all patients with posterior packs require ENT consultation for possible hospital admission. Posterior packs are removed 2 to 3 days after placement.
9. All patients with nasal packs should be started on antibiotic prophylaxis with **amoxicillin/clavulanate** 500/125 milligrams PO 3 times daily.

Complications of nasal packing include dislodgment of the pack, recurrent bleeding, sinusitis, and toxic shock syndrome. Treatment of elevated blood pressure during an acute episode of epistaxis is generally not advised except in consultation with an otolaryngologist for cases of persistent epistaxis not uncontrolled by the above measures.

## **Nasal Fractures**

Nasal fracture is a clinical diagnosis suggested by the injury mechanism, swelling, tenderness, crepitance, gross deformity, and periorbital ecchymosis. Radiographic diagnosis usually is not necessary in the ED. Intermittent ice application, analgesics, and over-the-counter decongestants are the normal treatment. ENT follow-up within 6 to 10 days for reexamination and possible fracture reduction is prudent.

The nose should be examined for a septal hematoma. If left untreated, a septal hematoma may result in abscess formation or necrosis of the nasal

septum. The treatment is local **incision and drainage** with subsequent placement of an anterior nasal pack.

A fracture of the cribriform plate may violate the subarachnoid space and cause cerebrospinal fluid rhinorrhea. Symptoms may be delayed for several weeks. If a cribriform plate injury is suspected, CT and immediate neurosurgical consultation should be obtained.

## **Nasal Foreign Bodies**

Nasal foreign bodies should be suspected in patients with unilateral nasal obstruction, foul rhinorrhea, or persistent unilateral epistaxis. After topical vasoconstriction with 0.05% **oxymetazoline** and possibly local anesthesia with 4% nebulized **lidocaine**, the foreign body should be removed under direct visualization. Tools for removal include forceps, suction catheters, hooked probes, and balloon-tipped catheters. ENT consultation is required for any unsuccessful removal.

## **Sinusitis & Rhinosinusitis**

Sinusitis is inflammation of the mucosal lining of the paranasal sinuses (maxillary, frontal, ethmoid and frontal). Rhinosinusitis is sinusitis also involving the nasal cavity, almost always involves rhinitis, and is extremely common. It can be classified as acute, sub-acute, or chronic.

Symptoms include nasal congestion or blockage, facial pain or pressure, hyposmia, nasal discharge, tooth pain, fever, and sinus pressure with head/body movement. There may be pain and tenderness with sinus percussion, mucosal swelling, facial swelling and redness.

Complications include meningitis, cavernous sinus thrombosis, intracranial abscess and empyema, orbital cellulitis, and osteomyelitis. Patients with these deeper complications usually appear systemically ill or have focal neurologic findings.

The diagnosis of uncomplicated acute rhinosinusitis is clinical, and imaging is not necessary. CT scans are helpful in evaluating toxic patients and possible intracranial extension. Patients with chronic rhinosinusitis or

recurrent acute rhinosinusitis warrant bacterial cultures and a sinus CT, preferably as an outpatient.

Treatment for acute uncomplicated disease is generally supportive. Nasal irrigation with or without nasal decongestants (0.05% **oxymetazoline** 2 sprays/nostril 2 times daily or 0.25% **phenylephrine** 2 sprays/nostril 4 times daily) is first-line therapy. Decongestants use is to be limited to  $\leq 3$  days. Oral antibiotic should be reserved for patients with purulent nasal secretions and severe symptoms  $\geq 7$  days. If prescribed, choices for a 10-day antibiotic regimen include **amoxicillin** 1 gram PO 3 times daily (first-line), **trimethoprim/sulfamethoxazole** 160/800 milligrams 2 times daily or **erythromycin** 250 to 500 milligrams 2 times daily, (if penicillin allergic), and **levofloxacin** 500 milligrams daily (if antibiotics in prior 6 weeks).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 237, “Common Disorders of the External, Middle, and Inner Ear,” by Mark Silverberg and Michael Lucchesi; and Chapter 239, “Epistaxis, Nasal Emergencies and Rhinosinusitis,” by Shane M. Summers and Tareg Bey.

# CHAPTER 152

## Oral and Dental Emergencies

Steven Go

### OROFACIAL PAIN

#### Tooth Eruption and Pericoronitis

Eruption of the primary teeth (“teething”) in children may be the primary cause of pain, irritability, and drooling, but NOT fever and diarrhea; therefore, other causes of these latter symptoms must be excluded. Adequate hydration, by giving the child a frozen, damp towel to suck on and **acetaminophen** 15 milligrams/kilogram orally (PO) 6 hours usually will control symptoms. Topical anesthetics should be used with great caution in young infants due to its potential to depress the gag reflex.

Adults and teens may experience pericoronitis (pain and local inflammation) with the eruption of the third molars (“wisdom teeth”). **Penicillin VK** 500 milligrams PO 4 times daily or **clindamycin** 300 milligrams PO 4 times daily, **ibuprofen** 400 to 800 milligrams PO thrice daily (with or without **hydrocodone** 5 milligrams/ **acetaminophen** 325 milligrams 1 to 2 tablets PO 4 times daily), and **warm saline mouth rinses** will be beneficial until the third molar can be extracted by an oral surgeon or general dentist.

#### Dental Caries and Pulpitis

*Dental caries* are caused by bacteriogenic acid eroding through the enamel. Examination sometimes finds a grossly decayed tooth, although frequently there is no visible pathology—in these cases, localization may be accomplished by percussing individual teeth with a metallic object. If dental

caries are not treated, pulpitis is the result. *Reversible pulpitis* is characterized by sudden, transient pain lasting seconds, often triggered by heat or cold. In contrast *irreversible pulpitis* pain lasts minutes to hours. Although antibiotics (**penicillin VK** 500 milligrams PO 4 times daily or **clindamycin** 300 milligrams PO 4 times daily) are commonly prescribed for reversible pulpitis, their efficacy is controversial. **Ibuprofen** 400 to 800 milligrams PO thrice daily, **hydrocodone** 5 milligrams/**acetaminophen** 325 milligrams 1 to 2 tablets PO 4 times daily, **warm saline mouth rinses**, and referral to a dentist for definitive management are all reasonable. Antibiotics do not appear to improve toothache in irreversible pulpitis, but pain control and dental referral as listed above are appropriate. If an abscess is present, antibiotics, incision and drainage should be considered. (see below).

## **Facial Cellulitis**

Odontogenic infections can spread readily to the facial spaces. Therefore, it is imperative to exclude deep-space involvement whenever a dental infection is encountered. *Ludwig angina* is a cellulitis involving the submandibular spaces and the sublingual space that can spread to the neck and mediastinum, causing airway compromise, overwhelming infection, and even death. If dental infections spread to the infraorbital space, a *cavernous sinus thrombosis* may result. This condition may present with limitation of lateral gaze, meningeal signs, sepsis, and coma. Intravenous antibiotics and emergent surgical consultation are mandatory for both conditions, with anticoagulation added for cavernous sinus thrombosis. (See [Chapter 153](#) “Neck and Upper Airway Disorders” and [Chapter 150](#) “Face and Jaw Emergencies.”)

## **Postextraction Pain and Postextraction Alveolar Osteitis (Dry Socket)**

*Periosteitis* is pain experienced within 24 to 48 hours after a tooth extraction and responds well to icepacks, head elevation, and analgesics.

*Postextraction alveolar osteitis* (“dry socket”) occurs 48 to 72 hours postoperatively when the clot from the socket is displaced, causing severe pain, foul odor, and taste. Dental radiographs are indicated to exclude a retained root tip or foreign body. Treatment consists of **saline irrigation** of the socket, followed by packing the socket with **eugenol-impregnated gauze**, which will relieve the pain. **Penicillin VK** 500 milligrams PO 4 times daily or **clindamycin** 300 milligrams PO 4 times daily should be prescribed in severe cases, with daily packing changes and dental follow-up in 24 hours.

## **Postextraction Bleeding**

Bleeding after dental extraction usually is controlled by **direct pressure** for 20 min applied by biting on gauze. If bleeding persists, packing with **Gelfoam, Avitene, or Surgicel** into the socket may be effective. **Loosely approximating sutures** can be used to hold these packing agents in place. Other ED treatments may include local injection of **1% lidocaine with epinephrine** or **silver nitrate** application. Failure of these measures warrants a screening coagulation profile and consultation with an oral surgeon.

## **Periodontal Abscess**

A periodontal abscess results from plaque and debris entrapped between the tooth and gingiva. Small abscesses resolve with **penicillin VK** 500 milligrams PO 4 times daily or **clindamycin** 300 milligrams PO 4 times daily, **analgesics**, and short term **0.1% chlorhexidine mouth rinses** twice daily (to avoid tooth discoloration). Larger abscesses require incision and drainage. All patients need prompt dental referral.

## **Acute Necrotizing Ulcerative Gingivitis**

Acute necrotizing ulcerative gingivitis presents with pain, ulcerated or “punched out” interdental papillae, gingival bleeding, foul taste, lymphadenopathy, and fever. The associated putrid breath gives this disorder its nickname of “trench mouth.” It occurs mainly in patients with lowered resistance due to HIV, stress, malnourishment, substance abuse, and various infections. Treatment consists of **metronidazole** 500 milligrams PO thrice daily and **0.1% chlorhexidine mouth rinses** twice daily, along with a protein rich soft diet, multivitamins, and PO fluids. Symptomatic improvement is dramatic within 24 hours, and the patient should be referred for workup of predisposing factors.

## **SOFT TISSUE LESIONS OF THE ORAL CAVITY**

### **Oral Candidiasis**

Risk factors for candidal infection include extremes of age, immunocompromised states, use of intraoral prosthetic devices, concurrent antibiotic use, and malnutrition. Typical lesions consist of removable white, curd-like plaques on an erythematous mucosal base. Treatment consists of oral anti-fungal agents such as **nystatin oral suspension** 500 000 units/5 mL swish and swallow 4 times daily, or **clotrimazole troches** 10 milligrams 5 times daily, or **fluconazole** 100 milligrams PO daily. Treatment is continued for 2 days after symptoms and visible lesions have resolved.

### **Aphthous Stomatitis**

Aphthous stomatitis, or aphthous ulcer, presents with painful lesions, which are frequently multiple, involve the labial and buccal mucosa and measure from 2 mm to several centimeters in diameter. Treatment consists of 0.01% **dexamethasone** elixir 5 mL qid used as a mouth rinse or **0.05%**



**fluocinonide gel** applied topically to lesions. The lesions often heal within 48 h.

## **Herpes Simplex**

Herpes gingivostomatitis causes painful ulcerations of the mucosal and gingival surfaces. Fever, lymphadenopathy, and tingling often precede the eruption of numerous vesicles, which then rupture and form ulcerative lesions. Adequate pain management and hydration are paramount. In severe cases, **acyclovir**, 15 milligrams/kilogram/dose in divided doses 5 times/day (maximum daily dose, 2 grams) for 7 days is appropriate. Recurrent infections can occur and are often preceded with burning or tingling. For adults, **acyclovir** 400 milligrams PO thrice to 5 times daily for 5 days or **valacyclovir** 2000 milligrams PO twice daily for 1 day may be given during the prodromal phase to attenuate the clinical duration and severity of the outbreak.

## **Coxsackievirus Infections**

*Herpangina* presents with high fever, sore throat, headache, and malaise, followed by eruption of oral vesicles, which rupture to form painful, shallow ulcers. The soft palate, uvula, and tonsillar pillars are typically affected, whereas the buccal mucosa, tongue, and gingiva are spared (which helps distinguish it from herpes infection). *Hand, foot, and mouth disease* causes vesicles initially to form on the soft palate, gingiva, tongue, and buccal mucosa. The vesicles then rupture, leaving painful ulcers surrounded by red halos. Lesions also may appear on the buttocks, palms, and soles. Both infections last approximately 5 to 10 days. Treatment is supportive and consists of hydration and acetaminophen or NSAIDs. Topical anesthetics should be used with great caution in young infants due to its potential to depress the gag reflex. Infected children should be kept home from school until the lesions resolve.

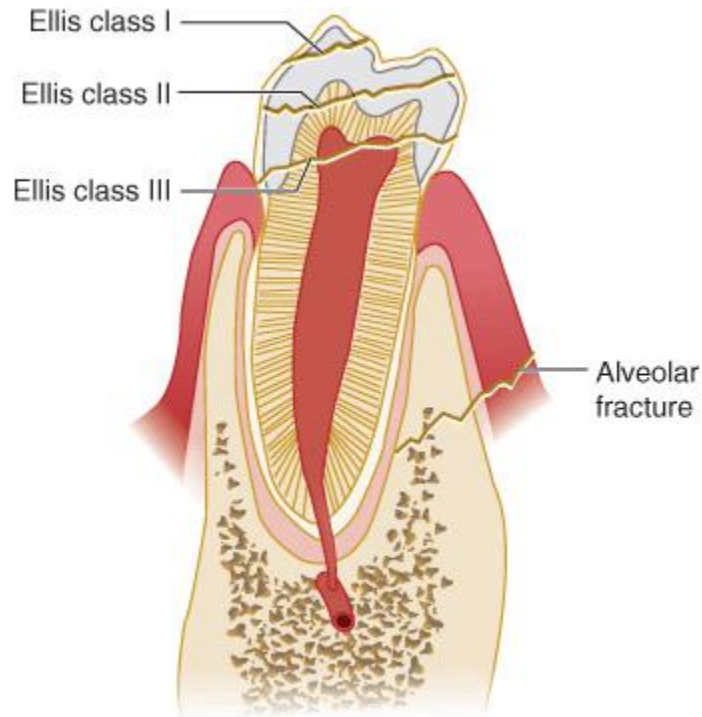
## **Oral Cancer**

Emergency physicians should be vigilant for oral cancers and their precursors because early diagnosis is associated with improved outcomes. Cancers can present early as *leukoplakia* (nonremovable white mucosal patches) and *erythroplakia* (red patch that cannot be classified as any other disease). The most common site for oral cancer to develop is the posterolateral border of the tongue. Symptoms and signs of oral cancer include pain, paresthesias, persistent ulcers, bleeding, lesion rigidity, induration, lymphadenopathy, and functional impairment. All suspicious or nonhealing lesions should prompt an urgent follow up with an oral surgeon for biopsy.

## OROFACIAL TRAUMA

### Dental Fractures

The Ellis system is used to classify the anatomy of fractured teeth. ([Fig. 152-1](#)) *Ellis class 1* fractures solely involve the enamel. These injuries may be smoothed with an emery board or referred to a dentist for cosmetic repair. *Ellis class 2* fractures reveal the creamy yellow dentin underneath the white enamel. The patient complains of air and temperature sensitivities. To decrease pulpal contamination, the dentin should be dried and promptly covered with **calcium hydroxide paste**, or **glass ionomer cement**. All patients should see a dentist within 24 hours. *Ellis class 3* fractures are tooth-threatening fractures that involve the pulp and can be identified by a red blush in the exposed dentin or a visible drop of blood after wiping the tooth. **Calcium hydroxide paste** should immediately be used as a base to cover the pulp, followed by an overlying layer of **glass ionomer cement** to completely cover the patch and dentin as well. Ideally, a dentist should evaluate the patient in the ED, but if the amount of exposed pulp is very small, the patched lesion can be evaluated by a dentist within 24 hours. Oral analgesics may be needed, but topical anesthetics are contraindicated. The use of prophylactic antibiotics is controversial.



**FIGURE 152-1.** Ellis classification for fractures of teeth.

## **Concussions, Luxations, and Avulsions**

*Concussion* injuries involve posttraumatic tenderness to percussion with no mobility. Posttraumatic tenderness to percussion and mobility without evidence of dislodgment is called *subluxation*, which has a higher incidence of future pulp necrosis. Management for these 2 entities includes **NSAIDs**, soft diet, and urgent referral to a dentist.

*Extrusive luxation* occurs when a tooth is partly avulsed out from the alveolar bone. Treatment involves gentle repositioning of the tooth to its original location (often with the aid of a dental block) and splinting with **zinc oxide** periodontal dressing. A dentist should evaluate these patients within 24 hours. When the tooth is laterally displaced with a fracture of the alveolar bone, the condition is called *lateral luxation*. Although manual relocation is possible, the treatment of such injuries is best done in consultation with a dentist. If the alveolar bone fracture is significant,

splinting by a dentist in the emergency department (ED) is required. An *intrusive luxation* occurs when the tooth is forced below the gingiva and often has a poor outcome. Treatment is the same as that of subluxation.

Dental *avulsion* is an emergency in which a tooth has been completely removed from the socket. *Primary teeth* in children are not replaced because of potential damage to the permanent teeth. *Permanent teeth* that have been avulsed for less than 3 hours must be **reimplanted immediately** to attempt to save the periodontal ligament fibers. At the scene, an avulsed tooth should be handled by the crown only, rinsed with water, and reimplanted immediately. If reimplantation at the scene is not possible due to risk of aspiration, the tooth should be rinsed and placed in a nutrient solution, such as **Hank solution** (preserves cell viability for up to 4 to 6 hours), sterile saline, or milk, and the tooth should be transported immediately with the patient to the ED. An effective, but somewhat unsettling, way to safely transport the avulsed tooth of the child is underneath the parent's tongue. Upon arrival in the ED, the clot in the socket should be removed and the socket gently irrigated with **sterile normal saline**. The tooth is then examined to determine whether the apex is open. Early consultation with a dentist is imperative, but **reimplantation with gentle pressure** should not be delayed while awaiting the arrival of the specialist. After reimplantation, adults should receive **doxycycline** 100 milligrams PO BID for 7 days. Children <12 years old should receive **penicillin VK** 12.5 milligrams/kilogram/dose 4 times a day for 7 days. If a patient arrives with an empty socket and the tooth cannot be located, adjacent tissue should be searched. Radiographs may be necessary to exclude displaced or aspirated teeth.

## **Soft Tissue Trauma**

Dental trauma should be stabilized before repairing soft tissue trauma. In addition, a thorough search for retained foreign bodies should take place before repair, and tetanus status should be updated.

Most *intraoral mucosal lacerations* will heal by themselves; however, they should be repaired if they are gaping (typically wider than 1 cm) or if flaps are present. Good anesthesia is essential. The wound should be

inspected for foreign bodies, and soft tissue radiographs may be useful to detect retained tooth fragments. The wound should be copiously irrigated, dead tissue debrided, and the laceration closed with a **5-0 absorbable suture**, taking care to bury the knots. The patient should be instructed to eat a soft diet with gentle **salt water rinses** after each meal. Adequate analgesic medication should be prescribed. Antibiotics are not generally indicated, and 48-hours follow-up is appropriate.

*Tongue lacerations* pose a special challenge due to the organ's vascularity. While massive bleeding or delayed venous swelling from tongue trauma can both obstruct the airway, the more common ED presentation involves localized injuries. The indications for closure of tongue lacerations remain controversial. Some authors recommend all lacerations be repaired while others allow nearly any wound to heal by secondary intention. Published indications for primary closure include bisection of the tongue, widely gaping wounds at rest, active bleeding, flap or U-shaped wounds, involvement of the tongue edge, and lacerations > 1 cm. There is general agreement that non-gaping, superficial, linear lacerations <1 cm require no repair. An assistant may be required to hold the tongue with gauze to allow repair. Local anesthesia can be obtained with a gauze soaked with **4% lidocaine** applied topically for 5 min. In some cases, local infiltration with **1% lidocaine with epinephrine** or a lingual nerve block (anterior 2/3 of the tongue) may be necessary. If greater control is required, an anesthetized tip of the tongue may be grasped with a towel clamp or a temporary silk suture may be placed. A dental bite block may be useful to prevent bites to health care providers during repair. Lacerations may be repaired with **4-0 or 5-0 absorbable sutures**. Sutures must be kept *loose* (to avoid necrosis in case the tongue swells significantly within the first 2 days), wide, and deep. All of the involved tongue layers should be closed with single-interrupted sutures that approximate all layers at once. Alternatively, a two- or three-layer closure technique may be used. Sometimes in children it is desirable to only close the deep muscle layer while eschewing surface layer closure. This avoids surface knots which can trouble children. Wound edges must be aligned as precisely as possible to avoid subsequent formation of clefts, which can have cosmetic and functional consequences. Every exposed stitch should be completed with several knots to avoid the suture becoming undone by subsequent tongue

movement. In about 7 days, sutures will fall out by themselves or will be absorbed. Aftercare is similar to that of other intraoral lacerations. Tongue lacerations in patients who require procedural sedation, large or complex lacerations, full or partial amputations, and difficulties with hemostasis should prompt specialty consultation. When a child presents with oral trauma, the possibility of abuse should be considered.

*Lip lacerations* can present a difficult challenge if they violate the vermilion border (the transition between lip tissue and the skin of the face). See [Chapter 14](#) “Lacerations of the Face,” for management. Laceration of the *maxillary labial frenulum* usually does not require repair. The *lingual frenulum* is very vascular and usually should be repaired with **4-0 absorbable sutures**.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 240, “Oral and Dental Emergencies,” by Ronald W. Beaudreau.

## **CHAPTER 153**

# **Neck and Upper Airway Disorders**

Aaron Barksdale

## **PHARYNGITIS AND TONSILLITIS**

### **Clinical Features**

Viral pharyngitis/tonsillitis may present with fever, odynophagia, and petechial or vesicular lesions on the soft palate and tonsils. These symptoms are often associated with cough, rhinorrhea, and congestion. Viral infections typically lack tonsillar exudates and cervical adenopathy except those associated with mononucleosis, influenza, and acute retroviral syndrome. Bacterial pharyngitis, particularly Group A  $\beta$ -Hemolytic Streptococcus Pharyngitis (GABHS), presents with acute onset of fever, sore throat, and odynophagia. Patients often display tonsillar erythema, exudates, and tender anterior cervical adenopathy. Cough, conjunctivitis, and rhinorrhea are typically lacking.

### **Diagnosis and Differential**

The Centor criteria for GABHS are (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) absence of cough, and (4) fever. Multiple authorities recommend no antibiotic therapy for patients with 0 or 1 criteria. For patients with 2 or more criteria, a rapid antigen test is recommended, and treatment is based on the results on the rapid test. For patients with 3 or more criteria, some authorities recommend empiric treatment while others recommend rapid antigen testing. The need for throat culture to follow negative rapid tests should be individualized as the false negative rate is 5%

to 10%; increased Centor scores are associated with increased likelihood of a positive throat culture after a negative rapid antigen test.

## **Emergency Department Care and Disposition**

1. Nonbacterial causes are treated with supportive care: **antipyretics, analgesics, and IV fluids** if dehydrated.
2. GABHS: single dose of **benzathine penicillin G** 1.2 million units IM or **penicillin VK** 500 milligrams orally PO 3 to 4 times daily for 10 days. Penicillin-allergic: macrolide or clindamycin.
3. **Dexamethasone** 10 milligrams PO or IM may be considered in moderate to severe cases.

## **PERITONSILLAR ABSCESS**

### **Clinical Features**

The patients may appear ill and often complain of fever, sore throat, odynophagia, trismus, dysphagia, and potentially a muffled voice (hot potato voice). The infected tonsil is typically displaced medially, causing deflection of the uvula to the opposite side.

### **Diagnosis and Differential**

Additional conditions to consider include peritonsillar cellulitis, infectious mononucleosis, retropharyngeal abscess, neoplasm, and internal carotid artery aneurysm. Diagnosis is typically made through the history and physical, but needle aspiration, CT, or US may be required for confirmation. Needle aspiration has the advantage of simultaneously confirming the diagnosis and treating the condition.

## **Emergency Department Care and Disposition**



1. **Needle aspiration** (18- or 20-gauge needle) or **I&D** after local anesthesia.
2. After adequate aspiration patients able to tolerate PO, may be discharged home on antibiotics. **Penicillin VK** (500 milligrams PO 4 time daily) or **clindamycin** (300 to 450 milligrams PO 3 to 4 times daily) for 10 days.
3. Otolaryngology should be consulted in cases that the emergency physician feels uncomfortable in managing themselves.

## **ADULT EPIGLOTTITIS (SUPRAGLOTTITIS)**

### **Clinical Features**

Patients often present with a 1 to 2 day history of worsening dysphagia, odynophagia, and dyspnea (worse when supine). They classically position themselves in the upright position, leaning forward, and may display drooling and inspiratory stridor.

### **Diagnosis and Differential**

Most cases are caused by Strep, Staph, viruses, and fungi. Diagnosis is made through history and physical, lateral cervical soft tissue radiograph (“thumb print sign”), and/or fiberoptic laryngoscopy.

### **Emergency Department Care and Disposition**

1. **Suspected epiglottitis requires immediate otolaryngology consultation, and the emergency physician must be prepared to establish a surgical airway.**
2. Patients should remain in the upright position. Initial airway management consists of humidified oxygen, IV hydration, cardiac monitoring, and pulse oximetry. **Heliox** can be given as a temporizing measure.

3. **Ceftriaxone** 2 grams IV is the recommended first-line drug. Steroids (**methyl-prednisolone**, 125 milligrams IV) may reduce airway inflammation and edema.
4. **Endotracheal intubation** is preferably performed in the operating room.

## **RETROPHARYNGEAL ABSCESS**

### **Clinical Features**

Common symptoms include sore throat, fever, torticollis, and dysphagia. Additionally, patients may complain of neck pain, muffled voice, cervical adenopathy, and respiratory distress. Stridor is more common in children.

### **Diagnosis and Differential**

Intravenous contrast-enhanced CT of the neck is the gold standard and differentiates cellulitis from an abscess.

### **Emergency Department Care and Disposition**

1. **Immediate otolaryngologic consultation** and **airway management** as required.
2. In adults, **clindamycin** 600 to 900 milligrams IV or **ampicillin/sulbactam** 3 grams IV.

## **ANGIOEDEMA OF THE UPPER AIRWAY**

### **Clinical Features**

Angioedema presents as nonpitting, nonpruritic swelling of the subcutaneous and deep dermal (mucosa) layers of the skin. May involve the

face, lips, tongue, and larynx, and can progress rapidly.

## **Diagnosis and Differential**

The diagnosis is made clinically. The common causes include IgE mediated type I hypersensitivity reaction, C1-esterase inhibitor deficiency (hereditary or acquired), angiotensin-converting enzyme inhibitor (ACE-I) induced, and idiopathic. Fiberoptic laryngoscopy may help to better identify the extent of laryngeal edema.

## **Emergency Department Care and Disposition**

1. Airway management and otolaryngology consultation, as indicated, by the severity of respiratory distress.
2. **Epinephrine** 1:1000 solution, 0.01 milligram/kilogram (maximum 0.3 milligram) administered IM, repeated every 5 min as needed. Additional medications that should be administered include **diphenhydramine** 50 milligrams IV, **ranitidine** 50 milligrams IV, **methylprednisolone** 125 milligrams IV. Patients with C1-esterase inhibitor deficiency and ACE-I induced angioedema classically do not respond to the above medications.
3. Discontinue any potential instigating medications (ACE-I). Patients with complete resolution of symptoms hours may be discharged after 3-4 hours of observation.
4. There are several new drugs currently undergoing trials for treatment of hereditary angioedema (HAE) and ACE-I induced angioedema. Currently approved medications for treatment of acute HAE in the United States are Berinert (human C1-esterase inhibitor) 20 units per kilogram, approximately three vials in the average person, and Ecallantide 30 milligrams subcutaneously in three 10 milligram injections.

## **LARYNGEAL TRAUMA**

Laryngeal injuries may present with hoarseness, dyspnea, stridor, dysphagia, hemoptysis, and aphonia. Physical examination may display anterior neck tenderness, tracheal displacement, or subcutaneous emphysema.

## **Diagnosis and Differential**

Consider fiberoptic nasopharyngolaryngoscopy, with the patient in upright position, to assess integrity of airway in those cases where airway compromise is suspected. If the patient is able tolerate supine position, acquire a CT of the neck; however, the patient with the potential to obstruct should not be left alone in the CT scanner.

## **Emergency Department Care and Disposition**

1. If laryngeal airway intact and endotracheal intubation indicated, perform directly or awake using fiberoptic bronchoscope. Caution in using rapid sequence intubation.
2. Emergency physician should be prepared to perform tracheostomy. Avoid cricothyrotomy if evidence of laryngeal injury.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 241, “Infections and Disorders of the Neck and Upper Airway,” by Rupali N. Shah, Trinitia Y. Cannon, and Carol G. Shores.

## **SECTION 17 Disorders of the Skin**

# CHAPTER 154

## Dermatologic Emergencies

Daniel A. Handel

### ERYTHEMA MULTIFORME AND STEVENS JOHNSON SYNDROME

Erythema multiforme (EM) strikes all ages, with the highest incidence in young adults (20 to 40 years), affects males twice as often as females, and occurs more commonly in the spring and fall.

#### Clinical Features

EM is an acute inflammatory skin disease with presentations that range from a mild papular eruption (EM minor) to diffuse vesiculobullous lesions with mucous membrane involvement and systemic toxicity (EM major or Stevens-Johnson syndrome). Precipitating factors include infection (mycoplasma and herpes simplex), drugs (antibiotics and anticonvulsants), and malignancy. No cause is found in about 50% of cases.

Malaise, arthralgias, myalgias, fever, a generalized burning sensation, and diffuse pruritus may precede skin lesions. The initial skin lesions are erythematous papules and maculopapules followed by target lesions in 24 to 48 hours. Urticarial plaques, vesicles, bullae, vesiculobullous lesions, and mucosal (oral, conjunctival, respiratory, and genitourinary) erosions may also develop ([Fig. 154-1](#)). Significant systemic toxicity along with significant fluid and electrolyte deficiencies and secondary infection may be seen in severe disease.



**FIGURE 154-1.** Erythema multiforme.

## **Diagnosis and Differential**

Target lesions are highly suggestive of EM. The presence of mucosal involvement suggests EM major/Stevens-Johnson syndrome. The differential diagnosis includes herpetic infections, vasculitis, toxic epidermal necrolysis, primary blistering disorders, Kawasaki disease, and toxic infectious erythemas.

## **Emergency Department Care and Disposition**

1. Patients without systemic manifestation and mucous membrane involvement may be managed as outpatients with dermatologic consultation. **Systemic steroid bursts (prednisone 60 to 80 milligrams**

- PO daily) are prescribed for mild disease but are unproven to change duration and outcomes. Acyclovir may reduce recurrent HSV-related EM.
2. Patients with extensive disease or systemic toxicity require critical care admission and consultation with a dermatologist and ophthalmologist. Intensive management of fluid, electrolyte, infectious, nutritional, and thermoregulatory issues is required. **Diphenhydramine** and **lidocaine rinses** provide symptomatic relief for stomatitis. Cool **Burrow solution** (5% aluminum acetate) compresses are applied to blistered regions.

## **TOXIC EPIDERMAL NECROLYSIS**

Toxic epidermal necrolysis (TEN) is a severe inflammatory skin disease that strikes all ages and both sexes equally. Some authorities consider TEN to be a variant of EM major/Stevens-Johnson syndrome.

### **Clinical Features**

Potential etiologies include medications, chemicals, infections, and immunologic factors. Malaise, anorexia, myalgias, arthralgias, fever, and upper respiratory infection symptoms may precede skin findings by 1 to 2 weeks. Skin findings progress from erythroderma to flaccid bullae to erosions with exfoliation ([Fig. 154-2](#)). A Nikolsky sign is present (slippage of the epidermis from the dermis when slight tangential pressure or rubbing is applied). Mucosal lesions are present. Systemic toxicity is common. Acute and chronic complications are similar to those encountered in EM major patients. TEN is fatal in 25% to 35% of cases. Predictors of poor prognosis include advanced age, extensive disease, multiple medication use, leucopenia, azotemia, and thrombocytopenia.





**FIGURE 154-2.** Toxic epidermal necrolysis.

## **Diagnosis and Differential**

Initial diagnosis is based on the clinical features. Definitive diagnosis is made via skin biopsy. The differential diagnosis includes EM major/ Stevens-Johnson syndrome, exfoliative erythroderma, primary blistering disorders, Kawasaki disease, and toxic infectious erythemas.

## **Emergency Department Care and Disposition**

1. TEN requires admission to a critical care setting or burn unit with appropriate cardiopulmonary, fluid, electrolyte, and sepsis resuscitation.
2. Emergent dermatologic consultation is required.

## **EXFOLIATIVE ERYTHRODERMA**

### **Clinical Features**

Exfoliative erythroderma is a cutaneous reaction to a drug, chemical, underlying systemic disease state, or skin disorder. Onset may be abrupt

(drug, chemical, underlying systemic disease, malignancy) or slow (underlying skin disorder). Symptoms include pruritus, tightening of the skin, and low-grade fever. Skin examination shows acute generalized warmth and erythema, flaking, scaling, and exfoliation. Patients with chronic disease have dystrophic nails, thinning of body hair, alopecia, and diffuse pigmentation changes.

## **Diagnosis and Differential**

Initial diagnosis is based on clinical features. Definitive diagnosis is made via skin biopsy. The differential diagnosis includes EM, TEN, and toxic infectious erythemas.

## **Emergency Department Care and Disposition**

1. Patients with significant disease require emergent dermatologic consultation, a search for the underlying precipitant and admission. Systemic steroids are frequently given after consultation.
2. Correct hypovolemia and hypothermia, if present.

## **TOXIC INFECTIOUS ERYTHEMAS**

Infectious syndromes caused by toxigenic bacteria with toxin-mediated dermatologic manifestations include staphylococcal toxic shock syndrome (TSS), streptococcal toxic shock syndrome (STSS), and staphylococcal scaled skin syndrome (SSSS). See [Chapter 88](#) for detailed discussion of TSS and STSS.

### **Clinical Features**

Both TSS and STSS present as multisystem illnesses with fever, shock, and erythroderma followed by desquamation. SSSS develops from a toxin

produced by *Staphylococcus aureus* and is seen primarily in infants and young children. The skin is initially tender, with diffuse erythroderma and may have a sandpaper texture. Nikolsky sign is present. Large flaccid bullae then appear followed by sloughing of skin, leaving normal skin in 7 to 10 days ([Fig. 154-3](#)).



**FIGURE 154-3.** Staphylococcal toxic shock syndrome in a young child.

### **Diagnosis and Differential**

For TSS and STSS, fever and hypotension with associated erythroderma should suggest the diagnosis. The differential diagnosis is broad and includes scarlet fever, Rocky Mountain spotted fever, leptospirosis, rubeola, meningococemia, SSSS, Kawasaki disease, TEN, Stevens-Johnson syndrome, gram-negative sepsis, and exfoliative drug eruptions. Infants and toddlers with fever and diffuse erythroderma suggest SSSS. The differential diagnosis for SSSS includes TEN, TSS, exfoliative drug eruptions, staphylococcal scarlet fever, and localized bullous impetigo.

## Emergency Department Care and Disposition

1. See [Chapter 88](#) for management of TSS and STSS.
2. Patients with diffuse SSSS require aggressive fluid resuscitation, treatment with parenteral antibiotics and admission. Antibiotic choices include **nafcillin** or **oxacillin** 2 grams IV every 6 hours (children: 100 milligrams/ kilogram/d IV divided every 6 hours). If methicillin resistance is suspected, **clindamycin** 600 milligrams IV every 6 hours (**children:** 40 milligrams/kilogram/d IV divided every 6 hours IV) or vancomycin 1 gram IV every 6 hours (in children: 10 to 15 milligrams/kilogram IV every 12 hours up to 1 gram) may be added.

## MENINGOCOCCEMIA

### Clinical Features

Meningococemia is a potentially fatal illness caused by *Neisseria meningitides* that typically affects persons younger than 20 years. Clinical disease usually develops within 3 to 4 days after exposure. Features include severe headache, fever, altered mental status, nausea, vomiting, myalgias, arthralgia, and neck stiffness. Dermatologic manifestations include petechia, urticaria, hemorrhagic vesicles, and macules that evolve into palpable purpura with grey necrotic centers ([Fig. 154-4](#)).



**FIGURE 154-4.** Meningococemia diffuse petechiae in a patient with meningococemia. (Photo contributed by Kevin J. Knoop, MD, MS. Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ: *Atlas of Emergency Medicine*, 3rd ed. 2010, © McGraw-Hill, New York.)

## **Diagnosis and Differential**

The diagnosis should be considered in ill-appearing patients with petechial rash and associated symptoms. The differential diagnosis includes Rocky Mountain spotted fever, TSS, gonococemia, bacterial endocarditis, vasculitis, viral and bacterial infections, and disseminated intravascular coagulation.

## **Emergency Department Care and Disposition**

1. Administer **ceftriaxone 2 gram IV** and **vancomycin 1 gram IV** as soon as the disease is suspected. The use of steroids is controversial.
2. All patients require close monitoring, supportive care, and hospitalization.

# PEMPHIGUS VULGARIS

## Clinical Features

Pemphigus vulgaris is a generalized, mucocutaneous, autoimmune, blistering eruption with a grave prognosis. Primary lesions are clear, tense vesicles or bullae that vary in diameter, and are first noted on the head, trunk, and mucous membranes. Within 2 to 3 days, the bullae become turbid and flaccid then rupture, producing painful, denuded areas that are slow to heal and prone to secondary infection ([Fig. 154-5](#)).



**FIGURE 154-5.** Scattered bullous lesions intermixed with erosions and painful inflammatory plaques in a patient with pemphigus vulgaris.

## Diagnosis and Differential

Pemphigus vulgaris is suspected by the appearance of lesions and confirmed by skin biopsy and immunofluorescence testing. The differential diagnosis includes bullous pemphigoid, TEN, EM major, dermatitis herpetiformis, and other blistering skin diseases. Bullous pemphigoid is a mucocutaneous blistering disease of the elderly.

## **Emergency Department Care and Disposition**

1. Treat fluid and electrolyte disturbances aggressively.
2. Consult with a dermatologist for management. Corticosteroids and immunosuppressives are the mainstays of therapy. Plasmaphoresis and IV immunoglobins may also be needed.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 245, “Serious Generalized Skin Disorders,” by J. Jeremy Thomas, Andrew D. Perron, and William J. Brady.

# CHAPTER 155

## Other Dermatologic Disorders

Daniel A. Handel

### ACNEIFORM ERUPTIONS

*Acne fulminans* is a severe form of cystic acne with ulcerating lesions associated with systemic symptoms such as fever, myalgias, arthralgias, and hepatosplenomegaly. *Pyoderma faciale* is an inflammatory cystic acneiform eruption on the central face of young women. Severe scarring can result without treatment. Dissecting cellulitis of the scalp and neck is an inflammatory scarring process seen mostly in young black males. *Acne keloidalis nuchae* is a perifollicular inflammatory process of the scalp. These diagnoses are made clinically.

Acute treatment of *acne fulminans* and *pyoderma faciale* includes systemic corticosteroids (**prednisone** 40 to 60 milligrams daily) and continuation of **isotretinoin** if already on it. Dissecting cellulitis of the scalp is treated with **5% to 10% benzoyl peroxide washes** and oral **doxycycline** or **minocycline**. *Acne keloidalis nuchae* can be treated with topical **clindamycin**, topical corticosteroids (**fluocinonide**), and oral doxycycline or minocycline. Refer to a dermatologist for further management, including consideration of initiation of isotretinoin.

### HERPES ZOSTER INFECTION

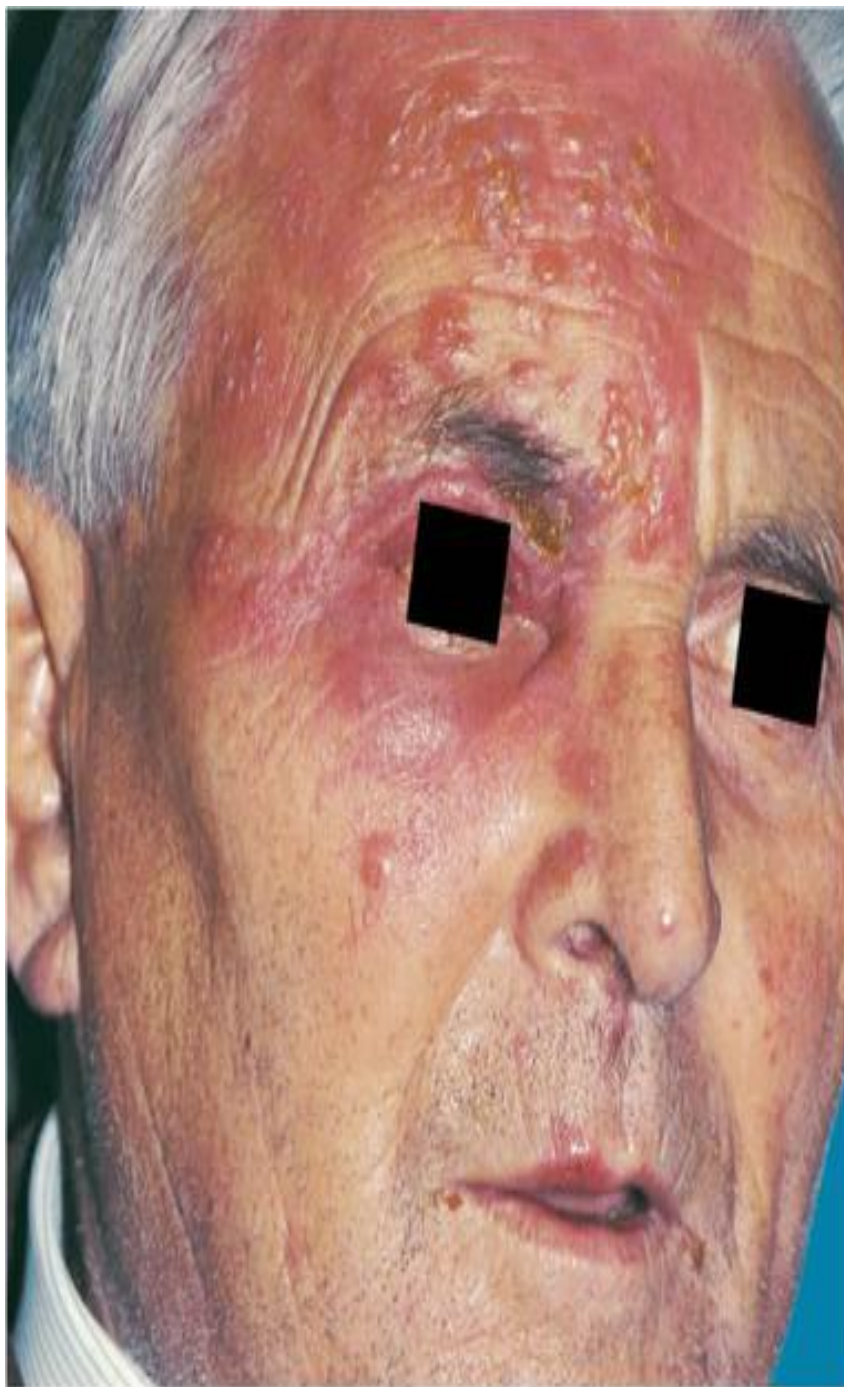
Herpes zoster results from activation of latent varicella zoster virus.

#### Clinical Features

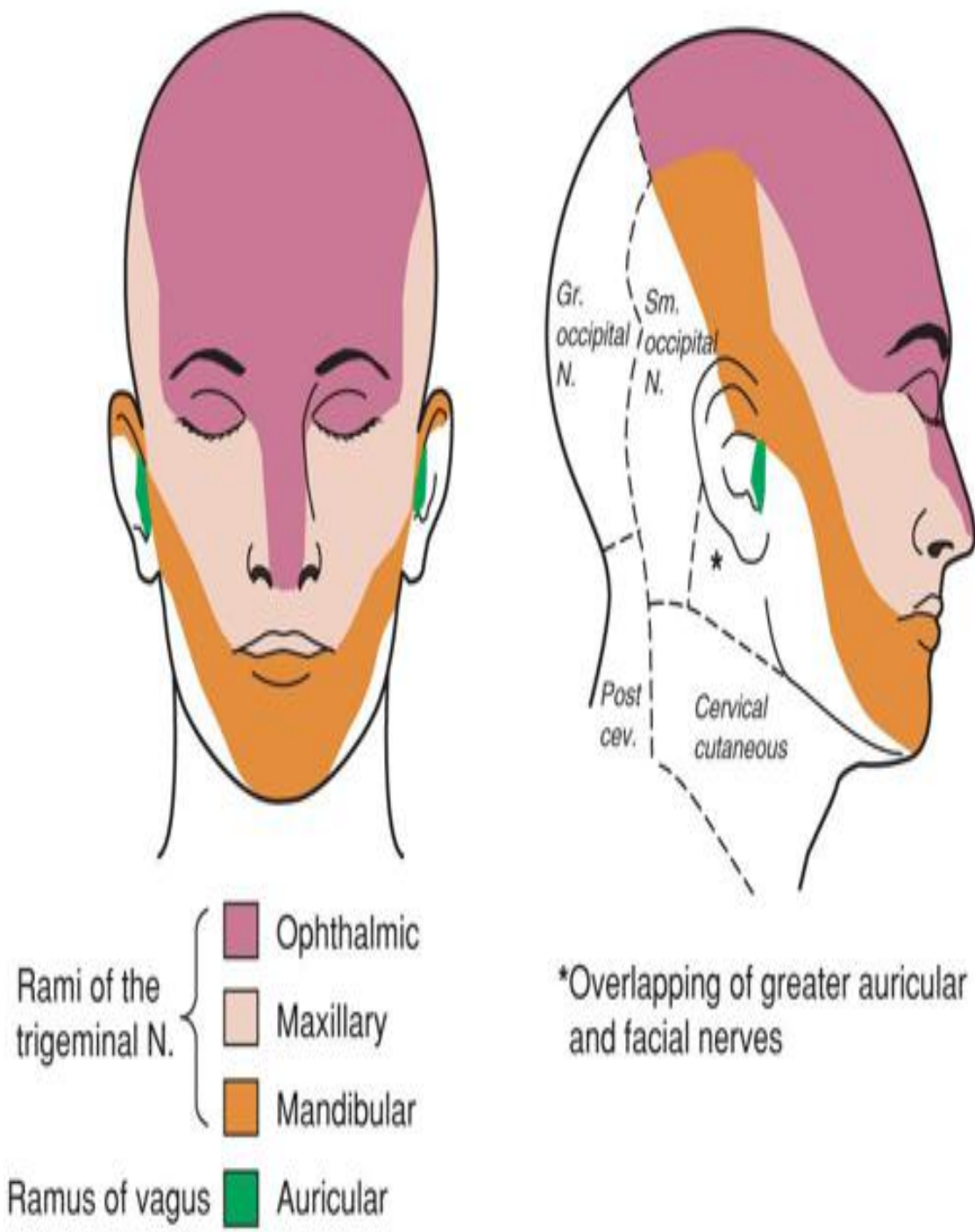
Pain or dysesthesia in the involved dermatome begins several days before lesions emerge. Erythematous papules develop first, progress to vesicular clusters, which crust after about a week. Lesions of the ophthalmic branch of the trigeminal nerve, especially if accompanied by lesions on the nose, are concerning for ophthalmic involvement (keratitis, ulceration) ([Fig. 155-1](#)). A thorough eye exam, including slit



lamp exam, should be performed (see [Chapter 149](#) “Ocular Emergencies”). Generalized eruptions may occur in immunocompromised patients.



**A**



**B**

**FIGURE 155-1.** A. Herpes zoster in trigeminal nerve distribution. Note lesion on the tip of the nose, which suggests nasociliary branch involvement. **B.** Dermatomes of the head and neck. cev. = cervical; Gr. = greater; N. = nerve; Sm. = smaller. (A

reproduced with permission from Fleischer A Jr, Feldman S, McConnell C, et al: *Emergency Dermatology: A. Rapid Treatment Guide*. New York, McGraw-Hill, 2002, p. 157. B. Reproduced with permission from Wolff K, Johnson R, Suurmond R: *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology, 5th ed*. New York, McGraw-Hill, 2005.)

## **Diagnosis and Differential**

The differential diagnosis includes herpes simplex, impetigo, and contact dermatitis. The key to diagnosis in patients is pronounced pain at the site and a unilateral distribution. A Tzanck prep and viral PCR can confirm the clinical diagnosis.

## **Emergency Department Care and Disposition**

1. Antivirals started in the first 72 hours of presentation shortens healing time, decreases formation of new lesions, and helps prevent posttherapeutic neuralgia. Antiviral choices include **acyclovir** 800 milligrams PO 5 times per day for 7 to 10 days or **valacyclovir** 1000 milligrams PO three times a day for 7 days. Patient with HIV/AIDS should take **acyclovir** for 21 days.
2. Aluminum acetate compressions three times daily and analgesics provide symptomatic relief.
3. Advise patients that herpes zoster is contagious to anyone who has not had chicken pox or the varicella zoster vaccine.
4. Consult with an ophthalmologist if eye involvement is suspected.

## **HERPES SIMPLEX VIRUS INFECTIONS**

Herpes simplex virus (HSV) lesions are painful grouped vesicles with an erythematous base. Primary disease may be preceded with or accompanied by constitutional symptoms. Tingling or burning precedes recurrent lesions. Oral lesions (“cold sores”) are usually caused by HSV1, but may be caused by HSV2. The diagnosis can be confirmed with a Tzanck preparation and HSV PCR test if necessary. Treatment ([Table 155-1](#)) is best if started within 24 hours of symptom onset. See [Chapter 87](#) “Sexually Transmitted Diseases” for discussion and treatment of genital herpes.

Condition	Acyclovir Treatment
First episode	
Adults, adolescents	400 milligrams orally thrice a day* for 7 days
Children	200 milligrams orally 5 times a day* for 7 days Maximum dose, 80 milligrams/kilogram/d
Recurrent episode	800 milligrams orally twice a day* for 5 days <i>or</i> Topical acyclovir cream or ointment, every 3 h for 7 days, applied as a 0.5-inch ribbon per 4 square inch of surface area

\*Frequency of daily dosing and amount of drug per dose can vary from 3 to 5 times per day, depending on patient convenience; see drug references for details.

**TABLE 155-1** Treatment of Herpes Simplex Virus Gingivostomatitis (Herpes Labialis)

## TINEA INFECTIONS

Tinea refers to skin infections caused by dermatophytes (fungi).

### Clinical Features

*Tinea capitis* is characterized by patchy areas of alopecia with broken off hairs and scales at the periphery. *Tinea barbae* presents with severe inflammatory plaques and follicular pustules in the beard area. Interdigital scaling, maceration, plantar or palmar erythema or scaling, and pruritis are seen in *Tinea pedis* (feet, also known as *athlete's foot*) and *Tinea manuum* (hand). Onchomycosis may occur. Findings in *Tinea cruris* (groin, commonly called *jock itch*) include erythema with a peripheral annular scaly edge that extends onto the thighs and buttocks but spares the penis and scrotum. *Candida intertrigo* involves the skinfolds. *Tinea Corporis* (trunk, neck, arms, and legs lesions) are typically circular, covered with scales and surrounded by a raised border.

### Diagnosis and Differential

Identification of fungal elements on a potassium hydroxide preparation or with fungal culture may be required if the diagnosis is uncertain. The differential diagnosis includes psoriasis, atopic, seborrheic, and chronic dermatitis ([Table 155-2](#)).

Condition	Distinguishing Clinical Features	Location	Special Signs
Psoriasis	Erythematous, well-margined papules and plaques with silver scale	Trunk, extensor surfaces, scalp	Auspitz sign; Koebner phenomenon, nail pitting
Seborrheic dermatitis	Greasy, yellow scales	Mid-chest, suprapubic, facial creases	Can overlap with psoriasis, "sebopsoriasis"
Lichen planus	4 P's: purple, pruritic, polygonal patches	Any skin, mucous membranes, hair follicles	Wickham striae; Koebner phenomenon
Pityriasis rosea	Lines of skin tension, collarette of scale	Trunk, in Christmas tree pattern following skin lines	Herald patch 1 to 2 weeks before general eruption
Tinea corporis	Sharply demarcated, erythematous, scaly patches; central clearing; may coalesce into gyrate patterns	Trunk, legs, arm, neck	May need KOH/culture to diagnose; septate hyphae on KOH
Pityriasis (tinea) versicolor	Versicolored—red, salmon, light brown, dark brown, hypopigmented; well-demarcated scaly patches	Central upper chest and back	Spaghetti and meatballs on KOH; nonseptate pseudohyphae and budding yeast
Secondary syphilis	At 2 to 10 weeks, macular erythema on trunk, abdomen, inner extremities; followed by papular or papulosquamous lesions	Palms, soles, trunk	Darkfield or serology
Scabies	Pruritic papules and burrows with crusting	Finger webs, wrists, axillae, areolae, umbilicus, abdomen, waistband, genitals	Scrapings show mites

Key: KOH = potassium hydroxide.

**TABLE 155-2** Comparison Features of Common Papulosquamous Eruptions

## Emergency Department Care and Disposition

1. Treat *Tinea capitis* and *barbae* with oral **ultramicrosized griseofulvin** 500 milligrams PO daily (children: 20 to 25 milligrams/kilogram/d) for 6 to 8 weeks, **terbinafine** 250 milligrams PO daily (children > 4 years of age, 125 milligrams daily) for 6 to 8 weeks, or **itraconazole** 200 milligrams daily (children 3 to 4 milligrams/kilogram/d) for 2 to 4 weeks. Consider baseline liver function tests. Initiation of oral antifungal treatment in patients with altered hepatic function is not recommended. Advise patients to wash hair with **selenium sulfide 2.5% shampoo** or **ketoconazole 2% shampoo** three times per week for 2 weeks.

2. Treat nonbullous *tinea pedis* and *manuum*, *intertrigo*, *tinea corpora*, *tinea cruris*, with topical antifungal agents such as **clotrimazole**, **miconazole**, **ketoconazole**, or **ciclopirox** twice daily for 2 to 4 weeks. Continue treatment 1 week after clearing has occurred. Antifungal powders used on a daily basis help prevent recurrences of *tinea cruris*.
3. Onchomycosis usually requires long term oral treatment.
4. Follow-up with a primary care physician or dermatologist is recommended, especially if the lesions are not resolved in 4 to 6 weeks.

## SCABIES AND LICE

See [Chapter 120](#) Bites and Stings for discussion of diagnosis and treatment of scabies and lice.

## CONTACT DERMATITIS

### Clinical Features

Contact dermatitis occurs after direct contact with an irritant or allergen. Reactions occasionally occur after exposure to aerosolized particles, such as burned poison ivy or oak. Detergents and soaps are common irritants; nickel, plants, cosmetic preservatives, contact lens solutions, and skin tape are common allergens. Physical findings include erythema, papules, vesicles and bullae. Scaling and fissuring are seen with chronic contact dermatitis.

### Diagnosis and Differential

The differential diagnosis includes dyshidrotic eczema, atopic dermatitis, and fungal infections.

### Emergency Department Care and Disposition

1. Treatment begins with removing the offending agent.
2. **Aluminum acetate or normal saline compresses two or three times** per day help ease acute irritation.

3. Oral antihistamines, such as **hydroxyzine** 25 to 50 milligrams PO 4 times a day, relieve pruritus.
4. Topical corticosteroids applied twice or thrice daily will reduce inflammation. The potency of the topical corticosteroid used is dependent on the severity of the reaction. Only low potency topical corticosteroids, such as **hydrocortisone 2.5%**, should be used on the face.
5. Patients with extensive severe allergic contact dermatitis may require a burst of systemic corticosteroids followed by a taper.

## PHOTOSENSITIVITY

Patients with sunburn have an inflammatory response to ultraviolet (UV) radiation and may present with minimal discomfort or extreme pain with extensive blistering. A tender, warm erythema is seen in sun-exposed areas; vesiculation may occur, representing a second-degree burn. *Exogenous photosensitivity* results from the topical application or ingestion of an agent that increases the skin's sensitivity when exposed to UV light. Topically applied furocoumarins (lime juice, various fragrances, figs, celery, parsnips), PABA esters (sunscreen), and topical psoralens can cause photosensitivity at the site of application. Numerous medications, including sulfonamides, thiazides, furosemide, fluoroquinolones, tetracyclines, cause photosensitivity eruptions which involves all sun-exposed areas. The exogenous photoeruption looks similar to a severe sunburn reaction, often with blistering.

Sunburn should be suspected in a patient with significant outdoor UV light exposure. The diagnosis of exogenous photosensitivity is based on identifying the offending agent. A linear rash appearance suggests an externally applied substance. Sunburns are treated symptomatically with tepid baths, NSAIDs, and wound care including topical antibiotics to blistered areas. Initial management of exogenous photosensitivity is similar to the sunburn reaction, including the avoidance of the sun until the eruption has cleared. Discontinue the causative agent, if possible.

## PSORIASIS

Psoriasis is a chronic skin disorder characterized by symmetric discrete erythematous plaques with a silver scale typically located on the extensor surfaces (knees, elbows), scalp and chest.  $\beta$ -blockers, lithium, and antimalarials may exacerbate the condition. Guttate psoriasis presents more acutely with scattered discrete lesions sometimes following an infection such as streptococcal pharyngitis. Pustular psoriasis presents with scaling and numerous painful pustules on the palms and soles. The differential

diagnosis of psoriasis can be found in [Table 155-2](#). The diagnosis of psoriasis is usually made clinically but a biopsy may be required. Treatment is initiated in consultation with a dermatologist. Topical treatments for localized plaques include moisturizers, topical corticosteroids, tar and vitamin D preparations. Antibiotics are indicated for streptococcal infections. Patients with diffuse pustular psoriasis often require admission for hydration and supportive care.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 246, “Disorders of the Face and Scalp,” by Dean S. Morrell and Emily J. Schwartz; Chapter 247, “Disorders of the Hands, Feet, and Extremities,” by Craig N. Burkhart and Dean S. Morrell; Chapter 248, “Disorders of the Groin and Skinfolds,” by Dean S. Morrell and Kelly Nelson; and Chapter 249, “Skin Disorders Common on the Trunk,” by Mark R. Hess and Suzanne P. Hess.



## **SECTION 18 Trauma**

# **CHAPTER 156**

## **Trauma in Adults**

Jonathan S. Ilgen

Trauma care should be guided by the concepts of rapid assessment, triage, resuscitation, serial reassessment, diagnosis, and therapeutic intervention.

### **CLINICAL FEATURES**

Trauma patients can sustain a multitude of injuries. Many will present with abnormal vital signs, neurologic deficits, or other gross evidence of injury. These signs must prompt both a thorough search for the specific underlying injuries and rapid interventions to correct the abnormalities. Nonspecific signs such as tachycardia, tachypnea, or mild alterations in consciousness must similarly be presumed to signify serious injury until proven otherwise. Further, without signs of significant trauma, the mechanism of injury may suggest potential problems, and these also should be pursued diligently.

### **DIAGNOSIS AND DIFFERENTIAL**

The assessment of trauma patients begins with a focused history from the patient, family members, witnesses, or prehospital providers. Patterns of injuries, and expected physiologic responses to these injuries, can be ascertained by collecting history regarding the circumstances of the event (eg, single vehicle crash, fall from height, smoke inhalation, or environmental exposures), ingestion of intoxicants, preexisting medical conditions, and medications.

To facilitate an organized approach to the trauma patient, the examination is divided into primary and secondary surveys ([Table 156-1](#)). The goal of the primary survey is to identify and immediately treat life-threatening conditions. To do so, the acronym ABCDE encourages the clinician to

examine the patient's airway, breathing, circulation, and disability (mental status, Glasgow Coma Scale (GCS), and neurologic examination), and to completely expose each patient so that occult injuries or exposures are visualized. After this initial primary survey, perform a thorough head-to-toe examination (the secondary survey, [Table 156-1](#)), then proceed with appropriate diagnostic testing and further therapeutic interventions.

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## **Primary Survey (Rapid identification and management of immediately life-threatening injuries)**

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### A. Airway and cervical spine

Assess, clear, and protect airway: jaw thrust/chin lift, suctioning.

Perform endotracheal intubation with in-line stabilization for patient with depressed level of consciousness or inability to protect airway.

Create surgical airway if there is significant bleeding or obstruction, or laryngoscopy cannot be performed.

---

### B. Breathing

Ventilate with 100% oxygen, monitor oxygen saturation.

Auscultate for breath sounds.

Inspect thorax and neck for deviated trachea, open chest wounds, abnormal chest wall motion, crepitus at neck or chest.

Consider immediate needle thoracostomy for suspected tension pneumothorax.

Consider tube thoracostomy for suspected hemopneumothorax.

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### C. Circulation

Assess for blood volume status: skin color, capillary refill, radial/femoral/carotid pulse, blood pressure.

Place 2 large-bore peripheral IV catheters.

Begin rapid infusion of warm crystalloid solution, if indicated.

Apply direct pressure to sites of brisk external bleeding.

Consider central venous access if peripheral sites are unavailable.

Consider pericardiocentesis for suspected pericardial tamponade.

Consider left lateral decubitus position in late-trimester pregnancy.

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#### D. Disability

Perform screening neurologic and mental status examination, assessing:

Pupil size and reactivity

Limb strength and movement, grip strength

Orientation, Glasgow Coma Scale score

Consider measurement of capillary blood glucose level in patients with altered mental status.

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#### E. Exposure

Completely disrobe the patient, inspect for burns, toxic exposures.

Log-roll patient, maintaining neutral position and in-line neck stabilization, to inspect and palpate thoracic spine, flank, back, and buttocks.

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### **Secondary Survey (Head-to-toe examination for rapid identification and control of injuries or potential instability)**

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Identify and control scalp wound bleeding with direct pressure, sutures, or surgical clips.

Identify facial instability, potential for airway instability.

Identify hemotympanum.

Identify epistaxis or septal hematoma; consider tamponade or airway control if bleeding is profuse.

Identify avulsed teeth, jaw instability.

Evaluate for abdominal distention and tenderness.

Identify penetrating chest, back, flank, or abdominal injuries.

Assess pelvic stability, consider pelvic wrap or sling.

Inspect perineum for laceration or hematoma.

Inspect urethral meatus for blood.

Consider rectal examination for sphincter tone and gross blood.

Assess peripheral pulses for vascular compromise.

Identify extremity deformities and immobilize open and closed fractures and dislocations.

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**TABLE 156-1** Primary and Secondary Survey in Trauma Resuscitation

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. The ED management of trauma patients begins prior to the patient's arrival. EMS providers inform the receiving ED about the mechanism of trauma, vital sign values, suspected injuries, and treatments provided.
2. Airway patency is confirmed at the outset of the primary survey. In patients making inadequate respiratory efforts, perform a jaw thrust, then insert an oral or nasal airway. Avoid nasal airway placement in patients with suspected basilar skull fractures. Endotracheal intubation is indicated in comatose patients (GCS <8) to protect the airway and prevent secondary brain injury from hypoxemia. Agitated trauma patients who need further diagnostic or therapeutic interventions or patients whose expected course necessitates immediate operative management are also candidates for intubation. Whenever possible, use a two-person spinal stabilization technique, in which one caregiver provides in-line immobilization of the cervical spine while the other manages the airway. Trauma patients are often difficult to intubate due to associated facial trauma, cervical spine immobilization, or the presence of blood or vomitus. In virtually all trauma patients requiring urgent intubation, a rapid sequence intubation technique should be used. In cases of extensive facial trauma or when endotracheal intubation is not possible, cricothyrotomy or another advanced airway technique may be used to secure the airway.
3. Once the airway is secured, examine the neck and thorax to detect abnormalities such as a deviated trachea (tension pneumothorax), crepitus (pneumothorax), paradoxical movement of a chest wall segment (flail chest), sucking chest wound, fractured sternum, or the absence of breath sounds on either side of the chest (simple or tension pneumothorax, massive hemothorax, or right mainstem intubation). Treat tension pneumothorax immediately with needle decompression followed by tube thoracostomy.

4. Rapidly assess the patient's hemodynamic status during the primary survey by noting the level of consciousness, skin color, and the presence and magnitude of peripheral pulses. Note the heart rate, blood pressure, and pulse pressure (systolic minus diastolic blood pressure). Hemorrhage of up to 30% of total blood volume may be associated with only mild tachycardia and a decrease in pulse pressure, but may quickly progress to shock if not recognized early. Establish 2 large-bore peripheral IV lines, and obtain blood samples for laboratory studies, particularly the blood type and screen. Establish a central line in patients who are unstable or in whom upper extremity peripheral veins are not easily cannulated.
5. Reassess hemodynamically unstable patients without an obvious indication for surgery after infusion of 2 L of warm crystalloid solution. If there is no marked improvement, consider the need to transfuse type O blood (O-negative for females of childbearing age). A focused assessment with sonography for trauma (FAST) examination screens for causes of shock immediately after the primary survey. If a patient is hemodynamically stable, definitive imaging can be performed with a CT scan of the abdomen and pelvis with IV contrast. In patients with penetrating abdominal trauma who are in shock, early operative intervention results in better outcomes.
6. Major trauma patients may develop a bleeding diathesis, which results in defective clotting and platelet function. If patients require >10 units of packed red blood cells (PRBC), patients should receive PRBC in a 1:1 ratio with fresh frozen plasma. Both acidosis and hypothermia contribute to the coagulopathy and should be corrected as soon as possible.
7. After the primary survey and stabilization, perform an abbreviated neurologic examination, including an assessment of the patient's level of consciousness, GCS, pupillary size and reactivity, and motor function. A search for the cause of depressed level of consciousness includes the measurement of capillary blood glucose levels and the consideration of possible intoxicants, though one should begin with the assumption this is due to significant traumatic brain injury (TBI). In order to quickly identify potentially operative intracranial injuries among patients with suspected traumatic brain injury and coma (GCS of 3 to 8), defer any procedures that do not correct a specific problem during the primary survey until after the CT scan of the head is performed. Intubated patients should undergo

continuous capnography. GCS assessment can be insensitive in patients with normal or near-normal scores, and a GCS score of 15 does not exclude the presence of TBI.

8. Once the patient is hemodynamically stable and the airway is secured, log-roll the patient with one team member assigned to maintain in-line cervical stabilization. Palpate the spinous processes of the thoracic and lumbar spine for tenderness or deformity. Consider performing a rectal examination to assess for gross blood, displaced prostate, or determining rectal tone in the setting of a suspected spinal injury.
9. Certain conditions, such as injuries to the esophagus, diaphragm, and small bowel, often remain undiagnosed even with diligent examination, and further imaging and hospital observation for delayed presentation may be required. The most frequently missed injuries are orthopedic.
10. Expeditiously transport patients with hemodynamic instability and ongoing bleeding to the operating room or transfer them to another facility with appropriate surgical or critical care resources. Serial examinations are essential in patients without obvious indications for surgery identified on the initial assessment. These examinations may be performed in the inpatient or, in some cases, the ED observation unit settings.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 250, "Trauma in Adults," by Patrick H. Brunett and Peter A. Cameron.



# CHAPTER 157

## Trauma in Children

Matthew Hansen

Trauma is the most common cause of death in children older than 1 year. Differences in anatomy and physiology mandate modifications to trauma evaluation and management in children.

### CLINICAL FEATURES

Head trauma is the most frequent pediatric injury resulting in death. Overall, motor vehicle crash is the most common mechanism, and it is the leading mechanism of traumatic death in children older than 1 year. There are many manifestations and consequences of trauma unique to pediatric patients that should be addressed in the primary survey ([Table 157-1](#)).

Identify	Intervene
<b>Airway</b> Inadequate airway Cervical spine injury	Securing and protection of airway Stabilization of cervical spine
<b>Breathing</b> Apnea Hypoxia Tension pneumothorax Massive hemothorax Open pneumothorax	Positive pressure ventilation Supplemental oxygen administration Needle decompression, tube thoracostomy Tube thoracostomy Occlusive dressing, tube thoracostomy
<b>Circulation</b> Hypovolemic shock Pericardial tamponade Cardiac arrest	Fluid bolus, blood products Fluid bolus, pericardiocentesis, thoracotomy Chest compressions (CPR) ED thoracotomy if penetrating trauma
<b>Disability</b> Spinal cord injury Cerebral herniation	Immobilization, steroids Mild hyperventilation, mannitol
<b>Exposure</b> Hypothermia Exsanguinating hemorrhage	Warmed fluids, external warming Direct pressure, air splints

## TABLE 157-1 Primary Survey Goals

**Airway:** Airway management in children can be challenging. Anatomic differences include a large occiput, large tongue, and cephalad location of the larynx.

**Breathing:** Infants younger than 6 months are nose breathers and facial trauma may cause respiratory distress. Tachypnea is often the first sign of dyspnea ([Table 157-2](#)).

Signs of hypoxemia
Cyanosis
Agitation
Poor capillary refill
Bradycardia
Desaturation measured by pulse oximetry
Signs of inadequate ventilation
Tachypnea
Nasal flaring
Grunting
Retractions
Stridor or wheezing

## TABLE 157-2 Signs of Hypoxemia and Inadequate Ventilation

**Circulation:** Children with compensated shock from hemorrhage have normal blood pressure and tachycardia. Other signs of shock include cap refill > 3 seconds, cool extremities, weak peripheral pulses, and altered mental status. Hypotension is a prearrest finding in children.

**Disability:** In younger children, an age-specific adaptation of the Glasgow Coma Scale should be used. A bulging anterior fontanelle may indicate elevated intracranial pressure.

**Exposure:** The ratio of surface area to mass is greater in children, putting them at greater risk for hypothermia. Care should be taken to maintain normothermia.

## DIAGNOSIS AND DIFFERENTIAL

### Head Injury

Infants and neonates are at the highest risk of significant intracranial injury ([Table 157-3](#)). Mental status may be difficult to assess due to frequent developmental changes and patient anxiety. Parietal and occipital skull fractures are frequently associated with intracranial bleeding. Noncontrast CT is the imaging modality of choice for intracranial injury in children. Scalp injuries, particularly in neonates, may result in significant blood loss and shock.

	Negligible Risk	Low Risk <sup>†</sup>	Moderate to High Risk
Findings	Normal examination findings No LOC No symptoms	Normal examination findings LOC <1 min Amnesia Headache Vomiting Lethargy	Altered mental status LOC >1 min Persistent vomiting Seizures Facial injury Multiple trauma Abnormal examination findings
Imaging	None	CT scan	CT scan
Disposition	Discharge	Observation	Referral as indicated

Key: LOC = loss of consciousness.

\*Defined as applicable for patients aged 2–20 y, previously healthy, with isolated head injury.

<sup>†</sup>Risk of positive CT findings of up to 7%.

Based on information at AAP Policy Web site, American Academy of Pediatrics. Available at: <http://aappolicy.aappublications.org>. Accessed October 10, 2008.

**TABLE 157-3 Risk Groups for Pediatric Head Injury\***

## Spine Injuries

The increased flexibility of the spine in preadolescent children is responsible for the relatively lower incidence of spinal fracture in this group; thus, young children with spinal cord injuries often do not have associated fractures. They may present with minimal symptoms followed by delayed progression of disability. “Clearing the cervical spine,” in children is challenging as there is little evidence to guide practice. Multisystem trauma or head trauma are general indications for neck immobilization and cervical spine imaging. Due to the low incidence of

spine fractures in younger children and the need to lower ionizing radiation, plain films of the cervical spine remain a useful tool ([Table 157-4](#)).

Moderate- or high-risk head injury
Multiple trauma
Signs or symptoms of spinal injury
Direct mechanism for spinal injury
Altered mental status or focal neurologic findings
Distracting painful injury
Agitation with possible mechanism for spinal injury

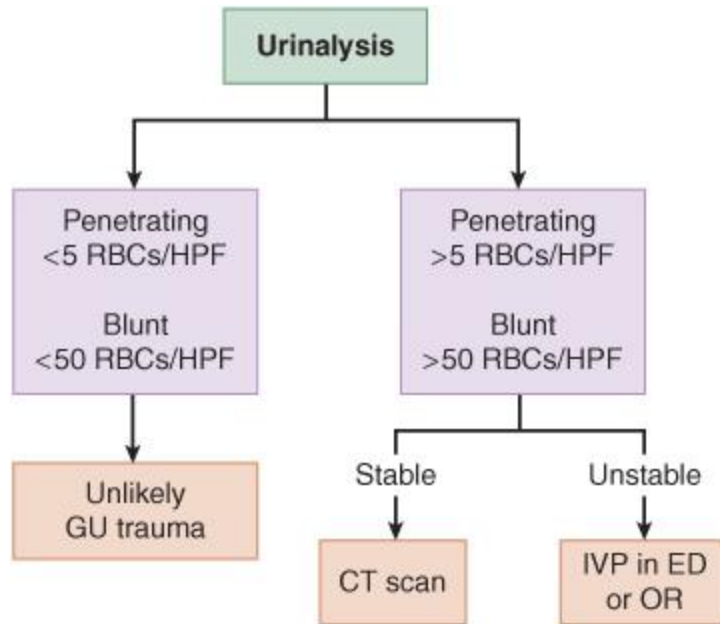
**TABLE 157-4** Considerations for Cervical Spine Imaging in Children

## Chest Trauma

The relatively compliant chest wall of the child means that serious injuries to intrathoracic structures can be present without significant external signs. Rib fractures are less common in children and generally require a significant mechanism of injury. The chest radiograph is an essential tool in the evaluation of any child with trauma to the torso.

## Abdominal and Genitourinary Trauma

The physical examination in children has been shown to be unreliable in determining the severity of injury in up to 45% of pediatric trauma patients. Indications for CT include: suspicious mechanism of injury, tenderness on exam, seatbelt sign, distention, vomiting, or more than 50 red blood cells per high-power field on urinalysis for blunt trauma. Identification of a pelvic fracture, particularly an anterior ring fracture, should prompt investigation for associated urethral or bladder injury ([Fig. 157-1](#)). The role of ultrasound for pediatric abdominal trauma, especially in stable patients, is not well established.



**FIGURE 157-1.** Algorithm for ED assessment of possible renal injury.

*Key:* HPF = highpower field; IVP = IV pyelography; OR = operating room; RBC = red blood cell.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Place all seriously injured patients on **100% oxygen**. Consider **elevating the torso** in smaller children. With difficult bagging consider oral airway placement and 2-person mask technique.
2. **Orotracheal intubation** is indicated for definitive airway management. The following formula is used to estimate the endotracheal tube size:  $\text{size} = 4 + (\text{age}/4)$ . Cuffed or uncuffed endotracheal tubes can be used; however, the appropriate cuffed tube size is one half size smaller than what is calculated using the formula above. Perform **rapid sequence intubation** using pretreatment with 100% oxygen, appropriate sedation, and pharmacologic paralysis for the patient with an unstable airway.
3. Vascular access can be challenging. Obtain **intraosseous access** early, as needed. If central venous access is needed, the femoral vein is the most

easily accessed site. Administer fluids in **20-mL/kg boluses of crystalloid**; if there is no response to 3 boluses, then administer **10-mL/kg boluses of packed red blood cells**.

4. For pain control, **fentanyl 1 microgram/killogram** or **morphine 0.05 to 0.1 milligram/killogram** are appropriate.
5. If a head injured patient has clinical signs of impending herniation, then transiently maintain the PaCO<sub>2</sub> at 30 to 35 mm Hg, optimize blood pressure with IV fluids, elevate the head of the bed 20° to 30° while keeping the head in a neutral position, and administer **mannitol 1 gram/killogram**.
6. Admit children with skull fractures, intracranial hemorrhage, spinal trauma, significant chest trauma, abdominal trauma with internal organ injury, significant burns, or other concerning injuries. Guidelines for referral to a pediatric trauma center are listed in [Table 157-5](#).

Mechanism of injury	Ejection from motor vehicle Fall from a height Motor vehicle collision with prolonged extrication Motor vehicle collision with death of another vehicle occupant
Anatomic injury	Multiple severe trauma More than three long-bone fractures Spinal fractures or spinal cord injury Amputations Severe head or facial trauma Penetrating head, chest, or abdominal trauma

Reproduced with permission from Harris BH, Barlow BA, Ballantine TV, et al: American Pediatric Surgical Association principles of pediatric trauma care. *J Pediatr Surg* 27: 423, 1992.

**TABLE 157-5** Indications for Transfer to a Pediatric Trauma Center

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 251, “Trauma in Children,” by William E. Hauda II.

# **CHAPTER 158**

## **Trauma in the Elderly**

O. John Ma

Emergency physicians need to stay abreast with many of the unique injury mechanisms and clinical features associated with geriatric trauma patients, and apply special management principles when caring for them.

### **CLINICAL FEATURES**

Falls are the most common cause of injury in patients over 65 years of age. Syncope, which has been implicated in many cases, may be secondary to dysrhythmias, venous pooling, autonomic derangement, hypoxia, anemia, or hypoglycemia. Motor vehicle crashes rank as the most common mechanism for fatal incidents in elderly persons through 80 years of age. Also, elderly pedestrians struck by a motor vehicle are much more likely to die compared to younger pedestrians. Emergency physicians should have a heightened suspicion for elder or parental abuse.

The geriatric trauma patient should be viewed as both a medical and a trauma patient. Since elderly patients may have a significant past medical history that impacts their trauma care, obtaining a precise history is vital. Often, the time frame for obtaining information about the traumatic event, past medical history, medications, and allergies is quite short. Family members, medical records, and the patient's primary physician may be helpful in gathering information regarding the traumatic event and the patient's previous level of function. Medications, such as cardiac agents, diuretics, psychotropic agents, and anticoagulants, must be carefully listed.

On physical examination, frequent monitoring of vital signs is essential. Emergency physicians should be wary of a "normal" heart rate in the geriatric trauma victim. A normal tachycardic response to pain, hypovolemia, or anxiety may be absent or blunted in the elderly trauma

patient. Medications such as  $\beta$ -blockers may mask tachycardia and delay appropriate resuscitation.

Special attention should be paid to anatomical variation that may make airway management more difficult. These include the presence of dentures, cervical arthritis, or temporomandibular joint arthritis. A thorough secondary survey is essential to uncover less serious injuries. These “minor” injuries may not be severe enough to cause problems during the initial resuscitation, but cumulatively may cause significant morbidity and mortality. Seemingly stable geriatric trauma patients can deteriorate rapidly and without warning.

## **DIAGNOSIS AND DIFFERENTIAL**

### **Head Injury**

It would be a grave error to assume that alterations in mental status are due solely to any underlying dementia or senility when evaluating the elderly patient’s mental status. Elderly persons suffer a much lower incidence of epidural hematomas than the general population; however, there is a higher incidence of subdural hematomas. The rate of intracranial hemorrhage approaches 7% to 14% in anticoagulated patients with blunt head injury who are experiencing no or minimal symptoms. More liberal indications for computed tomography (CT) scanning are justified.

### **Cervical Spine Injuries**

The pattern of cervical spine injuries in the elderly is different than in younger patients, as there is an increased incidence of C1 and C2 fractures with the elderly. Emergency physicians need to place special emphasis on maintaining cervical immobilization until the cervical spine is properly assessed. Because underlying cervical arthritis may obscure fracture lines, the elderly patient with persistent neck pain and negative plain radiographs should undergo CT of the neck.



## **Chest Trauma**

In blunt trauma, there is an increased incidence of rib fractures due to osteoporotic changes. The pain associated with rib fractures, along with any decreased physiologic reserve, may predispose patients to respiratory complications. More severe thoracic injuries, such as hemopneumothorax, pulmonary contusion, flail chest, and cardiac contusion, can quickly lead to decompensation in elderly individuals whose baseline oxygenation status may already be diminished. Arterial blood gas analysis may provide early insight into elderly patients' respiratory function and reserve.

## **Abdominal Trauma**

The abdominal examination in elderly patients is notoriously unreliable compared to younger patients. The adhesions associated with previous abdominal surgical procedures may increase the risk of performing diagnostic peritoneal lavage. The focused assessment with sonography for trauma (FAST) examination may assist in evaluating for hemoperitoneum and the need for exploratory laparotomy in hemodynamically unstable patients. CT with contrast is a valuable diagnostic test for patients who are stable. It is important to ensure adequate hydration and baseline assessment of renal function prior to the contrast load for the CT scan. Some patients may be volume depleted due to medications, such as diuretics. This hypovolemia coupled with contrast administration may exacerbate any underlying renal pathology.

## **Orthopedic Injuries**

Hip fractures occur primarily in 4 areas: intertrochanteric, transcervical, subcapital, and subtrochanteric. Intertrochanteric fractures are the most common, followed by transcervical fractures. Emergency physicians must be aware that pelvic and long bone fractures are not infrequently the sole etiology for hypovolemia in elderly patients. Timely orthopedic

consultation, evaluation, and treatment with open reduction and internal fixation should be coordinated with the diagnosis and management of other injuries.

Long bone fractures of the femur, tibia, and humerus may produce a loss of mobility with a resulting decrease in the independent lifestyle of elderly patients. Early orthopedic consultation for intramedullary rodding of these fractures may result in increased early mobilization.

The incidence of Colles fractures and humeral head and surgical neck fractures in elderly patients are increased by falls on the outstretched hand or elbow. Localized tenderness, swelling, and ecchymosis to the proximal humerus are characteristic of these injuries. Early orthopedic consultation and treatment with a shoulder immobilizer or surgical fixation should be arranged.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

As in all trauma patients, the primary survey should be assessed expeditiously.

1. The main therapeutic goal is maintaining adequate oxygen delivery. **Consider prompt tracheal intubation** and use of mechanical ventilation in patients with more severe injuries, respiratory rates greater than 40 breaths/min, or when the  $\text{PaO}_2$  is  $<60$  mm Hg or  $\text{PaCO}_2 >50$  mm Hg. While nonventilatory therapy helps to prevent respiratory infections and is always desirable, early mechanical ventilation may avert the disastrous results associated with hypoxia.
2. Geriatric trauma patients can decompensate with over-resuscitation just as quickly as they can with inadequate resuscitation. Elderly patients with underlying coronary artery disease and cerebrovascular disease are at a much greater risk of suffering the consequences of ischemia to vital organs when they become hypotensive after sustaining trauma. During the initial resuscitative phase, administer crystalloid judiciously since elderly patients with diminished cardiac compliance are more susceptible

to volume overload. Strong consideration should be made for early and more liberal use of **packed red blood cell transfusion**. This practice early in the resuscitation enhances oxygen delivery and helps minimize tissue ischemia.

3. **Early invasive monitoring** has been advocated to help physicians assess the elderly's hemodynamic status. One study found that urgent invasive monitoring provides important hemodynamic information early, aids in identifying occult shock, limits hypoperfusion, helps prevent multiple organ failure, and improves survival. Survival was improved because of enhanced oxygen delivery through the use of adequate volume loading and inotropic support.
4. If the insertion of invasive monitoring lines is impractical in the ED, every effort should be made by emergency physicians to expedite care of elderly trauma patients and prevent unnecessary delays. While it is vital to be thorough in the diagnosis of occult orthopedic injuries, expending a great deal of time in the radiology suite may compromise patient care. Only a few radiologic studies, such as emergent head and abdominal CT scans, should take precedence over obtaining vital information from invasive monitoring. Elderly trauma patients will benefit most from an expeditious transfer to the intensive care unit for invasive monitoring; in that setting, patients can be assessed for subtle hemodynamic changes that may compromise those with limited physiologic reserve.
5. Emergency physicians, in consultation with the trauma surgeon, should have a low threshold for having the geriatric trauma patient admitted for further evaluation and observation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 252, "Geriatric Trauma," by O. John Ma, Jennifer H. Edwards, and Stephen W. Meldon.

# **CHAPTER 159**

## **Trauma in Pregnancy**

Nicole M. DeIorio

Trauma is the leading cause of nonobstetric morbidity and mortality in pregnant women. Fetal survival is highly dependent on stabilization of the mother.

### **CLINICAL FEATURES**

Physiologic changes of pregnancy make determination of severity of injury problematic. Heart rate increases 10 to 20 beats/min in the second trimester while systolic and diastolic blood pressures drop 10 to 15 mm Hg. Blood volume can increase by 45%, but red cell mass increases to a lesser extent, leading to a physiologic anemia of pregnancy. It may be difficult to determine whether tachycardia, hypotension, or anemia is due to blood loss or normal physiologic changes. Due to the hypervolemic state, the patient may lose 30% to 35% of her blood volume before manifesting signs of shock. Pulmonary changes in pregnancy include elevation of the diaphragm and decreases in residual volume and function residual capacity. Tidal volume increases, resulting in hyperventilation with associated respiratory alkalosis. Renal compensation causes the serum pH to remain unchanged.

Anatomic changes with pregnancy affect the types of injuries seen in the mother. After week 12 of gestation, the enlarging uterus emerges from the pelvis and by 20 weeks reaches the level of the umbilicus. Uterine blood flow increases, making severe maternal hemorrhage from uterine trauma more likely. The uterus also can compress the inferior vena cava when the patient is supine, leading to the “supine hypotension syndrome.” As pregnancy progresses, the small intestines are pushed cephalad, increasing their likelihood of injury in penetrating trauma to the upper abdomen. Decreased intestinal motility is associated with gastroesophageal reflux, thus predisposing the patient to vomiting and aspiration. The bladder moves

into the abdomen in the third trimester, thereby increasing its susceptibility to injury. Splenic injury remains the most common cause of abdominal hemorrhage in the pregnant trauma patient.

Abdominal trauma affects the fetus and the mother. Fetal injuries are more likely to be seen in the third trimester, often associated with pelvic fractures or penetrating trauma. Uterine rupture is rare but is associated with a very high fetal mortality rate. More common complications of trauma include preterm labor and abruptio placentae. Second only to maternal death, abruptio placentae is a common cause of fetal death. Classically, the mother will demonstrate abdominal pain, vaginal bleeding, uterine contractions, and signs of disseminated intravascular coagulation. Fetal–maternal hemorrhage occurs in more than 30% of cases of significant trauma and may result in rhesus (Rh) isoimmunization of Rh-negative women.

## **DIAGNOSIS AND DIFFERENTIAL**

Because maternal stability and survival offer the best chance for fetal well-being, no critical interventions or diagnostic procedures are withheld out of concern for potential adverse effects to the fetus. The initial sequence of trauma resuscitation is unchanged. Direct special attention to the gravid abdomen, examining for evidence of injury, tenderness, or uterine contractions. If abdominal or pelvic trauma is suspected, perform a sterile pelvic examination to assess for genital trauma, vaginal bleeding, or ruptured amniotic membranes. Fluid with a pH of 7 in the vaginal canal suggests amniotic rupture, as does a branch-like pattern, or “ferning,” on drying of vaginal fluid on a microscope slide.

Initial laboratory studies include a complete blood count, blood type, Rh status, and coagulation studies including fibrin split products and fibrinogen to determine the presence of disseminated intravascular coagulation. Order radiographs based on fundamental principles of trauma management. However, shield the uterus when possible and limit radiographs to those that will significantly affect the patient’s care. Adverse fetal effects from radiation are negligible from doses lower than 5 rad, which is an exposure

far greater than that received from most plain radiographs. Radiation exposure from CT may be decreased by reducing the number of imaging cuts. Bedside ultrasonography is a highly sensitive, specific, and radiation-free alternative for imaging the abdomen. In addition to evaluating fetal heart rate, ultrasonography can assess gestational age, fetal activity or demise, placental location, and amniotic fluid volume. MRI has not been associated with adverse fetal outcomes. Diagnostic peritoneal lavage remains a valid modality for evaluating the pregnant abdominal trauma patient, though it has largely been replaced by ultrasonography. If it is indicated, use the open supraumbilical technique.

Auscultate fetal heart tones to determine fetal viability and identify fetal distress early in the evaluation. A Doppler stethoscope or ultrasound facilitates this assessment. A normal fetal heart rate is in the range of 120 to 160 beats/min. Fetal bradycardia is most likely a result of hypoxia due to maternal hypotension, respiratory compromise, or placental abruption. Fetal tachycardia is most likely due to hypoxia or hypovolemia. In the setting of blunt abdominal trauma, external fetal monitoring is indicated for at least 4 to 6 hours for all patients beyond week 20 of gestation. Fetal tachycardia, lack of beat-to-beat or long-term variability, or late decelerations on tocodynamometry are diagnostic of fetal distress and may be indications for emergent cesarean section if beyond the viable gestational age.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

As is the case of all trauma patients, initial priorities are the primary and secondary surveys directed at the pregnant trauma patient. Coordinate care with surgical and obstetric consultants.

1. Aggressive maternal resuscitation is the best fetal resuscitation.
2. Initiate supplemental oxygen and crystalloid infusions. For patients beyond week 20 of gestation who must remain supine, place a wedge under the right hip, tilting the patient 30° to the left, thus reducing the

likelihood of supine hypotension syndrome. Otherwise, keep the patient in a left lateral decubitus position.

3. Perform early gastric intubation to reduce the risk of aspiration.
4. Avoid **vasopressors** if possible as they can have deleterious effects on uterine perfusion.
5. Administer **tetanus prophylaxis** when indicated.
6. Consider **Rho (D) immunoglobulin** 300 micrograms IM for all nonsensitized Rh-negative pregnant patients after abdominal trauma.
7. Institute cardiotocodynamometry as soon as possible to monitor for fetal distress and uterine contractions.
8. **Tocolytics** have a variety of side effects, including fetal and maternal tachycardia. Administer only in consultation with an obstetrician.
9. Indications for emergent laparotomy in the pregnant patient remain the same as those in the nonpregnant patient.
10. The decision to admit or discharge a pregnant trauma patient is first based on the nature and severity of the presenting injuries. Admit patients who display evidence of fetal distress or increased uterine irritability during initial observation.
11. Instruct discharged patients to seek medical attention immediately if they develop abdominal pain or cramps, vaginal bleeding, leakage of fluid, or perception of decreased fetal activity.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 253, "Trauma in Pregnancy," by Nicole M. DeIorio.

# **CHAPTER 160**

## **Head Trauma in Adults and Children**

O. John Ma

### **CLINICAL FEATURES**

Traumatic brain injury (TBI) is the impairment in brain function after direct or indirect forces to the brain. The force of an object striking the head or a penetrating injury causes direct injury. Indirect injuries occur from acceleration/deceleration forces that result in the movement of the brain within the skull.

TBI can be classified as mild, moderate, and severe. Mild TBI includes patients with a Glasgow Coma Scale (GCS, see [Table 160-1](#)) score  $\geq 14$ . Patients may be asymptomatic with only a history of head trauma, or may be confused and amnesic of the event. They may have experienced a brief loss of consciousness and complain of a diffuse headache, nausea, and vomiting. Patients at high risk in this subgroup include those with a skull fracture, large subgaleal swelling, focal neurologic findings, coagulopathy, age > 60 years, or drug/alcohol intoxication.



	4 years to Adult	Child <4 years	Infant
<b>Eye opening</b>			
4	Spontaneous	Spontaneous	Spontaneous
3	To speech	To speech	To speech
2	To pain	To pain	To pain
1	No response	No response	No response
<b>Verbal response</b>			
5	Alert and oriented	Oriented, social, speaks, interacts	Coos, babbles
4	Disoriented conversation	Confused speech, disoriented, consolable, aware	Irritable cry
3	Speaking but nonsensical	Inappropriate words, inconsolable, unaware	Cries to pain
2	Moans or unintelligible sounds	Incomprehensible, agitated, restless, unaware	Moans to pain
1	No response	No response	No response
<b>Motor response</b>			
6	Follows commands	Normal, spontaneous movements	Normal, spontaneous movements
5	Localizes pain	Localizes pain	Withdraws to touch
4	Moves or withdraws to pain	Withdraws to pain	Withdraws to pain
3	Decorticate flexion	Decorticate flexion	Decorticate flexion
2	Decerebrate extension	Decerebrate extension	Decerebrate extension
1	No response	No response	No response
3 to 15			

Note: In intubated patients, the Glasgow Coma Scale verbal component is scored as a 1 and the total score is marked with a "T" (or tube) denoting intubation (eg, 8T).

**TABLE 160-1** Glasgow Coma Scale for All Age Groups

Moderate TBI include patients with a GCS score of 9 to 13. Overall, 40% of these patients have a positive CT scan and 8% require neurosurgical intervention.

The mortality of severe TBI (GCS score <9) approaches 40%. The immediate clinical priority in these patients is to prevent secondary brain injury, identify other life-threatening injuries, and identify treatable neurosurgical conditions.

Prehospital medical personnel often may provide critical parts of the history, including mechanism and time of injury, presence and length of unconsciousness, initial mental status, seizure activity, vomiting,

verbalization, and movement of extremities. For an unresponsive patient, contact family and friends to gather key information including past medical history; medications (especially anticoagulants); and recent use of alcohol or drugs.

Clinically important features of the neurologic examination that should be addressed include assessing the mental status and GCS; pupils for size, reactivity, and anisocoria; cranial nerve function; motor, sensory, and brainstem function; and noting any development of decorticate or decerebrate posturing.

Infants with TBI demonstrate a global diminished level of responsiveness. Pupillary or facial asymmetry, extremity motor function abnormality, or a decreased sucking reflex may be found. Signs of increased intracranial pressure in infants include decreased arousal, lethargy, seizure, vomiting, apnea, and bradycardia. Signs or symptoms of TBI in the older child include headache, nausea, vomiting, diminished level of consciousness, motor weakness, visual changes, hypertension, bradycardia, and respiratory arrest.

## **Specific Injuries**

*Skull Fractures.* Depressed skull fractures are classified as open or closed, depending on the integrity of the overlying scalp. Although basilar skull fractures can occur at any point in the base of the skull, the typical location is in the petrous portion of the temporal bone. Findings associated with a basilar skull fracture include hemotympanum, cerebrospinal fluid (CSF) otorrhea or rhinorrhea, periorbital ecchymosis (“raccoon eyes”), and retroauricular ecchymosis (Battle sign). In children, linear skull fractures that result from a fall from a small height (<4 ft) generally are not associated with the development of clinically significant intracranial lesions. Significant intracranial injuries in children often occur after falls from more extreme heights or higher impact collisions.

*Cerebral Contusion and Intracerebral Hemorrhage.* Common locations for contusions are the frontal poles, the subfrontal cortex, and the temporal lobes. Contusions may occur directly under the site of impact or on the contralateral side (contrecoup lesion). The contused area is usually

hemorrhagic with surrounding edema, and occasionally associated with subarachnoid hemorrhage. Neurologic dysfunction may be profound and prolonged, with patients demonstrating mental confusion, obtundation, or coma. Focal neurologic deficits are usually present.

*Traumatic Subarachnoid Hemorrhage.* This condition results from the disruption of subarachnoid vessels and presents with blood in the CSF. Patients may complain of diffuse headache, nausea, or photophobia. Traumatic subarachnoid hemorrhage may be the most common CT abnormality in patients with moderate or severe TBI. Some cases may be missed if the CT scan is obtained less than 6 hours after injury.

*Epidural Hematoma.* An epidural hematoma results from an acute collection of blood between the inner table of the skull and the dura mater. It is typically associated with a skull fracture that lacerates a meningeal artery, most commonly the middle meningeal artery. Underlying injury to the brain may not necessarily be severe. In the classic scenario, the patient experiences loss of consciousness after a head injury. The patient may present to the ED with clear mentation, signifying the “lucid interval,” and then begin to develop mental status deterioration in the ED. A fixed and dilated pupil on the side of the lesion with contralateral hemiparesis is a classic late finding. The high pressure arterial bleeding of an epidural hematoma can lead to herniation within hours of injury. An epidural hematoma appears biconvex on CT scan.

*Subdural Hematoma.* A subdural hematoma (SDH), which is a collection of venous blood between the dura matter and the arachnoid, results from tears of the bridging veins that extend from the subarachnoid space to the dural venous sinuses. A common mechanism is sudden acceleration-deceleration. Patients with brain atrophy, such as in alcoholics or the elderly, are more susceptible to a SDH. In infants, SDH is strongly associated with nonaccidental trauma. In acute SDH, patients present within 14 days of the injury, and most become symptomatic within 24 hours of injury. After 2 weeks, patients are defined as having a chronic SDH. Symptoms may range from a headache to lethargy or coma. It is important to distinguish between acute and chronic subdural hematomas by history, physical examination, and CT scan. An acute subdural hematoma appears as a hyperdense, crescent-shaped lesion that crosses suture lines.

*Herniation.* Diffusely or focally increased intracranial pressure (ICP) can result in herniation of the brain at several locations. *Transtentorial (uncal) herniation* occurs when a subdural hematoma or temporal lobe mass forces the ipsilateral uncal of the temporal lobe through the tentorial hiatus into the space between the cerebral peduncle and the tentorium. This results in compression of the oculomotor nerve and parasympathetic paralysis of the ipsilateral pupil, causing it to become fixed and dilated. When the cerebral peduncle is further compressed, it results in contralateral motor paralysis. The increased ICP and brainstem compression result in progressive deterioration in the level of consciousness. Occasionally, the contralateral cerebral peduncle is forced against the free edge of the tentorium on the opposite side, resulting in paralysis ipsilateral to the lesion—a false localizing sign. *Central transtentorial herniation* occurs with midline lesions in the frontal or occipital lobes, or in the vertex. Bilateral pinpoint pupils, bilateral Babinski signs, and increased muscle tone are found initially, which eventually develop into fixed midpoint pupils, prolonged hyperventilation, and decorticate posturing. *Cerebellotonsillar herniation* through the foramen magnum occurs much less frequently. Medullary compression causes flaccid paralysis, bradycardia, respiratory arrest, and sudden death.

*Penetrating Injuries.* Gunshot wounds and penetrating sharp objects can result in penetrating injury to the brain. The degree of neurologic injury will depend on the energy of the missile, whether the trajectory involves a single or multiple lobes or hemispheres of the brain, the amount of scatter of bone and metallic fragments, and whether a mass lesion is present.

*Shaken Baby Syndrome.* This potential life-threatening head injury in children <2 years is caused by rapid acceleration and rotation of the head. Shearing injuries of the brain or intracranial vessels and cervical spine injuries may result. Almost half of children found with this syndrome exhibit no external signs of trauma, so clinical vigilance must remain high.

## **DIAGNOSIS AND DIFFERENTIAL**

[Tables 160-2](#) and [160-3](#) provide evidence-based indications for obtaining a CT scan of the head after injury.

New Orleans Criteria—GCS 15*	Canadian CT Head Rule—GCS 13-15*
Headache	GCS <15 at 2 h
Vomiting	Suspected open or depressed skull fracture
Age >60 years	Any sign of basal skull fracture
Intoxication	More than one episode of vomiting
Persistent antegrade amnesia	Retrograde amnesia >30 min
Evidence of trauma above the clavicles	Dangerous mechanism (fall >3 ft or struck as pedestrian)
Seizure	Age ≥65 years
<b>Identification of patients who have an intracranial lesion on CT</b>	
100% sensitive, 5% specific	83% sensitive, 38% specific
<b>Identification of patients who will need neurosurgical intervention</b>	
100% sensitive, 5% specific	100% sensitive, 37% specific

Key: GCS = Glasgow Coma Scale.

\*Presence of any one finding indicates need for CT scan.

**TABLE 160-2** New Orleans and Canadian CT Clinical Decision Rules

<p><i>Mild TBI even if no loss of consciousness</i> if one or more of the following is present:</p> <ul style="list-style-type: none"> <li>Glasgow Coma Scale score &lt;15</li> <li>Focal neurologic findings</li> <li>Vomiting more than twice</li> <li>Moderate to severe headache</li> <li>Age &gt;65 years</li> <li>Physical signs of basilar skull fracture</li> <li>Coagulopathy</li> <li>Dangerous mechanism of injury (eg, fall &gt;4 ft)</li> </ul>
<p><i>Mild TBI with loss of consciousness or amnesia</i> if one or more of the following is present:</p> <ul style="list-style-type: none"> <li>Drug or alcohol intoxication</li> <li>Physical evidence above the clavicles</li> <li>Persistent amnesia</li> <li>Posttraumatic seizures</li> </ul>

**TABLE 160-3** Summary of Indications for CT Scanning for Adults with Mild Traumatic Brain Injury (TBI)

Approximately 8% of patients suffering a severe TBI will have an associated cervical spine fracture. Obtain imaging studies of the cervical spine on all trauma patients who present with altered mental status, neck pain, intoxication, neurologic deficit, severe distracting injury, or if the mechanism of injury is deemed serious enough to potentially produce cervical spine injury.

Laboratory work should include type and crossmatching, complete blood count, basic metabolic panel, arterial blood gas analysis, directed toxicologic studies, and coagulation studies.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Initiate standard protocols for evaluation and stabilization of trauma patients (see [Chapter 156](#)). Search carefully for other significant injuries.
2. Administer **100% oxygen**, and secure cardiac monitoring and two IV lines. For patients with severe TBI, **endotracheal intubation** (via rapid sequence intubation) to protect the airway and prevent hypoxemia is the top priority. Provide cervical spine immobilization, and use an adequate sedation/induction agent when securing the airway.
3. Hypotension is associated with increased mortality rates. Restoration of an adequate blood pressure is vital to maintain cerebral perfusion. Resuscitation with **IV crystalloid fluid** to a mean arterial pressure (MAP)  $\geq 80$  mm Hg is indicated; if aggressive fluid resuscitation is not effective, then add **vasopressors** to maintain a **MAP  $\geq 80$  mm Hg**.
4. Obtain immediate **neurosurgical consultation** after a head CT scan demonstrating intracranial injury has been identified. Patients with new neurologic deficits from an acute epidural or subdural hematoma require emergent neurosurgical treatment.
5. All patients who demonstrate signs of increased ICP should have the **head of their bed elevated 30°** (provided that the patient is not hypotensive), adequate sedation, and maintenance of adequate arterial oxygenation. If the patient is not hypotensive, consider administering **mannitol**, 0.25 to 1.0 gram/kilogram IV bolus.

6. Hyperventilation is not recommended as a prophylactic intervention to lower ICP because of its potential to cause cerebral ischemia. Reserve hyperventilation as a last resort for decreasing ICP; if used, implement it as a temporary measure and monitor the  $PCO_2$  closely to maintain a range of 30 to 35 mm Hg.
7. Patients with signs of impending brain herniation may need emergency decompression by trephination (“burr holes”) when all other methods to control the elevated ICP have failed. CT scan prior to attempting trephination is recommended to localize the lesion and direct the decompression site.
8. Treat seizures immediately with **benzodiazepines**, such as lorazepam, and **fosphenytoin** at a loading dose of 18 to 20 milligrams PE/ kilogram IV.
9. Use of prophylactic anticonvulsants remains controversial, and its administration should be in consultation with the neurosurgeon.
10. Admit patients with a basilar skull fracture or penetrating injuries (gunshot wound or stab wound) to the neurosurgical service, and start them on prophylactic antibiotic (eg, ceftriaxone 1 gram every 12 hours) therapy.
11. Discharge patients who have an initial GCS score of 15 that is maintained during an observation period and who have normal serial neurologic examinations and a normal CT scan. Those who have an abnormal CT scan require neurosurgical consultation and admission. Patients who have an initial GCS score of 14 and a normal CT scan should be observed in the ED. If their GCS score improves to 15 and they remain symptom free and neurologically intact after serial examinations, they can be discharged home. Discharge patients home with a reliable companion who can observe them for at least 24 hours, carry out appropriate discharge instructions, and follow the Head Injury sheet instructions.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 254, “Head Trauma in Adults and Children” by David W. Wright and Lisa H. Merck.

## **CHAPTER 161**

# **Spine and Spinal Cord Injuries in Adults and Children**

Todd W. Ellingson

Spine and spinal cord injuries (SCIs) can be devastating, life-changing events that include injury to the bony elements (vertebral fracture), the neural elements (spinal cord and nerve root injury), or both.

## **CLINICAL FEATURES**

The spinal cord is most commonly injured by a direct mechanical cause, with resultant hemorrhage, edema, and ischemia. Patients may demonstrate neurogenic shock or spinal shock after a spinal cord injury. Neurogenic shock refers to the loss of sympathetic innervation leading to bradycardia and hypotension. The hypotension must be differentiated from hypovolemia due to hemorrhage. Spinal shock refers to the temporary loss or depression of spinal reflex activity below a spinal cord injury that can persist for days to weeks and prohibit the differentiation of an incomplete and complete lesion.

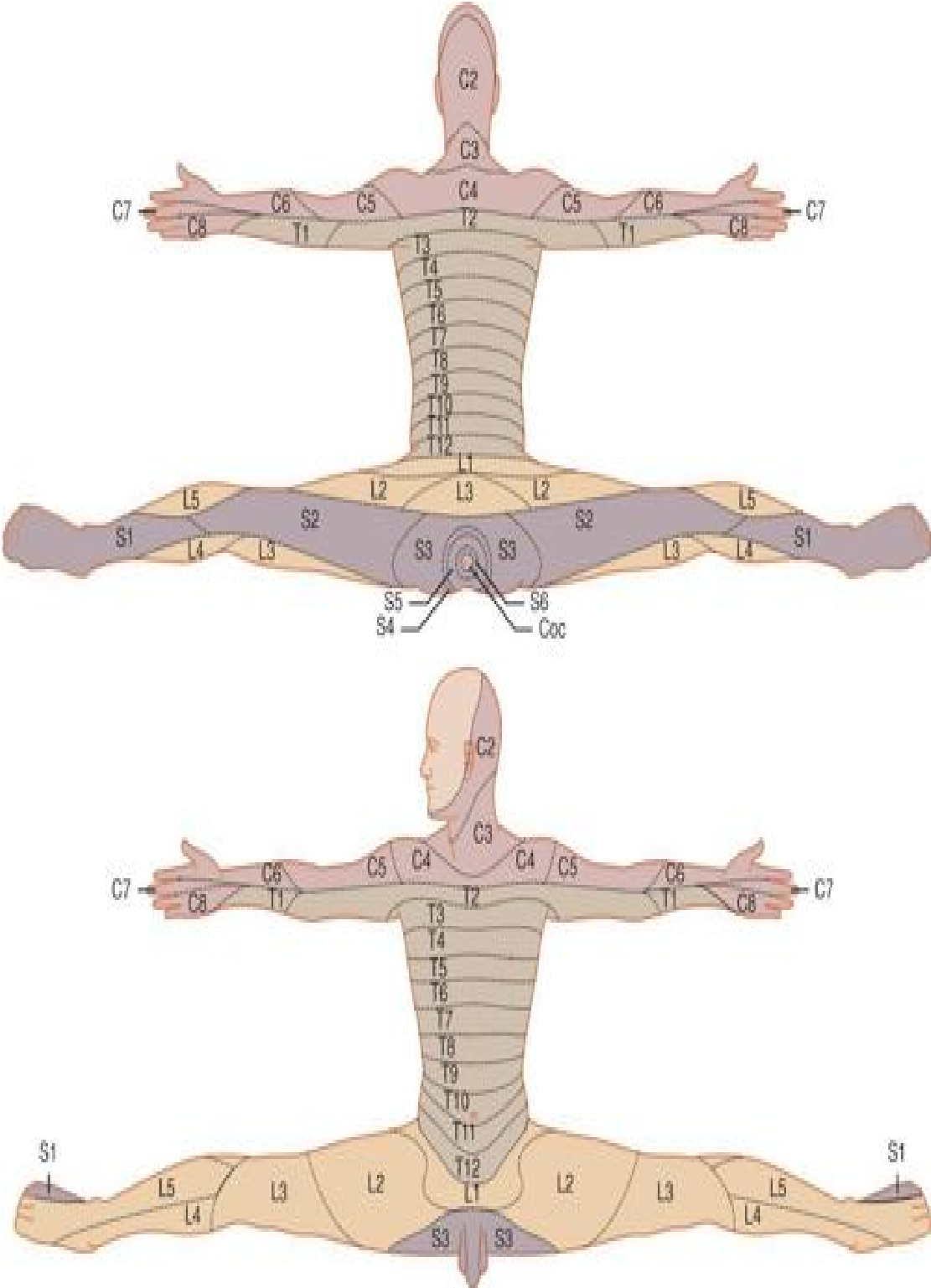
Patients may complain of neck and back pain, and close examination may note pain or bony abnormalities with palpation. Many unstable spinal fractures may present without spinal cord or nerve root trauma. Symptomatic patients may complain of paresthesias, dysesthesias, weakness, bowel or bladder incontinence, urinary retention, or other sensory disturbances with or without specific physical examination findings. More severely injured patients may have obvious neurologic deficits.

## **DIAGNOSIS AND DIFFERENTIAL**

Consider an injury to the spine or spinal cord in any patient with an appropriate traumatic mechanism. Suspect SCI with any neurologic complaints, even if transitory. A complete neurologic examination should include motor strength and tone (corticospinal tract), pain and temperature sensations (spinothalamic tract), proprioception and vibration sensations (dorsal columns), reflexes, perianal sensation and wink, and bulbocavernosus reflex. “Sacral sparing” denotes preservation of



reflexes and an incomplete SCI. [Fig. 161-1](#) demonstrates the dermatomes for the sensory examination.



**FIGURE 161-1.** Dermatomes for sensory examination.

Validated clinical guidelines exist to identify patients who benefit from cervical spine imaging. The NEXUS ([Table 161-1](#)) and the Canadian Cervical Spine Rule for Radiography ([Table 161-2](#)) are intended for alert, stable adult patients.

Absence of midline cervical tenderness
Normal level of alertness and consciousness
No evidence of intoxication
Absence of focal neurologic deficit
Absence of painful distracting injury

**TABLE 161-1** National Emergency X-Radiography Utilization Study Criteria: Cervical Spine Imaging Unnecessary in Patients Meeting These Five Criteria

Question or Assessment	Definitions
There are no high-risk factors that mandate radiography.	High-risk factors include: Age 65 years or older A dangerous mechanism of injury (fall from a height of >3 ft; an axial loading injury; high-speed motor vehicle crash, rollover, or ejection; motorized recreational vehicle or bicycle collision) The presence of paresthesias in the extremities
There are low-risk factors that allow a safe assessment of range of motion.	Low-risk factors include: Simple rear-end motor vehicle crashes Patient able to sit up in the ED Patient ambulatory at any time Delayed onset of neck pain Absence of midline cervical tenderness
The patient is able to actively rotate his/her neck.	Can rotate 45° to the left and to the right

**TABLE 161-2** Canadian Cervical Spine Rule for Radiography: Cervical Spine Imaging Unnecessary in Patients Meeting These Three Criteria

High-resolution CT is more sensitive and specific for cervical spine fractures than plain films, and is the modality of choice at most trauma centers for suspected cervical spine injuries. For plain radiography of the cervical spine, at least three views (lateral, odontoid, and anteroposterior) are necessary.

Both CT and plain radiography can miss purely ligamentous injuries. Flexion-extension films can identify cervical spine instability. As muscle tone can splint bones in a stable configuration causing these films to be normal when a ligamentous injury exists, reliable patients can be discharged with a hard collar for follow-up in 3 to 5 days by a spinal surgeon. Alternatively, MRI provides the most sensitive and specific view of the ligaments and neural structures. The mechanisms, characteristics, and stability of common cervical spine fractures are summarized in [Table 161-3](#).

Flexion
Anterior subluxation (hyperflexion sprain) (stable)*
Bilateral interfacetal dislocation (unstable)
Simple wedge (compression) fracture (usually stable)
Spinous process avulsion (clay-shoveler's fracture) (stable)
Flexion teardrop fracture (unstable)
Flexion-rotation
Unilateral interfacetal dislocation (stable)
Pillar fracture
Fracture of lateral mass (can be unstable)
Vertical compression
Jefferson burst fracture of atlas (potentially unstable)
Burst (bursting, dispersion, axial-loading) fracture (unstable)
Hyperextension
Hyperextension dislocation (unstable)
Avulsion fracture of anterior arch of atlas (stable)
Extension teardrop fracture (unstable)
Fracture of posterior arch of atlas (stable)
Laminar fracture (usually stable)
Traumatic spondylolisthesis (hangman's fracture) (unstable)
Lateral flexion
Uncinate process fracture (usually stable)
Injuries caused by diverse or poorly understood mechanisms
Occipital condyle fractures (can be unstable)
Occipitoatlantal dissociation (highly unstable)
Dens fractures (type II and III are unstable)

\*Usual occurrence. Overall stability is dependent on integrity of the other ligamentous structures.

**TABLE 161-3** Cervical Spine Injuries

Initial imaging of the thoracic or lumbar spine may include plain radiography, but the use of CT imaging primarily has become more common. Indications for imaging of the thoracic and lumbar spine are based on the mechanism, the physical examination, and the associated injuries ([Table 161-4](#)). The vertebral injuries discovered of the thoracic and lumbar spinal column are divided into minor and major injuries ([Table 161-5](#)).

Mechanism	Gunshot High energy Motor vehicle crash with rollover or ejection Fall > 10 ft or 3 m Pedestrian hit by car
Physical examination	Midline back pain Midline focal tenderness Evidence of spinal cord or nerve root deficit
Associated injuries	Cervical fracture Rib fractures Aortic injuries Hollow viscous injuries

**TABLE 161-4** Indications for Thoracic and Lumbar Imaging after Trauma

Minor Injuries	Major Injuries
Transverse process fracture	Compression (wedge) fractures
Spinous process fracture	Burst fractures
Pars interarticularis fractures	Flexion-distraction ("seat-belt") injuries Fracture-dislocation (translation) injuries

**TABLE 161-5** Thoracic and Lumbar Spine Fractures

For patients with obvious SCI, the differential diagnosis includes complete lesions and a number of incomplete lesions or syndromes. The differences between complete lesions, the absence of sensory and motor function below the level of injury, and incomplete lesions is crucial; prognosis for complete lesions is poor. The characteristics of some of the more common incomplete syndromes are listed in [Table 161-6](#). Spinal cord injury without radiographic abnormality (SCIWORA) is an entity seen most often in pediatric population. Numbness, paresthesias, or other neurologic complaints with normal plain radiographs or CT should prompt further evaluation with MRI as the modality of choice.

Syndrome	Etiology	Symptoms	Prognosis*
Anterior cord	Direct anterior cord compression Flexion of cervical spine Thrombosis of anterior spinal artery	Complete paralysis below the lesion with loss of pain and temperature sensation Preservation of proprioception and vibratory function	Poor
Central cord	Hyperextension injuries Disruption of blood flow to the spinal cord Cervical spinal stenosis	Quadriparesis—greater in the upper extremities than the lower extremities; some loss of pain and temperature sensation, also greater in the upper extremities	Good
Brown-Séquard	Transverse hemisection of the spinal cord Unilateral cord compression	Ipsilateral spastic paresis, loss of proprioception and vibratory sensation, and contralateral loss of pain and temperature sensation	Good
Cauda equina	Peripheral nerve injury	Variable motor and sensory loss in the lower extremities, sciatica, bowel/bladder dysfunction, and "saddle anesthesia"	Good

\*Outcome improves when the effects of secondary injury are prevented or reversed.

**TABLE 161-6** Incomplete Spinal Cord Syndromes

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

Treat blunt and penetrating injuries to the spine with identification and stabilization of identified injuries, and prevention of secondary injuries.

1. Airway assessment and management with **inline cervical immobilization** is the first treatment in the ED. Maintain a low threshold for endotracheal intubation for patients with cervical spine injury at C5 and above because the diaphragm is innervated by C3 through C5. Place the patient on high-flow oxygen and establish 2 large-bore IV lines. Fluid resuscitation facilitates spinal cord resuscitation; control obvious bleeding, and rapidly assess for other life-threatening injuries.
2. Roll patients (while maintaining inline spinal immobilization) to identify any obvious fractures or associated injuries and remove from hard backboards to prevent skin breakdown. Perform a thorough neurologic examination and note any abnormalities.
3. Obtain spine surgeon consultation as soon as an injury is identified. Treat spinal shock and neurogenic shock with oxygen, IV fluids, and, if necessary, positive inotropic pressors. Bradycardia may require a pacemaker. Previously, high-dose steroids (methylprednisolone) were recommended for spinal cord injuries as a result of blunt trauma; however, other studies of its efficacy indicated harmful side effects that outweighed the potential clinical benefit. Consultation with the spinal surgeon is recommended prior to administration of the high-dose steroid protocol. Should they be considered, the steroid algorithm includes a bolus followed by a 23-hour infusion ([Table 161-7](#)). Steroids are not recommended for penetrating spinal cord injuries.

Indications	Blunt trauma Neurologic deficit referable to the spinal cord Treatment can be started within 8 h of injury
Treatment	Methylprednisolone, 30 milligrams/kilogram bolus, is administered IV over 15 min Followed by a 45-min pause Methylprednisolone, 5.4 milligrams/kilogram/h, is then infused for 23 h

**TABLE 161-7** The National Acute Spinal Cord Injury Study Protocol

4. Admit any patient with a significant injury to the spine or spinal cord or any patient with significant associated injury to the hospital. Further, admit any patient with intractable pain, nerve root injury, or intestinal ileus.
5. Discharge patients who are adequately evaluated and found to have no indications for admission with appropriate follow-up in 3 to 5 days. Provide these patients analgesics (nonsteroidal anti-inflammatory drugs or opioids) and specific return precautions.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 255, “Spine and Spinal Cord Trauma,” by Bonny J. Baron, Kevin J. McSherry, James L. Larson, Jr., and Thomas M. Scalea.

# CHAPTER 162

## Facial Injuries

Jonathan S. Ilgen

Severe facial injuries are associated with injuries to the brain, orbit, cervical spine, and lungs. Upon stabilization of life-threatening injuries during the primary survey, a thorough secondary survey should identify facial injuries that could affect the patient's normal appearance, vision, smell, mastication, and sensation.

### CLINICAL FEATURES

To help localize injuries, a thorough history should begin with questions directed toward whether the patient has vision changes, malocclusion, or facial numbness ([Table 162-1](#)). The physical examination begins with inspection, noting facial asymmetry, facial elongation, exophthalmos or enophthalmos, and periorbital or mastoid ecchymoses. Next, palpate the entire face, noting step-offs and tenderness that suggest fractures, and crepitus that suggests a sinus fracture. Finally, perform a focused and thorough examination of the eyes, nose, ears, and mouth, as described in [Table 162-1](#).

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### History

How is your vision?

Binocular diplopia suggests entrapment of the extraocular muscles; monocular diplopia suggests a lens dislocation.

Do any parts of your face feel numb?

Anesthesia suggests damage to the supraorbital, infraorbital, or mental nerves.

Does your bite feel normal?

Malocclusion typically occurs with mandibular or maxillary fractures.

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### Inspection

Lateral view for dish face with Le Fort III fractures.

Frontal view for donkey face with Le Fort II or III fractures.

Bird's eye view for exophthalmos with retrobulbar hematoma.

Worm's view for endophthalmos with blow-out fractures or flattening of malar prominence with zygomatic arch fractures.

Raccoon's eyes (bilateral periorbital ecchymosis) and Battle sign (mastoid ecchymosis) typically develop over several hours, suggesting basilar skull fracture.

---

### Palpation

Palpating the entire face will detect the majority of fractures.

Intraoral palpation of the zygomatic arch, palpating lateral to posterior maxillary molars to distinguish bony from soft tissue injury.

Assess for Le Fort fractures by gently rocking the hard palate with one hand while stabilizing the forehead with the other.

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<p><b>Eye</b></p> <p>Examine early before swelling of lids, or use retractors. Document visual acuity. Fat through eyelid wound indicates an orbital septum perforation. Widening of the distance between the medial canthi, or telecanthus, suggests serious nasoethmoidal-orbital complex trauma. Widening of the distance between the pupils, or hypertelorism, results from orbital dislocation and often is associated with blindness. Examine extraocular muscle movements. Limited upward gaze occurs with entrapment of the inferior rectus or inferior oblique muscles, or damage to the oculomotor nerve. Systematically examine the eye. Specifically, the pupil for teardrop sign pointing to globe rupture, the anterior chamber for hyphema, and swinging flashlight test for afferent papillary defect. Perform a fluorescein test for corneal abrasions or ulcers. Check intraocular pressure for evidence of orbital compartment syndrome only in absence of globe injury.</p>
<p><b>Nose</b></p> <p>Crepitus over any facial sinus suggests sinus fracture. Septal hematoma appears as blue, boggy swelling on nasal septum. Should be incised and drained to avoid a saddle nose deformity.</p>
<p><b>Ears</b></p> <p>Auricular hematomas should be incised and drained to avoid a cauliflower deformity. Cerebrospinal fluid leak, auditory canal lacerations, and hemotympanum suggest basilar skull fracture.</p>
<p><b>Oral</b></p> <p>Jaw deviation due to mandible dislocation or condyle fracture. Malocclusion occurs in mandible, zygomatic, and Le Fort fractures. Missing or injured tooth. Lacerations and mucosal ecchymosis suggest mandible fracture. Place finger in external ear while the patient gently opens and closes jaw to detect condyle fractures. Tongue blade test: Patient without fracture can bite down on a tongue blade enough to break blade twisted by examiner.</p>

**TABLE 162-1** Important Clinical Issues in Facial Trauma

## **DIAGNOSIS AND DIFFERENTIAL**

Diagnosis of many maxillofacial injuries is made clinically and with radiographs. Plain films are helpful if CT is not available or to screen for injuries in low-risk patients. Facial CT is frequently required to make the definitive diagnosis and guide surgical management. Imaging recommendations based on suspected injury sites and pretest clinical suspicion are summarized in [Table 162-2](#).

Level	Low Suspicion	Significant Clinical Findings	Additional Considerations
Frontal bone	Head CT	Head CT (skull windows)	Facial CT with orbital involvement. Cervical spine CT with significant clinical findings.
Midface	Waters' view	Face CT with coronal and axial sections	Coronal face sections require cervical spine clearance for positioning. Computer-generated, 3-dimensional reconstructions with complex injuries. Head CT can replace Water view.
Mandible	Panorex	Mandible CT	Facial CT detects mandible fractures.

**TABLE 162-2** Recommendations for Imaging Based on Level of Injury and Clinical Findings

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

During the primary survey of facial trauma patients, the airway must be secured and stabilized. When endotracheal intubation is required, the orotracheal route is preferred over the nasotracheal route because of concern for nasocranial intubation or severe epistaxis. While rapid sequence intubation is the preferred method of airway management in trauma, always plan for a difficult airway in patients with facial trauma. To prevent the “can’t intubate/can’t oxygenate” failed airway, do not administer paralytics unless a patient can be bagged effectively or alternative airway devices or plans are in place. Awake intubation with sedation and local airway anesthesia may allow the emergency physician to determine the feasibility of orotracheal intubation while still preserving a patient’s airway reflexes. When endotracheal intubation appears impossible or is unsuccessful, perform emergent cricothyroidotomy to secure the airway. The laryngeal mask airway may be used as a temporizing measure, but it does not protect the airway from aspiration of stomach contents and may not be possible with injuries involving the pharynx.

Severe midfacial and mandibular injuries can result in significant hemorrhage from the sphenopalatine and greater palatine branches of the

external carotid artery. Posterior nasal epistaxis can be controlled with nasal tampons, dual balloon devices, or Foley catheter placement, again being careful to avoid intracranial placement in severe midfacial fractures. Rarely, reduction of significantly displaced nasal fractures and Le Fort injuries is needed to stop arterial bleeding. If bleeding persists, immediate operative intervention may be required in order to ligate injured vessels. Alternatively, arterial embolization may be pursued to control bleeding from branches of the external carotid artery.

Management decisions will be dictated by the location and severity of the facial fractures, as well as concurrent injuries. All patients with sinus fractures should receive oral or intravenous antibiotics, such as first-generation cephalosporins, clindamycin, or amoxicillin/clavulanate.

**Frontal sinus fractures** are uncommon, and increase the immediate risk of traumatic brain injury, additional facial fractures, and cervical spine injury. Because the dura is adherent to the posterior table of frontal sinus, operative repair of through-and-through frontal sinus fractures is necessary to prevent pneumocephalus, cerebrospinal fluid (CSF) leak, and infection. Patients with depressed fractures also require admission for IV antibiotics and operative repair. Patients with isolated anterior table fractures of the frontal sinus may be discharged with appropriate follow-up with a facial surgeon.

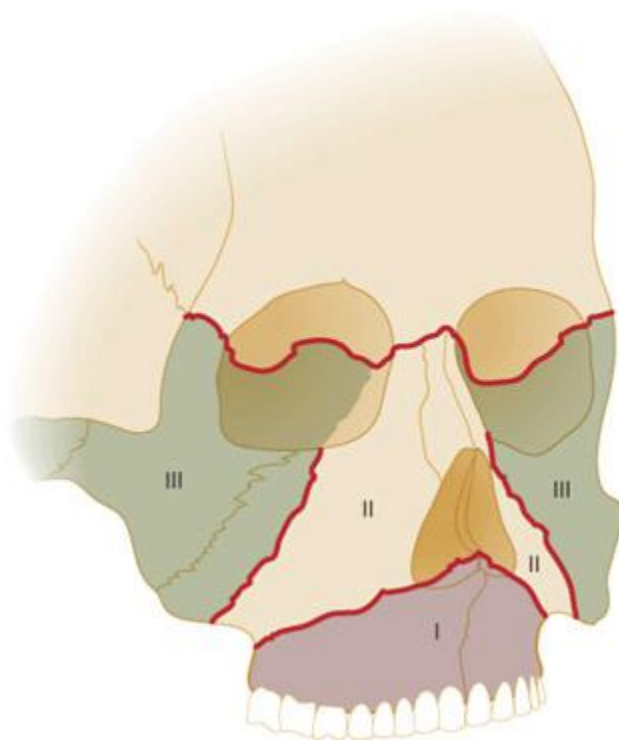
**Naso-orbito-ethmoid fractures** result from significant trauma to the nasal bridge, and often have associated injury to the lacrimal duct, dural tears, and traumatic brain injury. Patients with these fractures require admission for specialty consultation with facial surgery and neurosurgery.

**Orbital blowout fractures** occur when a blunt object strikes the globe, transmitting force through the fluid-filled eye, and fracturing the medial or inferior orbital wall. Surgery may be required if these injuries result in extraocular muscle or oculomotor nerve entrapment, or significant enophthalmos. A fracture involving the superior orbital fissure can damage the oculomotor and ophthalmic divisions of the trigeminal nerve (the “orbital fissure syndrome”), and can involve the optic nerve as well (the “orbital apex syndrome”). Patients with either of these syndromes require emergent ophthalmologic consultation. All other patients with isolated orbital fractures can be managed expediently as an outpatient with oral

antibiotics, decongestants, and instructions to avoid nose blowing until the defect has been repaired.

**Zygoma fractures** occur in 2 major patterns: tripod fractures and isolated zygomatic arch fractures. **Tripod fractures** involve disruption of the infraorbital rim, the zygomaticofrontal suture, and the zygomaticotemporal junction. These fractures require admission for open reduction and internal fixation. Patients with isolated fractures of the zygomatic arch can have elective outpatient repair.

**Midfacial fractures** are high-energy injuries and are often seen in victims of multisystem trauma. Patients frequently require endotracheal intubation for airway control. **Le Fort injury** patterns are illustrated in [Fig. 162-1](#). Visual acuity should be tested, especially with Le Fort III fractures, in which the incidence of blindness is high. Both Le Fort II and III injuries can result in CSF leaks. Le Fort injuries require admission for management of significant associated injuries, IV antibiotics, and surgical repair.



**FIGURE 162-1.** Le Fort Injury patterns. Illustration of the fracture lines of Le Fort I (alveolar), Le Fort II (zygomatic maxillary complex), and Le Fort III (cranial facial dysostosis) fractures.

(Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ: *Atlas of Emergency Medicine, 3rd ed.* 2010, © McGraw-Hill, New York.)

**Mandible fractures** are often diagnosed in the setting of malocclusion and pain with attempted movement. Always look for multiple mandibular fractures, with one injury at the site of impact and a second subtle injury on the opposite side of the ring. A careful intraoral examination is important to exclude small breaks in the mucosa seen with open fractures, sublingual hematomas, and dental or alveolar ridge fractures. Patients with closed fractures may be given urgent outpatient follow-up, while open fractures require admission for IV antibiotics and operative repair. In the patient with a stable airway, a Barton bandage, an ace wrap over the top of the head and underneath the mandible, will stabilize the fracture and help relieve pain.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 256, “Trauma to the Face,” by John Bailitz.

# CHAPTER 163

## Trauma to the Neck

Katrina A. Leone

Blunt and penetrating neck trauma cause a diverse combination of injuries because of the high concentration of vascular, aerodigestive, and neurologic structures in the neck. Initial presenting signs of neck injury may be subtle or obscured by trauma to other body regions, especially in the setting of blunt trauma. Missed injuries and delays in diagnosis lead to patient morbidity and mortality.

### CLINICAL FEATURES

Historical and physical examination findings of neck injury are characterized as hard or soft signs ([Table 163-1](#)), with hard signs most suggestive of significant injuries. Laryngotracheal symptoms are shown in [Table 163-2](#). Pharyngeal and esophageal injuries may initially present with few symptoms. The presence of hematemesis, hemoptysis, dysphagia, or neck emphysema is suggestive of significant injury. Vascular injuries present with a range of symptoms. Expanding hematomas have the potential to cause airway distortion and obstruction. Focal neurologic abnormalities caused by cerebral ischemia occur in the setting of carotid artery injury. A carotid bruit or thrill is a subtle examination finding of vascular injury. Neurologic injuries result from trauma to the cervical spine, lower cranial nerves, or brachial plexus. Symptoms range from sensory complaints to quadriplegia.

Hard Signs	Soft Signs
Hypotension in ED	Hypotension in field
Active arterial bleeding	History of arterial bleeding
Diminished carotid pulse	Unexplained bradycardia (without central nervous system injury)
Expanding hematoma	
Thrill/bruit	Nonexpanding large hematoma
Lateralizing signs	Apical capping on chest radiograph
Hemothorax > 1000 mL	Stridor
Air or bubbling in wound	Hoarseness
Hemoptysis	Vocal cord paralysis
Hematemesis	Subcutaneous emphysema
Tracheal deviation	Seventh cranial nerve injury

**TABLE 163-1** Signs and Symptoms of Neck Injury

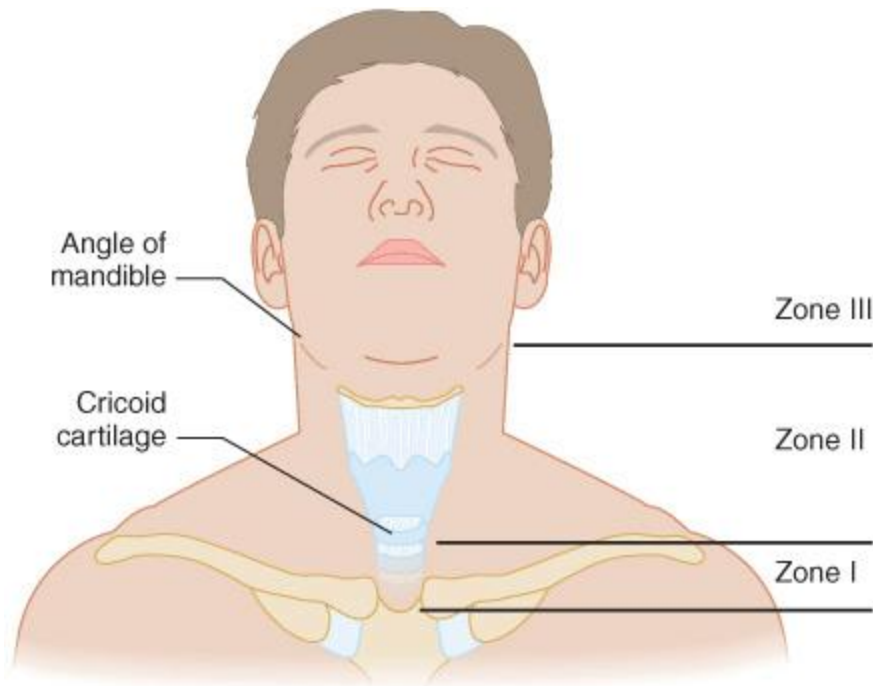
Symptoms	Signs
Dysphonia	Stridor
Hoarseness	Neck tenderness
Dysphagia	Subcutaneous emphysema
Odynophagia	Cervical ecchymosis or hematoma
Dyspnea	Tracheal deviation
Hemoptysis	Laryngeal edema or hematoma
	Restricted vocal cord mobility

**TABLE 163-2** Symptoms and Signs of Laryngotracheal Injury

Strangulation is a unique mechanism of blunt neck injury caused by hanging, ligature application, or manual neck compression. The clinical presentation of strangulation depends upon the duration and amount of force applied to the neck. Cardiac arrest, cervical spine fractures, cerebral anoxia, hyoid bone, and laryngeal injuries are possible. Increased venous pressure above the location of a ligature causes facial and conjunctival petechial hemorrhages.

## **DIAGNOSIS AND DIFFERENTIAL**

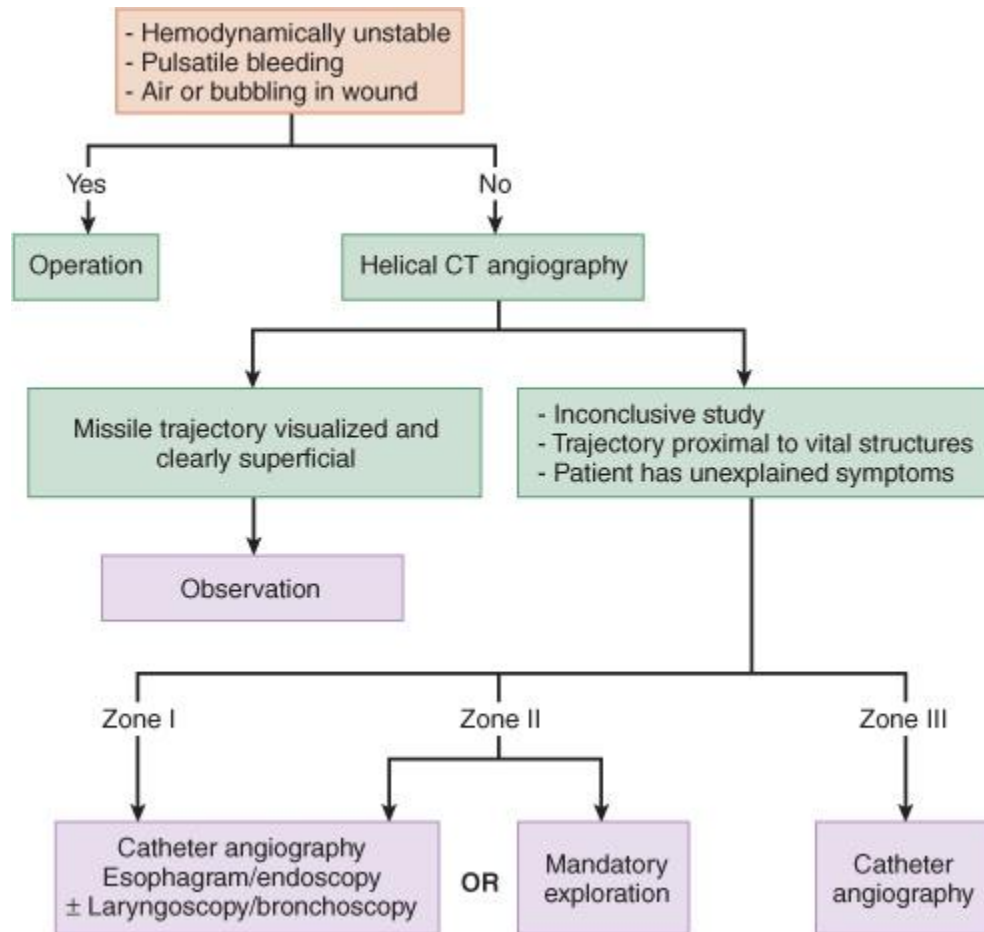
The zone classification summarizes structures placed at risk for injury in penetrating neck trauma ([Fig. 163-1](#)). Zone I structures include the lung apices, thoracic vessels, distal trachea, esophagus, cervical spine, and vertebral and carotid arteries. Zone II structures include the mid carotid and vertebral arteries, jugular veins, esophagus, cervical spine, larynx, and trachea. Zone III structures include the proximal carotid and vertebral arteries, oropharynx, and cervical spine.



**FIGURE 163-1.** Zones of the neck.

Diagnostic studies utilized in the workup of neck trauma include chest radiography, CT of the cervical spine, helical CT angiography of the neck, angiography, laryngoscopy or bronchoscopy, esophagram, and esophagoscopy. An algorithm for the diagnosis and management of penetrating neck injuries is shown in [Fig. 163-2](#). [Table 163-3](#) describes pros and cons of the diagnostic and therapeutic interventions available for vascular injuries.





**FIGURE 163-2.** Algorithm for management of penetrating neck injuries.

Imaging Modality	Advantages	Disadvantages
Catheter angiography	Gold standard Both diagnostic and therapeutic Access to zone I and III injuries where surgical repair is difficult	Invasive Expensive Labor intensive Requires skilled operators
Helical CT angiography	Readily available Fast Minimally invasive Visualization of missile trajectory High-resolution images of vascular, aerodigestive, and bone structures with single study	Only diagnostic, not therapeutic Requires IV contrast Image quality affected by technique of contrast injection Metallic streak artifact may obscure findings Limited evaluation of low zone I and high zone III injuries May miss small intimal flaps, pseudoaneurysms, and arteriovenous fistulas
Surgical exploration	Direct visualization and repair of vascular and aerodigestive injuries May find injuries missed by diagnostic studies Low complication rate Repair of complex skin defects	Invasive Large percentage of negative explorations Difficult vascular control in zones I and III
Duplex ultrasonography	Noninvasive Inexpensive No contrast medium	Highly operator dependent Limited view of zones I and III Obscured by subcutaneous emphysema and hematomas May miss small lesions

**TABLE 163-3** Vascular Evaluation of Penetrating Neck Trauma

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Initiate standard ATLS protocol for the stabilization of trauma patients. Establish oxygen, cardiac and respiratory monitoring, and IV access.
2. Management of the airway is of utmost concern. Endotracheal intubation is indicated for patients with symptoms described in [Table 163-4](#). If oral or nasal intubation is not possible or is contraindicated, perform a cricothyrotomy.

Acute respiratory distress
Airway obstruction from blood or secretions
Massive subcutaneous emphysema of the neck
Tracheal shift
Alteration in mental status
Expanding neck hematoma

**TABLE 163-4** Clinical Factors Indicating Need for Aggressive Airway Management

3. Injuries at the base of the neck predispose patients to simultaneous injury to the chest. Perform a needle thoracostomy to relieve tension pneumothorax, and tube thoracostomy is indicated for pneumothorax and hemothorax.
4. Apply direct pressure to control active hemorrhage. Blind clamping of blood vessels is contraindicated due to a risk of subsequent neurovascular injury. Initiate fluid resuscitation with crystalloid followed by blood products.
5. Immobilize the cervical spine in patients with a blunt mechanism of injury, altered level of consciousness, or penetrating mechanism with neurologic deficits.
6. Penetrating wounds that violate the platysma muscle mandate surgical consultation. Surgical exploration is indicated for aerodigestive injury or hemodynamic instability with Zone II vascular injuries. Stable patients should undergo diagnostic evaluation for deep structure injuries ([Fig. 163-2](#)).
7. Penetrating wounds that do not violate the platysma require standard wound care and closure. If asymptomatic, discharge these patients home with close follow-up after 4 to 6 hours of observation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 257, “Trauma to the Neck” by Bonny J. Baron.

# CHAPTER 164

## Cardiothoracic Trauma

Ross J. Fleischman

Amongst injuries associated with blunt chest trauma are tension pneumothorax, hemothorax, and cardiac tamponade, for which bedside diagnosis and immediate intervention by the emergency provider may be lifesaving.

### GENERAL PRINCIPLES AND CONDITIONS

Initial assessment and management of airway, breathing, circulation, and disability should follow the Advanced Trauma Life Support (ATLS) primary survey guidelines. Intubate patients in respiratory distress ([Table 164-1](#)). Maintaining good oxygenation is especially important in preventing secondary injury in head-injured patients. Investigate for tension pneumothorax and cardiac tamponade during the primary survey for all patients in shock after chest trauma.

Altered mental status
Hypovolemic shock
Multiple injuries
Multiple blood transfusions
Elderly patient
Preexisting pulmonary disease
Respiratory rate >30 to 35 breaths/min
Vital capacity <10 to 15 mL/kg
Negative inspiratory force <25 to 30 cm of water

**TABLE 164-1** Considerations for Early Ventilatory Assistance after Thoracic Trauma

Physical examination should include assessment for tracheal deviation, unequal chest rise, abnormal breath sounds, visible trauma to the chest wall, subcutaneous emphysema (suggestive of pneumothorax), open chest

wounds, and bowel sounds in the chest (suggestive of diaphragmatic injury).

In the hemodynamically unstable, polytrauma patient who requires immediate operation without CT imaging, exclude immediate threats to life with rapid bedside tests (physical examination, chest radiograph, ultrasound, and chest tube, as needed). Excessive fluid administration may worsen edema in patients with pulmonary contusions. Administer fluids judiciously with crystalloids to maintain perfusion, and use blood products early in resuscitation.

If subclavian venous cannulization is attempted, it should be done on the side of suspected injury so that an iatrogenic pneumothorax does not result in bilateral pneumothoraces. Check for a tension pneumothorax or tube displacement in any patient who suddenly decompensates while on mechanical ventilation.

## **CHEST WALL INJURIES**

A small open (sucking) chest wound can progress to a tension pneumothorax through a one-way valve effect. Cover the wound with sterile petroleum gauze taped on 3 sides to allow air to exit but not enter. Immediately insert a chest tube at another site and not through the wound.

Flail chest occurs when fracture of a section of ribs in multiple places allows instability of a section of the chest wall. Intubation and positive pressure ventilation will stabilize the flail segment, so intubate patients with respiratory compromise, along with those with evidence of shock, severe head injury, preexisting pulmonary disease, fracture of eight or more ribs, and age > 65 years. Insert a chest tube to relieve an identified or suspected pneumothorax. Surgical fixation may be needed.

Rib fractures may suggest other injuries or cause morbidity in themselves. Fractures of the first and second ribs require great force, and should therefore cause high suspicion and evaluation for other major thoracic injuries including myocardial, vascular, and bronchial injuries. Multiple lower rib fractures should raise suspicion for liver or splenic injuries. Simple rib fractures are often a clinical diagnosis, with up to 50%

not being seen on plain radiographs. Direct diagnostic imaging at excluding other injuries, such as pneumothorax, pulmonary contusion, and intraabdominal injury.

Even in the absence of coexisting injury, the pain of rib fractures may eventually lead to splinting, ventilatory compromise, and pneumonia. Consider intercostal nerve blocks and epidural anesthesia for pain control. Patients being discharged should generally receive NSAIDs and opioid analgesics. They should be reminded to breathe deeply or taught more formal incentive spirometry exercises. Admit patients with multiple fractures, medical comorbidities, or older age for a period of observation until they are stabilized on a regimen of pain control and pulmonary toilet to avoid complications. Attempts to stabilize the chest wall with tape or binding are no longer recommended. Assess patients with a sternal fracture for cardiac injury by ECG and cardiac monitoring.

Assume patients with subcutaneous emphysema have a pneumothorax even if not seen on the initial chest radiograph. Penetrating wounds should never be deeply probed.

## **LUNG INJURIES**

Tension pneumothorax occurs when air enters the pleural space, either by escaping from damaged lung, tracheobronchial tissue, or from an open chest wound and becomes pressurized during respiration, causing respiratory and circulatory compromise. Patients may have dyspnea, tachycardia, hypotension, distended neck veins, tracheal deviation, and unequal breath sounds. Recognize and treat tension pneumothorax immediately without waiting for radiographs. Immediate needle decompression treatment involves insertion of a 14-gauge, 4.5-cm over-the-needle catheter in the second intercostal space at the midclavicular line (a shorter catheter may not reach the pleural space in many patients). A rush of air through the catheter is confirmatory. Leave the catheter in until a chest tube can be inserted, as the catheter converts the tension pneumothorax to an open pneumothorax.

Supine chest radiograph is a relatively insensitive screening tool (52%) for pneumothorax and for hemothoraces of <200 mL. Up to 1000 mL may appear as only diffuse haziness. Lung collapse from intubation of a mainstem bronchus can have a similar appearance. If the patient can safely sit up, upright and expiratory views can increase sensitivity. Ultrasound has been shown to have good sensitivity for pneumothorax. Using a high-frequency linear probe, signs of pneumothorax include loss of the sliding pleura sign. A hemothorax will show fluid in the dependent portion of the chest. CT scan is highly sensitive for both of these conditions. A small stab wound may develop into a delayed pneumothorax, so a repeat chest radiograph at 4 to 6 hours after initial presentation is prudent.

Treat a small pneumothorax with inpatient observation; insertion of a chest tube may not be necessary. For a larger pneumothorax without suspicion of hemopneumothorax, insert a 24-to-28F (8.0 to 9.3 mm) chest tube. If blood is suspected in the chest, insert a 32-to-40F (10.7 to 13.4 mm) chest tube. Patients with pneumothoraces of any size and those with subcutaneous emphysema (requiring presumption of an occult pneumothorax) who will be intubated or who will be transported by air should have a chest tube inserted, as positive pressure ventilation and decreased barometric pressure can cause expansion of trapped air to convert into a tension pneumothorax. Never clamp a chest tube, but always place on water seal when taken off suction. See [Table 164-2](#) for causes of the lung not to fully reinflate after chest tube placement.

Improper connections or leaks in the external tubing or water-seal collection apparatus
Improper positioning of the chest tube
Occlusion of bronchi or bronchioles by secretions or foreign body
Tear of one of the large bronchi
Large tear of the lung parenchyma

**TABLE 164-2** Causes for Failure of Complete Lung Expansion or Evacuation of a Pneumothorax

Treat a hemothorax with tube thoracostomy. Indications for surgery include an immediate return of 1 L of blood or ongoing bleeding of 150 to 200 mL/h for 2 to 4 hours. Consider using a heparinized autotransfusion device.

Pulmonary contusions are direct injuries to the lung parenchyma without laceration. Hypoxia ensues since contused lung tissue is compromised by bleeding and edema. Seventy percent of pulmonary contusions are not immediately visible on initial chest radiograph, but may appear as patchy opacities. CT scan is much more sensitive. Radiographic findings of fat embolism and aspiration pneumonia usually appear as patchy opacities 12 to 24 hours after injury, while pulmonary contusions appear within the first 6 hours. Initial management should include pain control to prevent hypoventilation, avoidance of unnecessary fluid infusion to prevent pulmonary edema, and strict pulmonary toilet. A trial of positive pressure ventilation by mask is reasonable in a patient with normal mental status who requires limited respiratory support. Patients with involvement of > 25% of lung tissue will likely require intubation, but should not be intubated obligatorily. If intubated, use positive end expiratory pressure. Use diuretics if the patient is thought to have volume overload. Steroids are not recommended.

## **PNEUMOMEDIASTINUM AND TRACHEOBRONCHIAL INJURIES**

Major deceleration injuries can result in injuries to the trachea and large airways, usually within 2 cm of the carina or at the origin of lobar bronchi. Dyspnea, hemoptysis, subcutaneous emphysema in the neck, a crunching sound with the cardiac cycle (Hamman sign), and a massive continued air leak through a chest tube suggest tracheobronchial injury. Mediastinal air, large pneumothorax, and a round appearance of the endotracheal tube balloon on plain radiograph or CT also suggest tracheobronchial injuries.

Coughing, heavy breathing (such as seen in drug inhalation), or exertion can rupture alveoli and release air into the mediastinum. In the absence of major trauma, treat this type of pneumomediastinum expectantly once pneumothorax has been excluded.

## **DIAPHRAGMATIC INJURIES**



All penetrating injuries from the level of the nipples to the umbilicus have the potential to injure the diaphragm. Small lacerations can be asymptomatic and then progress to the rupture of abdominal contents into the chest weeks to months later. The diagnosis is obvious if imaging shows herniation of abdominal contents into the chest or coiling of a gastric tube within the chest. Subtle abnormalities may also be seen on chest radiograph, CT, or upper GI series with contrast. Laparotomy or laparoscopy remain the gold standards to exclude diaphragmatic injuries. All diaphragmatic lacerations require surgical repair.

## **PENETRATING CARDIAC INJURIES**

The right ventricle is the most commonly injured portion of the heart because of its large anterior exposure. Recognition of cardiac tamponade by the emergency provider is critical because cardiac tamponade can potentially be temporized in the ED by pericardiocentesis prior to definitive operative management. Accumulation of blood in the pericardial space compresses the heart, which prevents filling during diastole. Beck's triad of hypotension, distended neck veins, and muffled heart tones may be seen. Confirm the diagnosis by bedside ultrasound. Pericardiocentesis is technically difficult and may result in laceration of a coronary artery, injury to the myocardium, or inability to withdraw blood. Therefore, it should only be attempted for a patient in shock with confirmed cardiac tamponade, in which case it may be lifesaving. Stable patients should have a pericardial window or thoracotomy performed in the operating room.

Patients with penetrating chest injuries with signs of life in the field but who subsequently and suddenly become pulseless may be candidates for ED thoracotomy. Relieving cardiac tamponade, cross clamping the descending aorta, or repairing a myocardial laceration may be lifesaving.

## **BLUNT INJURIES TO THE HEART**

Blunt cardiac injury can lead to death from damage to cardiac structures, coronary artery injury and thrombosis, and contusion of the myocardium resulting in impaired contractility and arrhythmias. If a patient with myocardial rupture survives to ED arrival, a “splashing mill wheel” murmur may be heard. Confirm the diagnosis by echocardiogram and treat with emergency thoracotomy. ECG changes consistent with ischemia suggest coronary artery dissection or thrombosis, which are evaluated and treated by cardiac catheterization and stenting. A direct blow to the chest such as when a young athlete is struck by a hard ball can induce ventricular fibrillation cardiac arrest even without myocardial injury (commotio cordis).

A patient with cardiac injury may present with chest pain, tachycardia unexplained by hemorrhage, and arrhythmias. Bedside echocardiogram by the emergency provider should be performed as a first screen for cardiac tamponade and grossly impaired contractility. Patients with hypotension not explained by another cause, arrhythmias, and impaired contractility should undergo further evaluation by formal echocardiography and cardiac enzymes, with transesophageal echocardiogram being three times more sensitive than transthoracic echocardiogram for blunt myocardial injury. Give anti-arrhythmic and inotropic medications according to advanced cardiac life support (ACLS) algorithms. Indications for admission include abnormalities on echocardiogram, ECG, or cardiac enzymes. Discharge patients with normal vital signs, normal initial ECG, no underlying cardiac disease, and age <55 years after 4 to 6 hours of normal cardiac monitoring.

## **PERICARDIAL SYNDROME**

## **INFLAMMATION**

Patients with pericardial inflammation syndrome may develop chest pain, fever, and a friction rub 2 to 4 weeks after cardiac trauma or surgery. ECG may show the diffuse ST-segment elevation of pericarditis. Pericardial and pleural effusions may be seen on echocardiography and chest radiograph, respectively. Treatment is with nonsteroidal anti-inflammatory medications, such as **indomethacin** 25 to 50 milligrams every 6 hours.

# TRAUMA TO THE GREAT VESSELS

Trauma to the major thoracic vessels is often lethal, with 90% of those sustaining blunt aortic injury dying at the scene. The most common site of blunt aortic injury is at the proximal descending aorta between the left subclavian artery and the ligamentum arteriosum. Injury to the subclavian and innominate arteries can be related to shoulder belts, fractures of the first and second ribs, and proximal clavicle. Clinical findings may include unequal bilateral blood pressures, diminished lower extremity pulses, and new murmurs. Descending aortic injuries may cause paraplegia, mesenteric ischemia, anuria, and lower extremity ischemia if they affect flow to the relevant arteries. [Table 164-3](#) shows radiographic findings of thoracic aortic injury, although a normal chest radiograph does not exclude major vascular injury. All patients with a mechanism highly concerning for great vessel injury should undergo CT angiogram with IV contrast. Conventional aortography is still used in some cases to assess injuries and guide operative planning. Transesophageal echocardiogram is useful for diagnosing aortic intimal lesions, but is contra-indicated in airway compromise or suspected cervical spinal injury.

Fractures
Sternum
Scapula
Multiple ribs
Clavicle in multisystem-injured patients
First rib
Mediastinal clues
Obliteration of the aortic knob contour
Widening of the mediastinum
Depression of the left mainstem bronchus >140° from trachea
Loss of paravertebral pleural stripe
Calcium layering at aortic knob
Abnormal general appearance of mediastinum
Deviation of nasogastric tube to the right at T4
Lateral displacement of the trachea
Lateral chest x-ray
Anterior displacement of the trachea
Loss of the aortic/pulmonary window
Other findings
Apical pleural hematoma (cap)
Massive left hemothorax
Obvious diaphragmatic injury

Reproduced with permission from Mattox KL, Wall MJ Jr, LeMaire SA: Injury to the thoracic great vessels, in Mattox KL, Feliciano DV, Moore EE (eds): *Trauma*, 4th ed. New York, McGraw-Hill, 2000, pp. 559–582.

**TABLE 164-3** Radiographic Findings Suggestive of a Great Vessel Injury

With gunshot wounds, a discrepancy between the number of presumed entrance and exit wounds and bullets seen on imaging should make the provider consider entry into a vessel with embolization to another part of the body. A fuzzy appearance of a projectile on plain radiograph suggests an intravascular missile vibrating with pulsatile blood flow.

Indications for immediate operation for vascular injury are hemodynamic instability, radiographic evidence of a rapidly expanding hematoma, or large chest tube output. Patients with other injuries, advanced age, or uncontrolled medical comorbidities may require stabilization before definitive operative or intravascular repair. Control hypertension in order to decrease shear stress on the vessel wall by titration of narcotic pain medications and sedatives. A short-acting  $\beta$ -blocker, such as **esmolol** 50 to 300 micrograms/kilogram/min infusion, may be titrated to a systolic blood pressure of 100 to 120 mm Hg and a heart rate above 60 beats/min. If

bradycardia prevents further dosing of a  $\beta$ -blocker, infuse an arterial dilator such as **sodium nitroprusside** 0.25 to 10 micrograms/kilogram/min IV.

## **ESOPHAGEAL AND THORACIC DUCT INJURIES**

Penetrating, and occasionally blunt, trauma may cause injury to the thoracic esophagus. If suspected, evaluate the patient by esophagram with water-soluble contrast, which is less likely to cause mediastinitis. A negative study with water-soluble contrast should be followed by the use of barium contrast, which has a higher sensitivity for injury. Flexible esophagoscopy is an alternative modality for assessing injury. Delayed diagnosis of esophageal injury has a high mortality if mediastinitis ensues. Injuries to the area of the left proximal subclavian vein may result in chylothorax, which usually is discovered as a delayed right-sided pleural effusion. Initial treatment is with chest tube drainage.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 258, "Pulmonary Trauma," by Patrick H. Brunett, Lalena M. Yarris, and Arif Alper Cevik; and Chapter 259, "Cardiac Trauma," by Christopher Ross and Theresa M. Schwab.

# **CHAPTER 165**

## **Abdominal Injuries**

O. John Ma

The primary goal in the evaluation of abdominal trauma is to promptly recognize conditions that require immediate surgical exploration. The most critical error is to delay surgical intervention when it is needed.

### **CLINICAL FEATURES**

#### **Solid Visceral Injuries**

Injury to the solid organs causes morbidity and mortality, primarily as a result of acute blood loss. The spleen is the most frequently injured organ in blunt abdominal trauma and is commonly associated with other intraabdominal injuries. The liver also is commonly injured in blunt and penetrating injuries. Tachycardia, hypotension, and acute abdominal tenderness are the primary physical examination findings. Kehr sign, representing referred left shoulder pain, is a classic finding in splenic rupture. Lower left rib fractures should heighten clinical suspicion for splenic injury. Some patients with solid organ injury occasionally may present with minimal symptoms and nonspecific findings on physical examination. This is commonly associated with younger patients and those with distracting injuries, head injury, or intoxication. A single physical examination is insensitive for diagnosing abdominal injuries. Serial physical examinations on an awake, alert, and reliable patient are important for identifying intraabdominal injuries.

#### **Hollow Visceral Injuries**

These injuries produce symptoms by the combination of blood loss and peritoneal contamination. Perforation of the stomach, small bowel, or colon is accompanied by blood loss from a concomitant mesenteric injury. Gastrointestinal contamination will produce peritoneal signs over time. Patients with head injury, distracting injuries, or intoxication may not exhibit peritoneal signs initially.

Small bowel and colon injuries are most frequently the result of penetrating trauma. However, a deceleration injury can cause a bucket-handle tear of the mesentery or a blow-out injury of the antimesenteric border. Suppurative peritonitis may develop from small bowel and colonic injuries. Inflammation may take 6 to 8 hours to develop.

## **Retroperitoneal Injuries**

The diagnosis of retroperitoneal injuries can be difficult. Signs and symptoms may be subtle or absent at initial presentation. Duodenal injuries most often are associated with high-speed vertical or horizontal decelerating trauma. These injuries may range in severity from an intramural hematoma to an extensive crush or laceration. Duodenal ruptures are usually contained within the retroperitoneum. Clinical signs of duodenal injury are often slow to develop. Patients may present with abdominal pain, fever, nausea, and vomiting, although these symptoms may take hours to become clinically apparent.

Pancreatic injury often accompanies rapid deceleration injury or a severe crush injury. The classic case is a blow to the midepigastrium from a steering wheel or the handlebar of a bicycle. Pancreatic injuries can present with subtle signs and symptoms, making the diagnosis elusive. Leakage of activated enzymes from the pancreas can produce retroperitoneal autodigestion, which may become superinfected with bacteria and produce a retroperitoneal abscess.

## **Diaphragmatic Injuries**

Presentation of diaphragm injuries is often insidious. Only occasionally is the diagnosis obvious when bowel sounds can be auscultated in the thoracic cavity. Herniation of abdominal contents into the thoracic cavity or a nasogastric tube coiled in the thorax confirms the diagnosis on chest radiograph. In most cases, however, the only finding on chest radiograph is blurring of the diaphragm or an effusion.

(Urologic injuries may occur from abdominal trauma and are discussed in [Chapter 167](#).)

## **DIAGNOSIS AND DIFFERENTIAL**

### **Plain Radiographs**

A chest radiograph is helpful in evaluating for herniated abdominal contents in the thoracic cavity and for evidence of free air under the diaphragm. An anteroposterior pelvis radiograph is important for identifying pelvic fractures, which can produce significant blood loss and be associated with intraabdominal visceral injury.

### **Ultrasonography**

The focused assessment with sonography for trauma (FAST) examination is an accurate screening tool for abdominal trauma. The underlying premise behind the use of the FAST examination is that clinically significant injuries will be associated with the presence of free fluid (eg, blood) accumulating in dependent areas. Advantages and disadvantages of the FAST examination are listed in [Table 165-1](#).



Advantages	Disadvantages
Accurate, sensitive, and specific for detecting free intraperitoneal fluid	Inability to determine the exact etiology of the free intraperitoneal fluid
Rapid (<4 min)	Operator-dependent
Noninvasive Repeatable Portable	Difficulty in interpreting the images in patients who are obese or have subcutaneous air or excessive bowel gas
No nephrotoxic contrast material needed	Inability to distinguish intraperitoneal hemorrhage from ascites
No radiation exposure Can evaluate for free pericardial and pleural fluid	Cannot evaluate the retroperitoneum as well as CT scan
Can evaluate for pneumothorax	
No risk for patients who are pregnant, coagulopathic, or have had previous abdominal surgery	

**TABLE 165-1** Advantages and Disadvantages of the Focused Assessment with Sonography for Trauma Examination

### Computed Tomography

CT of the abdominal/pelvis has a greater specificity than ultrasonography, thus making it the initial diagnostic test of choice. Most CT protocols for imaging the abdomen and pelvis following blunt trauma utilize intravenous contrast. Patients with penetrating wounds to the retroperitoneal structures (particularly the colon and rectum) may not exhibit any clinical manifestations for hours after injury. **A triple-contrast helical CT scan (PO, IV, and PR contrast) can quickly discern either contrast extravasation or the presence of air or fluid.** Advantages and disadvantages of CT are listed in [Table 165-2](#).

Advantages	Disadvantages
Ability to precisely locate intraabdominal lesions	Expense
Ability to evaluate the retroperitoneum	Need to transport the trauma patient to the radiology suite
Ability to identify injuries that may be managed nonoperatively	Need for contrast materials
Noninvasive	Radiation exposure

**TABLE 165-2** Advantages and Disadvantages of CT in Abdominal Trauma

## Diagnostic Peritoneal Lavage

The wide availability of CT and ED ultrasound has relegated diagnostic peritoneal lavage (DPL) to a second-line screening test for evaluating abdominal trauma. Advantages and disadvantages of DPL are listed in [Table 165-3](#).

Advantages	Disadvantages
Sensitivity	Invasive
Availability	Potential for iatrogenic injury
Relative speed with which it can be performed	Lack of specificity
Low complication rate	Inability to identify injuries that may be managed nonoperatively
Ability to detect early evidence of bowel perforation	Misapplication for evaluation of retroperitoneal injuries
No nephrotoxic contrast material needed	
No radiation exposure	

**TABLE 165-3** Advantages and Disadvantages of Diagnostic Peritoneal Lavage in Abdominal Trauma

For blunt trauma, indications for DPL include (a) patients who are too hemodynamically unstable to leave the ED for CT and (b) unexplained hypotension in patients with an equivocal physical examination. DPL is considered positive if more than 10 mL of gross blood is aspirated immediately, the red blood cell count is higher than 100,000 cells/mm<sup>3</sup>, the white blood cell count is higher than 500 cells/mm<sup>3</sup>, bile is present, or if vegetable matter is present.

The only absolute contraindication to DPL is when surgical management is clearly indicated, in which case the DPL would delay patient transport to the operating room. Relative contraindications include patients with advanced hepatic dysfunction, severe coagulopathies, previous abdominal surgeries, or a gravid uterus.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Standard protocols for evaluation and stabilization of trauma patients should be initiated (see [Chapter 156](#)).
2. Administer **100% oxygen**, attach cardiac monitoring, and secure 2 large-bore IV lines.
3. Administer **IV crystalloid fluid** to hypotensive abdominal trauma patients. Transfuse with O-negative or type-specific packed red blood cells as indicated.
4. Laboratory work for patients with abdominal trauma should be based on the mechanism of injury (blunt vs penetrating); it may include type and crossmatching, complete blood count, electrolytes, arterial blood gas, directed toxicologic studies, coagulation studies, hepatic enzymes, and lipase.
5. [Table 165-4](#) lists the **indications for exploratory laparotomy**. When a patient presents to the ED with an obvious high-velocity gunshot wound to the abdomen, do not perform DPL or the FAST examination because it will only delay transport of the patient to the operating room. If organ evisceration is present, cover the wound with a moist, sterile dressing before surgery.

	Blunt	Penetrating
Absolute	Anterior abdominal injury with hypotension Abdominal wall disruption Peritonitis Free air under diaphragm on chest radiograph Positive FAST or DPL in hemodynamically unstable patient CT-diagnosed injury requiring surgery (ie, pancreatic transection, duodenal rupture, diaphragm injury)	Injury to abdomen, back, and flank with hypotension Abdominal tenderness GI evisceration High suspicion for transabdominal trajectory after gunshot wound CT-diagnosed injury requiring surgery (ie, ureter or pancreas)
Relative	Positive FAST or DPL in hemodynamically stable patient Solid visceral injury in stable patient Hemoperitoneum on CT without clear source	Positive local wound exploration after stab wound

Key: DPL = diagnostic peritoneal lavage; FAST = focused assessment with sonography for trauma.

**TABLE 165-4** Indications for Laparotomy

6. For an equivocal stab wound to the abdomen, surgical consultation for local wound exploration is indicated. If the local wound exploration demonstrates no violation of the anterior fascia, the patient can be discharged home.
7. For the hemodynamically stable, blunt trauma patient with a positive FAST examination, further evaluation with CT may be warranted before admission to the surgical service.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 260, “Abdominal Trauma,” by Thomas M. Scalea, Sharon A. Boswell, Bonny J. Baron, and O. John Ma.

# **CHAPTER 166**

## **Penetrating Trauma to the Flank and Buttocks**

Christine Sullivan

Challenges in evaluating penetrating trauma to the flank and buttocks are recognizing peritoneal and retroperitoneal injuries and determining which patients need immediate surgery and which can be managed more conservatively. Mechanism and time of injury, weapon characteristics, and determining the bullet path or stab wound depth may assist in diagnosis.

### **PENETRATING FLANK TRAUMA**

#### **Clinical Features**

Presentation may vary significantly from hemodynamic shock and peritonitis to stable vital signs with an innocuous-appearing wound. Gross blood on rectal examination suggests bowel injury. Blood at the urethral meatus or hematuria suggests genitourinary injury.

#### **Diagnosis and Differential**

CT with oral and IV contrast is the diagnostic modality of choice for hemodynamically stable patients. Include rectal contrast if suspicious for rectal or sigmoid injuries. Contrast-enhanced CT can often determine stab wound depth.

#### **Emergency Department Care and Disposition**

1. Follow standard trauma resuscitation protocols. Patients who require emergent exploratory laparotomy include those who are

hemodynamically unstable, display peritonitis, and have sustained gunshot wounds to the flank.

2. If the patient displays signs of peritonitis, administer broad-spectrum antibiotics (eg, **pipercillin/tazobactam** 3.375 grams IV).
3. Most patients with stab wounds can be managed conservatively. Highrisk patients (stab wounds with penetration beyond deep fascia) require surgical consultation and admission. Low-risk patients (stab wounds superficial to deep fascia) may be discharged if serial examinations are unremarkable throughout an observation period.

## **PENETRATING BUTTOCK TRAUMA**

### **Clinical Features**

Gunshot wounds are much more likely to require laparotomy than stab wounds. Gunshot wounds above the level of the greater trochanter and gross hematuria predict the need for surgery. Rectal examination to assess for gross blood, assessment of lower extremity pulses, and neurologic examination to assess for sciatic and femoral nerve injury should be performed.

### **Diagnosis and Differential**

Hemodynamically stable patients should undergo CT with oral, IV, and rectal contrast (to avoid missed colon and rectal injuries). Cystourethrogram should be performed with findings of hematuria or wounds near the genitourinary tract. CT angiography or traditional angiography and venography may be indicated if pelvic hematoma is found on CT.

### **Emergency Department Care and Disposition**

1. Follow standard trauma resuscitation protocols. Patients who are hemodynamically unstable, display peritonitis, or have an intrapelvic or transabdominal bullet path require exploratory laparotomy.
2. If the patient displays signs of peritonitis, administer broad-spectrum antibiotics (eg, **pipercillin/tazobactam** 3.375 grams IV).
3. Interventional angiography may be required to treat extensive intrapelvic bleeding.
4. Wound exploration is of limited value. Only very superficial stab wounds may be managed and discharged from the ED. Most of these patients require admission and observation due to the risk of occult injuries.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 261, “Penetrating Trauma to the Flank and Buttocks” by Alasdair K. T. Conn.

# CHAPTER 167

## Genitourinary Injuries

Matthew C. Gratton

Genitourinary (GU) injuries frequently occur in the setting of multiple trauma, so a thorough evaluation is necessary to avoid missing significant injuries.

### CLINICAL FEATURES

Injuries should be suspected with any blunt or penetrating trauma near the GU tract, including any rapid deceleration, which can cause major vascular or parenchymal injury even without specific signs or symptoms. Hematuria of any amount raises the index of suspicion for GU injury, and difficulty with urination can be due to bladder or urethral injury or associated concomitant spinal cord injury. Flank contusions or hematomas, evidence of lower rib fractures, or penetrating flank injuries raise concern for renal injury. Lower abdominal pain, tenderness, ecchymosis, or evidence of a pelvic fracture as well as perineal or scrotal edema is consistent with possible bladder injury. Vaginal bleeding, high-riding prostate, perineal hematoma, and/or blood at the urethral meatus are consistent with urethral disruption.

### DIAGNOSIS AND DIFFERENTIAL

There is no direct relationship between the degree of hematuria and the severity of renal injury. There is some evidence that gross hematuria or microscopic hematuria in patients with a blood pressure <90 mm Hg is associated with more significant injury. An IV contrast-enhanced abdominal/pelvic CT scan is the imaging “gold standard” for the stable trauma patient with suspected kidney injury ([Table 167-1](#)). A “one-shot” intraoperative IV urogram is recommended by some for the unstable patient, though this is controversial. A retrograde cystogram (plain film or CT) is the “gold standard” for demonstrating bladder injury, and a retrograde urethrogram is indicated for demonstrating urethral injury. Color Doppler ultrasonography is the preferred imaging technique for investigating closed scrotal and testicular injury.



Blunt Trauma	Penetrating Trauma
Gross hematuria	Any degree of hematuria
Adult with blood pressure <90 mm Hg and any degree of hematuria	
Child with >50 red blood cells per high power field	
High index of suspicion for renal trauma	
Deceleration injuries (especially vertical) even with no hematuria	
Suspected other associated intraabdominal or intrapelvic injuries (multiple trauma patient)	

**TABLE 167-1** Indications for Imaging in Patients with Suspected Renal Trauma

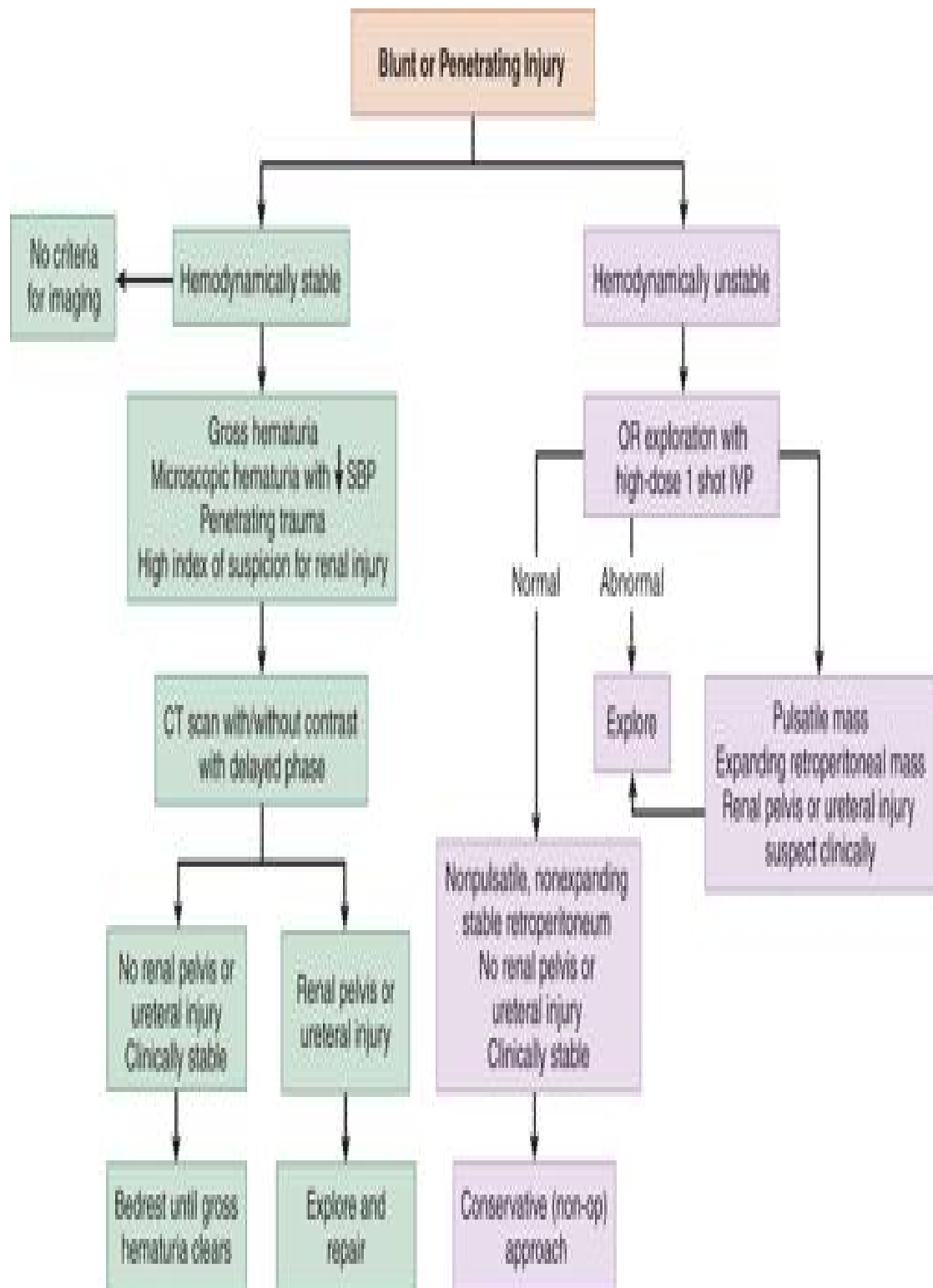
## EMERGENCY DEPARTMENT CARE AND DISPOSITION

Take a standardized approach to all multiple trauma patients to identify and treat life-threatening injuries (primary survey) and then perform a thorough secondary survey, including a GU examination, to diagnose all injuries. Obtain appropriate diagnostic imaging and laboratory testing as indicated by the initial history and examination.

## MANAGEMENT OF SPECIFIC INJURIES

### Kidney

Various kidney injuries include contusions, hematomas, lacerations, and completely shattered kidneys with or without vascular injury. Eighty percent of patients with kidney injury have additional visceral or skeletal injuries that complicate their management. Most renal injuries are handled nonoperatively, but indications for operative treatment include life-threatening bleeding from the kidney; expanding, pulsatile, or noncontained hematoma (thought to be from an avulsion injury); and renal avulsion injury ([Fig. 167-1](#)). There are little data to support specific treatment recommendations for patients with isolated renal trauma. Patients with microscopic hematuria and no indication for imaging can be discharged home with instructions for no strenuous activity and follow-up in 1 to 2 weeks for repeat urinalysis. Those with a contusion (normal imaging and microscopic hematuria) can be discharged as above. Those with a higher grade injury and/or gross hematuria should be admitted for observation (to include repeat hematocrit and urinalysis), hydration, and rest until gross hematuria clears, or general improvement ensues.



**FIGURE 167-1.** Algorithm for the conservative management of renal parenchymal injuries.

*Key:* IVP = IV pyelogram; non-op = nonoperative; OR = operating room; SBP = systolic blood pressure.

## **Ureter**

Ureteral injuries are almost always due to iatrogenic complications of instrumentation or penetrating trauma. Treatment is operative, including stenting in some cases. The absence of hematuria does not exclude injury.

## **Bladder**

Bladder injury occurs in about 2% of blunt abdominal trauma patients and 80% are associated with pelvic fractures. Gross hematuria is present in about 95% of patients with significant injury. Extraperitoneal rupture is most common and can usually be treated by bladder catheter drainage alone. Intraperitoneal rupture always requires surgical exploration and repair.

## **Urethra**

Posterior urethral injuries (membranous and prostatic urethra) are typically related to major blunt force trauma and are associated with pelvic fractures. Treatment is via suprapubic bladder drainage followed by surgical repair in several weeks. Because a urinary catheter can disrupt a partial posterior urethral injury, one should not be placed if there is suspicion of injury without first obtaining a retrograde urethrogram. Anterior urethral injuries usually occur due to direct trauma as from a straddle injury or a direct blow to the bulbar or penile urethra. Treatment is supportive, which may include a urinary catheter. Penetrating trauma to the anterior urethra generally requires operative repair.

## **Testicles and Scrotum**

Evaluate blunt testicular trauma with an ultrasound examination. If testicular rupture is present, exploration and repair is indicated. If the testicle is intact, then conservative treatment with ice, elevation, scrotal support, and pain medication is appropriate. Hematomas and hematoceles are managed on a case-by-case basis.

Penetrating testicular trauma warrants surgical exploration and repair. Scrotal lacerations can be directly repaired and scrotal avulsions require surgical repair with the testicle covered in the remaining scrotum.

## **Penis**

Simple contusions are managed conservatively with cold packs, rest, and pain medications. Simple lacerations involving skin only can be directly repaired, but deeper lacerations and/or penetrating injuries require operative exploration and repair. Amputation requires microsurgical reimplantation if the amputated segment is viable. Penile fractures require exploration and repair.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 262, "Genitourinary Trauma," by John McManus, Matthew C. Gratton, and Peter J. Cuenca.

# CHAPTER 168

## Penetrating Trauma to the Extremities

Amy M. Stubbs

Injuries to the extremities from gunshots, stab wounds, and other penetrating trauma can cause significant morbidity by damaging bones, nerves, soft tissue, and blood vessels. Early identification and treatment of these injuries is important to prevent permanent disability or loss of limbs.

### CLINICAL FEATURES

Ascertain the events surrounding the injury, including the type of weapon and number of shots or stabs. Obtain a thorough history of any prior injuries, deficits, or ischemic events in the affected limb. Perform a detailed vascular and neuromuscular examination.

When examining the patient, early identification of any arterial injury is crucial. Note pulses distal to the injury, capillary refill, and the color and temperature of the limb. Any “hard” signs of arterial injury should prompt immediate surgical consultation and intervention. “Soft” signs of arterial injury should also be noted and require observation and surgical consultation ([Table 168-1](#)).

Hard signs
Absent or diminished distal pulses
Obvious arterial bleeding
Large expanding or pulsatile hematoma
Audible bruit
Palpable thrill
Distal ischemia (pain, pallor, paralysis, paresthesias, coolness)
Soft signs
Small, stable hematoma
Injury to anatomically related nerve
Unexplained hypotension
History of hemorrhage
Proximity of injury to major vascular structures
Complex fracture

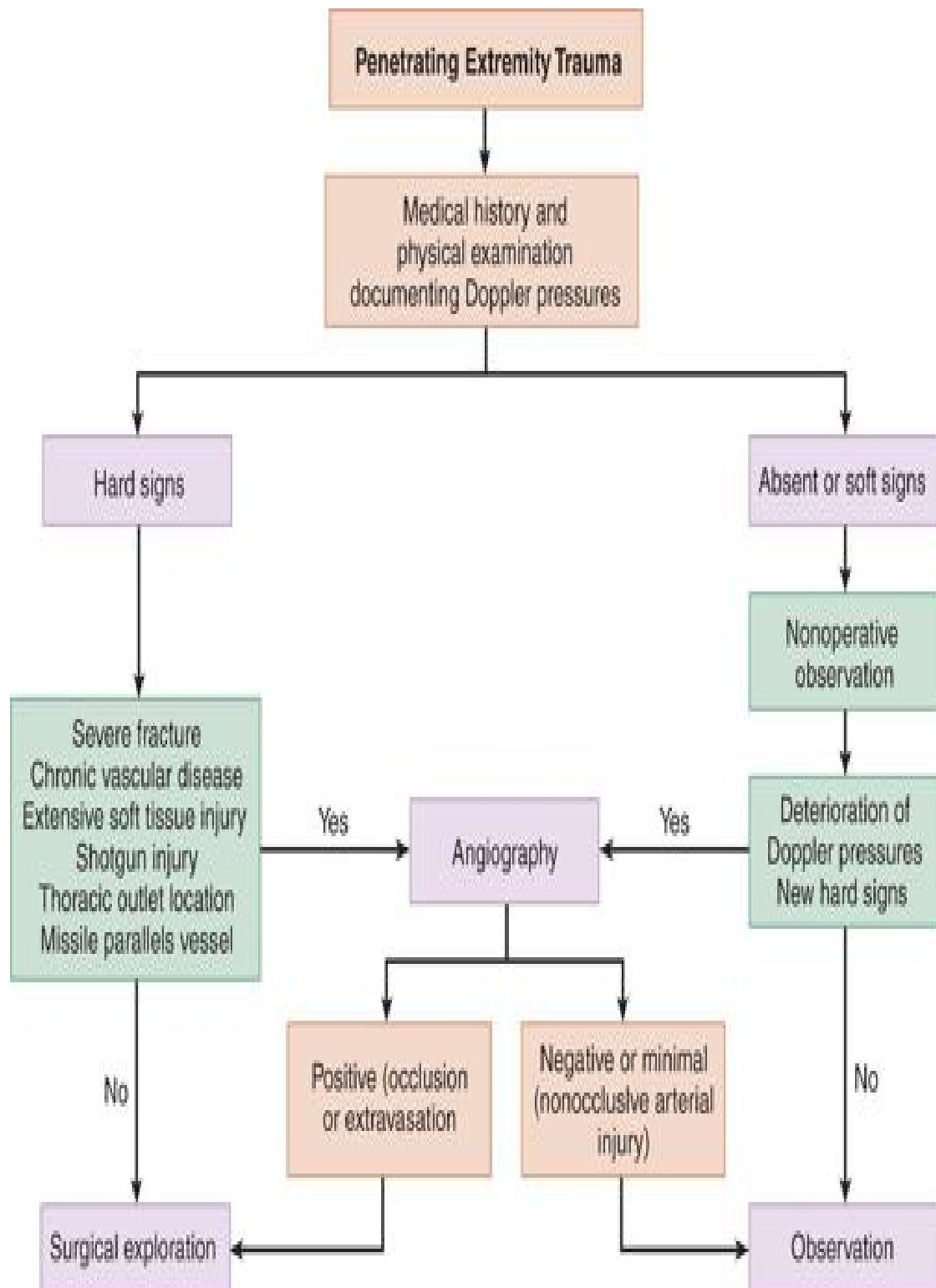
**TABLE 168-1** Clinical Manifestations of Extremity Vascular Trauma

Document the size and shape of each wound, as well as any bony deformities or soft tissue defects. Evaluate the surrounding area for pain with palpation or range of

motion. Carefully evaluate joints in the proximity of the wound for the possibility of an open joint. Perform detailed strength and sensory exams on the affected limb to check for peripheral nerve injury. Consult the appropriate surgical specialist for signs of injury to an artery, nerve, joint, or bone, or suspicion of compartment syndrome.

## **DIAGNOSIS AND DIFFERENTIAL**

Diagnosing significant injuries to the extremities requires a meticulous exam. Ankle-Brachial indices (ABIs) and imaging may also be indicated. The decision to obtain vascular imaging in the absence of hard signs of injury is controversial and should be made in conjunction with a careful history and examination ([Fig. 168-1](#)).



**FIGURE 168-1.** Algorithm for the evaluation of an injured extremity for vascular trauma.

Obtain ABIs on the affected and unaffected limb, though they have variable sensitivity and specificity for arterial injury and do not reliably detect injuries such as intimal flaps or pseudoaneurysms. Perform them in all 4 extremities with the patient supine, using the Doppler and a manual blood pressure cuff. The ankle systolic pressure is then divided by the greatest systolic pressure from the upper extremities. A result of 0.5 to 0.9 indicates injury to a single arterial segment, while a result of <0.5 indicates either severe arterial injury or injury to multiple segments of the artery.

Plain radiographs of the affected limb are necessary to evaluate for bone or joint injuries. Retained foreign bodies or embolized bullet fragments may also be seen. Image the joint above and below the injury site.

Conventional angiography has long been considered the gold standard for arterial injury and is still appropriate in some cases; however, multidetector CT (MDCT) angiography is also being used in many institutions now. MDCT is rapid and noninvasive, and provides high quality images that may also aid in the evaluation of bony injuries and foreign bodies. Ultrasound may also be useful for identifying vascular injuries and foreign bodies.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Immediate operative intervention is typically indicated when hard signs of vascular injury are identified. For some injuries, angiography (or MDCT) may be appropriate prior to surgery.
2. Management of patients with soft signs of injury is variable, but common practice is admission for 24 hours of observation with serial exams, reserving further imaging or operative intervention if clinical evidence of vascular injury develops. Obtain early surgical consultation for signs of a compartment syndrome.
3. Control bleeding with direct pressure. Vessels should not be clamped or ligated to avoid injury to adjacent nerves.
4. Bone or joint capsule injuries should be evaluated by an orthopedic surgeon as patients are at risk for infection, posttraumatic arthritis, and loss of function. Splint patients with injuries in close proximity to a joint without penetration of the capsule, and provide 24-hours orthopedic follow-up. Fractures due to penetrating injuries should be treated as open fractures, requiring surgical debridement and admission for **intravenous antibiotics** (cephalosporin +/- aminoglycoside).
5. Update tetanus and irrigate wounds copiously with saline or tap water. Closure of the wound depends on the time of presentation and amount of contamination. Repair low risk wounds and arrange follow-up in 24 hours. Consider delayed



primary closure after 72 to 96 hours for higher risk wounds. Antibiotics are generally not indicated for low risk injuries (if there is no fracture), but may be considered for high-risk cases such as hand injuries, gross contamination, or immunocompromised patients.

6. The decision to remove foreign bodies such as bullet fragments depends on the size, location, and composition of the object. As aggressive exploration may cause further tissue damage and increase infection risk, the risk/benefit ratio should be carefully considered.
7. Discharge patients with no signs of significant injury, minimal tissue damage, and no signs of developing compromise after an observation period and serial exams. Provide strict return precautions for worsening swelling, pain, numbness, or signs of infection.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 263, "Penetrating Trauma to the Extremities," by Roberta Capp and Richard D. Zane.

# **SECTION 19 Injuries to the Bones, Joints, and Soft Tissue**

## CHAPTER 169

# Initial Evaluation and Management of Orthopedic Injuries

Michael P. Kefer

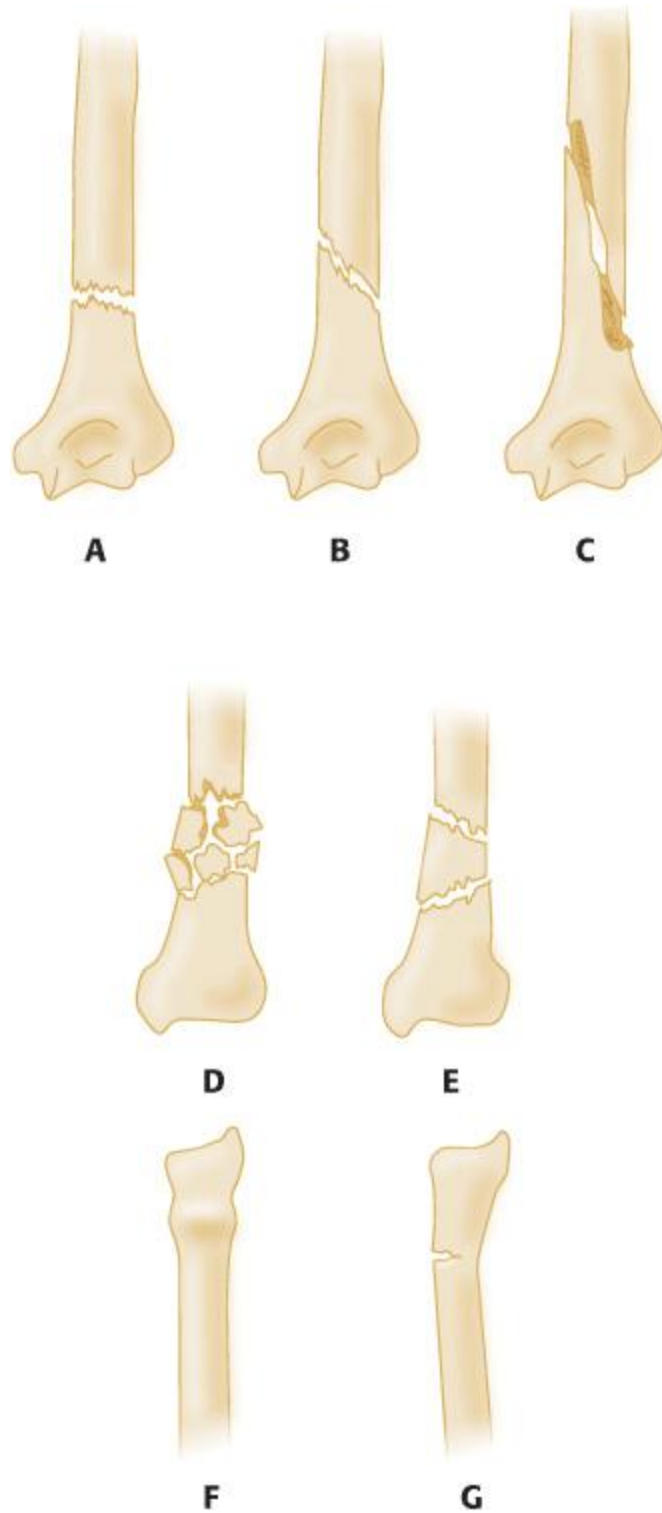
## CLINICAL FEATURES

Knowing the mechanism of injury and the patient's symptoms are important in diagnosing fracture or dislocation. Pain may be referred to an area distant from the injury (eg, hip injury presenting as knee pain). Careful palpation can prevent missing a crucial diagnosis due to referred pain. Neurovascular status distal to the injury also needs to be assessed.

Diagnostic imaging is based on the history and physical examination, not simply on where the patient reports pain. Radiographs of all long bone fractures should include the joint proximal and distal to the fracture to evaluate for coexistent injury. A negative radiograph does not exclude a fracture. This is common with scaphoid, radial head, and metatarsal shaft fractures. In this case, the diagnosis is often clinical and may not be confirmed until 7 to 10 days after the injury, when enough bone resorption has occurred at the fracture site to detect a lucency on the radiograph.

Include the following details for an accurate description of the fracture to the orthopedic consultant:

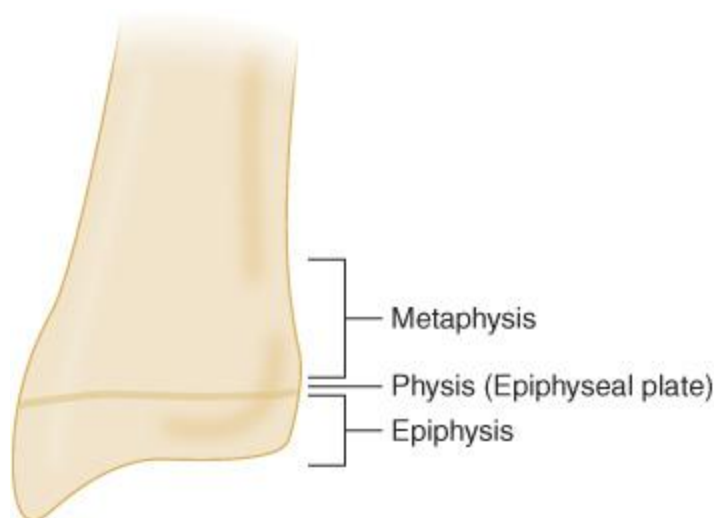
- Closed versus open: whether overlying skin is intact (closed) or not (open).
- Location: midshaft, junction of proximal and middle or middle and distal thirds, or distance from the bone end, or intraarticular. Anatomic bony reference points should be used when applicable. For example, a humerus fracture just above the condyles is described as supracondylar, as opposed to distal humerus.
- Orientation of fracture line (see [Fig. 169-1](#)).



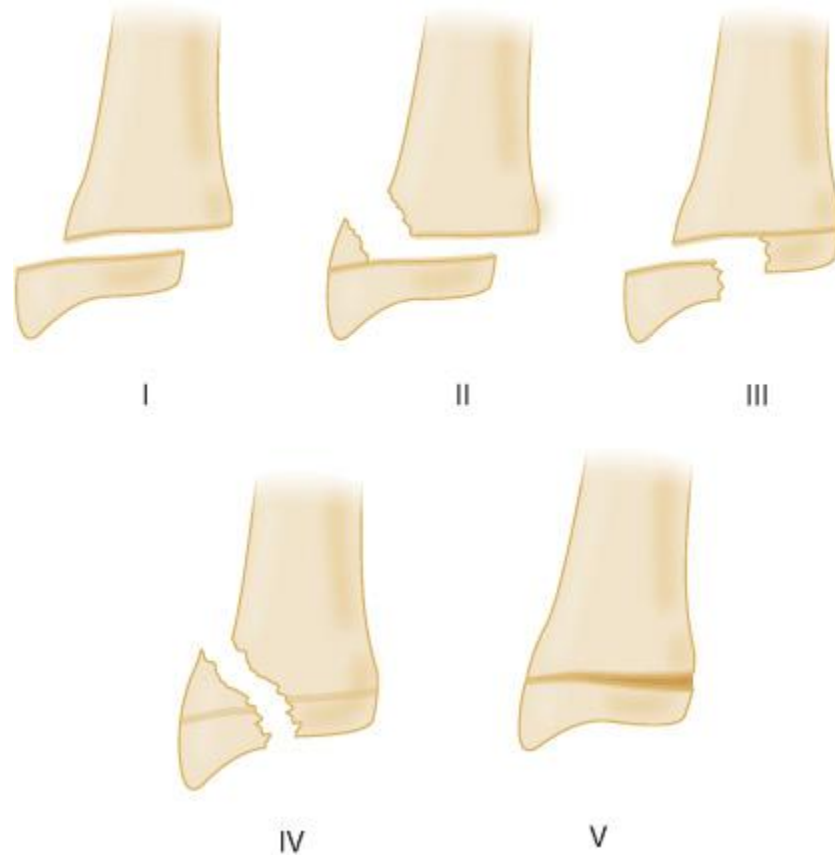
**FIGURE 169-1.** Fracture line orientation. A. Transverse. B. Oblique. C. Spiral. D. Comminuted. E. Segmental. F. Torus. G. Greenstick.

- Displacement: amount and direction distal fragment is offset from proximal fragment.
- Separation: amount 2 fragments have been pulled apart; unlike displacement, alignment is maintained.
- Shortening: reduction in bone length due to impaction or overriding fragments.
- Angulation: degree and direction of the angle formed by the distal fragment.
- Rotational deformity: degree distal fragment is twisted on the axis of normal bone; usually detected by physical examination and not seen on the radiograph.
- Associated disruption of proper joint alignment is described as fracture-dislocation (joint surfaces have no contact) or fracture-subluxation (joint surfaces still in partial contact).

Fractures involving the growth plate of long bones in pediatric patients are described by the Salter-Harris classification ([Figs. 169-2, 169-3](#), and [Table 169-1](#)). Note Type I and V may be radiographically undetectable.



**FIGURE 169-2.** Epiphyseal anatomy in the growing child.



**FIGURE 169-3.** Epiphyseal plate fractures based on the classification of Salter and Harris.

Salter Type	What Is Broken Off
I	The entire epiphysis.
II	The entire epiphysis <i>along with</i> a portion of the metaphysis.
III	A portion of the epiphysis.
IV	A portion of the epiphysis <i>along with</i> a portion of the metaphysis.
V	Compression injury of the epiphyseal plate. Nothing is "broken off."

**TABLE 169-1** Description of Salter-Harris Fractures

Complications resulting from neurovascular deficit may be immediate or delayed. Compartment syndrome that presents with the 5 classic signs—pain, pallor, paresthesias, pulselessness, and paralysis—is well advanced.

Long-term complications of fracture include malunion, nonunion, avascular necrosis, arthritis, and osteomyelitis.

# EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. Control swelling with cold packs and elevation. Provide pain control. Remove objects, such as rings or watches, which may constrict the injury before swelling progresses.
2. Reduction of fracture deformity with steady, longitudinal traction is indicated to: (a) relieve pain; (b) relieve tension on associated neurovascular structures; (c) minimize the risk of converting a closed fracture to an open fracture when a sharp, bony fragment tents overlying skin; and (d) restore circulation to a pulseless distal extremity. Obtain postreduction radiographs to confirm proper repositioning.
3. Open fractures require immediate prophylactic antibiotics, irrigation, and debridement to prevent osteomyelitis. A common antibiotic regimen is a first-generation cephalosporin (along with an aminoglycoside for contaminated wounds).
4. Immobilize the fracture or relocated joint. Fiberglass or plaster splinting material sets by an exothermic reaction. The amount of heat liberated is directly proportional to the temperature of the water. To avoid burns, use water slightly warmer than room temperature. Splints should be long enough to immobilize the joint above and below the fracture.
5. Discharge instructions should emphasize elevating the injury above heart level and to seek immediate reevaluation if increased swelling, cyanosis, pain, or decreased sensation develops.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 264, “Initial Evaluation and Management of Orthopedic Injuries,” by Jeffrey S. Menkes.

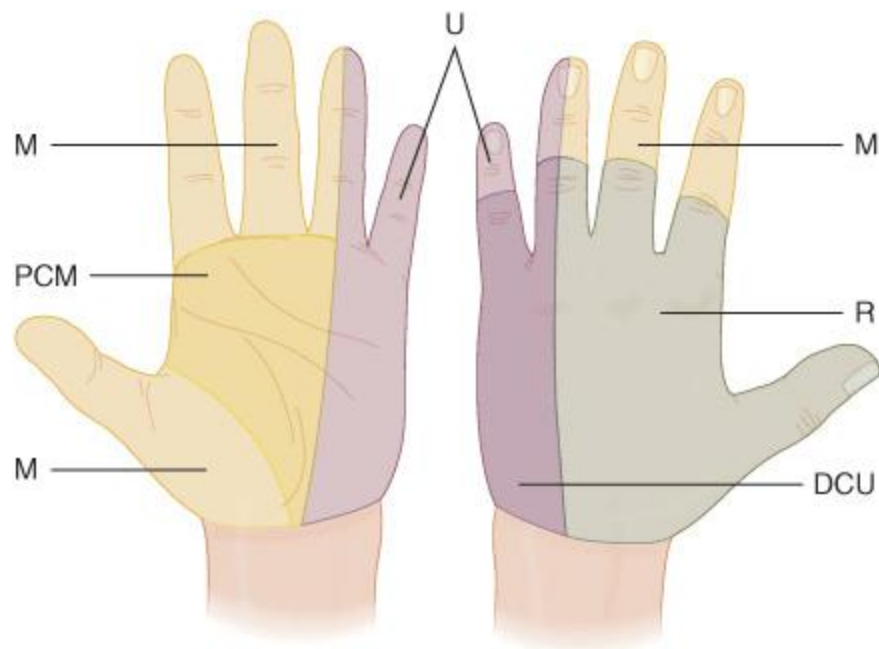
# CHAPTER 170

## Hand and Wrist Injuries

Michael P. Kefer

### HAND INJURIES

The hand is innervated by the median, ulnar, and radial nerves. Motor function of the median nerve can be screened by flexing the thumb distal phalanx against resistance, the ulnar nerve by spreading the fingers against resistance, and the radial nerve by maintaining extension of the wrist and fingers against resistance. Sensory innervation ([Fig. 170-1](#)) is best screened by the presence of normal 2-point discrimination (<6 mm). Injuries requiring hand surgery consultation are listed in [Tables 170-1](#) and [170-2](#).



**FIGURE 170-1.** The cutaneous nerve supply in the hand.

*Key:* DCU = dorsal branch of ulnar nerve; M = median; PCM = palmar branch of median nerve; R = radial; U = ulnar.



Vascular injury with signs of tissue ischemia or poorly controlled hemorrhage Irreducible dislocations Grossly contaminated wounds Severe crush injury Open fracture Compartment syndrome High pressure injection injury Hand/finger amputation
--

**TABLE 170-1** Immediate Hand Surgery Consultation Guidelines

Extensor/flexor tendon laceration (if not repaired in ED) Flexor digitorum profundus rupture (closed) Nerve injury (proximal to mid middle phalanx) Closed fractures Dislocations Ligamentous injuries with instability
--

**TABLE 170-2** Delayed Hand Surgery Consultation Guidelines

**Tendon injuries** can be missed if one does not know and examines the hand in the position it was in at the time of injury. Up to 90% of a tendon can be lacerated with preserved range of motion without resistance, so test function against resistance. Pain along the course of the tendon suggests a partial laceration even if strength is normal. Extensor tendon repair can often be performed by the emergency physician. Flexor tendon repair should be performed by the hand surgeon. It is common for the ED care of tendon lacerations to consist of closing the skin and splinting until definitive repair by the hand surgeon. Follow-up and rehabilitation of all tendon injuries are necessary, even those not requiring repair.

**Mallet finger** results when complete rupture of the extensor tendon occurs at the level of the distal phalanx. On examination, the distal interphalangeal (DIP) joint is flexed at 40°. Splint the DIP joint in slight hyperextension.

**Boutonniere deformity** results from an injury at the dorsal surface of the proximal interphalangeal (PIP) joint that disrupts the extensor hood apparatus. Lateral bands of the extensor mechanism become flexors of the PIP joint and hyperextensors of the DIP joint. Splint the PIP joint in extension.

**DIP joint dislocations** are uncommon because of the firm attachment of skin and fibrous tissue to underlying bone. Dislocations are usually dorsal. Reduction is performed under digital block anesthesia. The dislocated phalanx is distracted, slightly hyperextended, then repositioned. Splint the joint in full extension. An irreducible joint may be from an entrapped volar plate, profundus tendon, or avulsion fracture.

**PIP joint dislocations** are usually dorsal with rupture of the volar plate. Closed reduction is as described above for the DIP joint. Splint the joint in 30° flexion. Lateral dislocation results from rupture of one of the collateral ligaments. An irreducible joint from an entrapped volar plate or complete ligamentous disruption may require surgical intervention.

**Metacarpal phalangeal (MCP) joint dislocations** are usually dorsal and require surgical reduction due to volar plate entrapment. Attempt closed reduction with the wrist flexed and pressure applied to the proximal phalanx in a distal and volar direction. Splint with the MCP joint flexed 70° to 90°.

**Thumb IP joint dislocations** usually involve volar plate rupture. Closed reduction is as described above for the DIP joint. Place in a thumb spica splint.

**Thumb MCP dislocations** are usually dorsal and involve volar plate rupture. Reduce by flexing and abducting the metacarpal and apply pressure directed distally to the base of the proximal phalanx. Place in a thumb spica splint.

**Thumb MCP ulnar collateral ligament rupture** (gamekeeper or skier thumb) results from forced radial abduction at the MCP joint. This is the most critical of the collateral ligament injuries since it affects pincer function. The joint capsule and volar plate are usually involved. A complete tear is diagnosed when abduction stress on the proximal phalanx causes more than 40° of radial angulation relative to the metacarpal. Place in a thumb spica splint.

**Distal phalanx fractures** most commonly involve the tuft. These are associated with subungual hematoma and nail bed laceration. Place a volar or hairpin splint to the DIP joint. Dorsal avulsion fracture of the base may result in mallet finger.

**Proximal and middle phalanx fractures** of the base and neck that are nondisplaced and stable can be treated with buddy taping. Transverse or spiral midshaft fractures or intraarticular fractures often require surgical

fixation. Place a gutter splint with the MCP joint flexed at 90°, the PIP joint flexed at 20°, and the DIP joint flexed at 10°.

**Metacarpal (MC) fractures** that involve the fourth or fifth MC neck (boxer fracture) are the most common type. Angulation more than 20° in the fourth MC, 40° in the fifth MC, or 15° in the second or third MC should be reduced. Place an ulnar gutter splint for fractures of the fourth or fifth MC and a radial gutter splint for fractures of the second or third MC with the wrist extended at 20° and the MCP joint flexed at 90°. Thumb MC fractures usually involve the base with intraarticular involvement (Bennett and Rolando fractures). Place in a thumb spica splint.

**Compartment syndrome** of the hand may result from crush injury or extravasation of IV fluids. The patient will complain of pain that is out of proportion to exam findings. On examination, the hand, at a resting position, is extended at the MCP joint and slightly flexed at the PIP joint. There is tense edema and pain with passive stretch of the involved compartment. This is an orthopedic emergency.

**High pressure injection injury** occurs when substances in a high pressure device, such as grease, paint, or hydraulic fluid, are injected into the hand. Oil-based paint causes the most severe tissue reaction that can result in ischemia and amputation. Obtain hand and forearm radiographs searching for radiopaque substances and subcutaneous air. This is an orthopedic emergency.

## WRIST INJURIES

**Scapholunate dissociation** presents with wrist tenderness and swelling at the scapholunate joint. The PA radiograph demonstrates a space between the scaphoid and lunate that is > 3 mm. Treat with a radial gutter splint and prompt referral.

**Perilunate and lunate dislocations** are best noted on lateral wrist radiograph. In both injuries, the normal alignment of the radius-lunatecapitate (the “3 C’s” sign) is lost. With a perilunate dislocation, the lunate remains aligned with the radius, but the capitate is dislocated, usually dorsal to the lunate. With a lunate dislocation, the lunate dislocates volar to

the radius, but the remainder of the carpus aligns with the radius. Lunate dislocation on PA radiograph has a triangular shape, the “piece of pie” sign, and on lateral view, the “spilled teacup” sign. Emergent consult for closed reduction or surgical repair is indicated.

**Carpal bone fractures** are managed as summarized in [Table 170-3](#). The scaphoid is the most common carpal bone fractured. Fracture of the scaphoid, lunate, or capitate can cause avascular necrosis of the bone. Scaphoid and lunate fractures are often not detected on plain radiographs, so ED diagnosis and treatment should be based on clinical findings alone.

Carpal Bone	Mechanism of Injury	Examination	Initial ED Management
Scaphoid	Fall on outstretched hand	Snuffbox tenderness. Pain with radial deviation and flexion	Short arm, thumb spica, in dorsiflexion with radial deviation
Triquetrum	Avulsion fracture—twisting of hand against resistance or hyperextension Body fracture—direct trauma	Tenderness at the dorsum of the wrist, distal to the ulnar styloid	Short arm, sugar tong splint
Lunate	Fall on outstretched hand	Tenderness at shallow indentation of the mid-dorsum of the wrist, ulnar and distal to Lister tubercle	Short arm, thumb spica splint
Trapezium	Direct blow to thumb; force to wrist while dorsiflexed and radially deviated	Painful thumb movement and weak pinch strength Snuffbox tenderness	Short arm thumb spica splint
Pisiform	Fall directed on the hypothenar eminence	Tender pisiform, prominent at the base of the hypothenar eminence	Short arm, volar splint in 30° of flexion and ulnar deviation
Hamate	Interrupted swing of a golf club, bat, or racquet	Tenderness at the hook of the hamate, just distal and radial to the pisiform	Short arm, volar wrist splint with fourth and fifth metacarpal joints in flexion
Capitate	Forceful dorsiflexion of the hand with radial impact	Tenderness over the capitate just proximal to the third metacarpal	Short arm, volar wrist splint
Trapezoid	Axial load onto the index metacarpal	Tenderness over the radial aspect of the base of the index metacarpal	Short arm thumb spica splint

**TABLE 170-3** Summary of Carpal Bone Fractures and ED Management

**Colles, Smith, and Barton fractures** involve the distal radius at the metaphysis ([Table 170-4](#)). Most of these fractures can be treated with closed reduction and a sugar tong splint.

Colles fracture
Dorsal angulation of the plane of the distal radius
Distal radius fragment is displaced proximally and dorsally
Radial displacement of the carpus
Ulnar styloid may be fractured
Smith fracture
Volar angulation of the plane of the distal radius
Distal radius fragment is displaced proximally and volarly
Radial displacement of the carpus
The fracture line extends obliquely from the dorsal surface to the volar surface 1 to 2 cm proximal to the articular surface
Barton fracture
Volar and proximal displacement of a large fragment of radial articular surface
Volar displacement of the carpus
Radial styloid may be fractured

**TABLE 170-4** Radiographic Appearance of Distal Radius Fractures

**Radial styloid fracture** can produce carpal instability with scapholunate dissociation as major carpal ligaments insert here. Splint the wrist in mild flexion and ulnar deviation.

**Ulnar styloid fracture** may result in radioulnar joint instability. Place an ulnar gutter splint with the wrist in neutral position and slight ulnar deviation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 265, “Injuries to the Hand and Digits,” by Moira Davenport and Dean G. Sotereanos; and Chapter 266, “Wrist Injuries,” by Robert Escarza, Maurice F. Loeffel III, and Dennis T. Uehara.

# **CHAPTER 171**

## **Forearm and Elbow Injuries**

Sandra L. Najarian

### **BICEPS AND TRICEPS TENDON RUPTURES**

#### **Clinical Features**

Patients with proximal long-head biceps tendon ruptures typically describe a “snap” or “pop” and complain of pain in the anterior shoulder. Examination reveals tenderness, swelling, and crepitus over the bicipital groove in the anterior shoulder. A midarm “ball” (the distally retracted biceps) appears when the elbow is flexed. Elbow flexion strength is maintained due to the preserved action of the brachialis and supinators. This is in contrast to distal bicep tendon rupture where elbow flexion and supination is weak. Examination of distal biceps rupture reveals swelling, ecchymosis, tenderness, and inability to palpate the tendon in the antecubital fossa. With the patient seated and the elbow flexed and forearm resting on the patient’s lap, the examiner squeezes the muscle belly of the biceps causing the forearm to supinate (biceps squeeze test). If no supination is noted, then this is a positive test indicating a distal biceps tendon rupture. Patients with triceps tendon ruptures present with pain, swelling, and tenderness proximal to the olecranon; a sulcus with a proximal mass (the proximally retracted triceps tendon) may be palpable. Forearm extension is weak. A modified Thompson test can be used to assess triceps function. With the arm supported, elbow flexed at 90°, and forearm hanging in a relaxed position, squeezing the triceps muscle should produce extension of the forearm unless a complete tear is present.

#### **Diagnosis and Differential**

Diagnosis is clinical. Obtain radiographs to exclude an associated avulsion fracture.

## Emergency Department Care and Disposition

Treatment includes sling, ice, analgesics, and referral to an orthopedic surgeon for definitive management. Complete tendon tears in young active individuals often require surgical repair.

## ELBOW DISLOCATIONS

### Clinical Features

The majority of elbow dislocations are posterolateral. On examination, the patient holds the elbow in 45° flexion. Significant swelling of the elbow often obscures the olecranon, which is directed posteriorly. Neurovascular assessment is essential ([Table 171-1](#)). An open dislocation, absence of radial pulse before reduction, and presence of systemic injuries are all factors associated with arterial injury.

	Radial	Median	Ulnar
Test for sensory function	Dorsum of the thumb index web space	Two-point discrimination over the tip of the index finger	Two-point discrimination over the little finger
Test for motor function	Extend both wrist and fingers against resistance	"OK" sign with thumb and index finger; abduction of the thumb (recurrent branch)	Abduct index finger against resistance

**TABLE 171-1** Sensory and Motor Function Testing of the Radial, Median, and Ulnar Nerves

### Diagnosis and Differential

Radiographs confirm the diagnosis. The lateral view reveals both the ulna and radius displaced posterior. The AP view reveals either medial or lateral displacement of the ulna and radius with maintenance of their normal relationship to each other. The presence of associated fractures, especially to the radial head and coronoid process, can render the elbow joint unstable and complicate treatment.

## **Emergency Department Care and Disposition**

The goals of treatment are reduction with procedural sedation and recognition of neurovascular complications, associated fractures, and postreduction instability. Closed reduction of the elbow is achieved by placing gentle longitudinal traction on the wrist and forearm while an assistant applies countertraction on the arm. Any medial or lateral displacement is corrected with the other hand. A palpable clunk indicates a successful reduction. Range the elbow fully to assess stability. Assess neurovascular status after reduction and for a period of observation. Obtain postreduction films. Immobilize the elbow in a long-arm posterior splint with the elbow in slightly less than 90° of flexion, and arrange for close orthopedic follow-up. Immediate orthopedic consultation is necessary for patients with instability in extension, neurovascular compromise, or open dislocations.

## **ELBOW FRACTURES**

### **Clinical Features**

Radial head fractures present with pain, swelling, and tenderness over the lateral elbow, and inability to fully extend the elbow. Supracondylar and intercondylar fractures present with significant swelling, tenderness, and limited range of motion at the elbow. Supracondylar fractures may resemble a posterior elbow dislocation. Olecranon fractures present with pain,



swelling, and crepitus over the posterior elbow. A neurovascular assessment is essential in all elbow fractures. Potential complications of supracondylar fractures are numerous ([Table 171-2](#)). A decreased or absent radial pulse is common in children and often secondary to transient vasospasm. Signs of Volkmann ischemic contracture include refusal to open the hand, pain with passive extension of the fingers, and forearm tenderness.

Early complications	Neurologic Radial nerve Median nerve (anterior interosseous branch) Ulnar Vascular Volkmann ischemic contracture (compartment syndrome of the forearm)
Late complications	Nonunion Malunion Myositis ossificans Loss of motion

**TABLE 171-2** Complications of Supracondylar Fractures Early complications

## Diagnosis and Differential

Fracture lines may not be visible on standard AP and lateral radiographs of the elbow. Abnormal fat pads, posterior fat pad sign, or prominent anterior fat pad (“sail sign”) may be the only evidence of injury. Disruption of the radiocapitellar line may be another clue to injury. A line drawn from the center of the radial shaft should transect the radial head and capitellum in all views.

## Emergency Department Care and Disposition

Immobilization in a splint and orthopedic referral are appropriate for non-displaced fractures. Treat nondisplaced radial head fractures with sling immobilization. Immediate orthopedic consultation is warranted for all displaced fractures, open fractures, and evidence of neurovascular

compromise. Admit patients with significant swelling and displaced fractures for observation of neurovascular status.

## **FOREARM FRACTURES**

### **Clinical Features**

Both bone fractures present with swelling, tenderness, and deformity of the forearm. Isolated ulna or radius fractures present with localized swelling and tenderness. Monteggia fracture-dislocation, a fracture of the proximal third of the ulna with a radial head dislocation, presents with significant pain and swelling over the elbow. Galeazzi fracture-dislocation, a fracture of the distal radius with an associated distal radioulnar joint dislocation, presents with localized tenderness and swelling over the distal radius and wrist.

### **Diagnosis and Differential**

AP and lateral radiographs confirm the diagnosis. In a Monteggia fracture, the radiocapitellar line is disrupted, and the apex of the ulna fracture points in the direction of the radial head dislocation. In a Galeazzi fracture, the distal radioulnar joint space is widened on the AP view, and the ulna is displaced dorsally on the lateral view.

### **Emergency Department Care and Disposition**

Treat nondisplaced fractures with long-arm splint immobilization and referral to orthopedics. Immediate orthopedic consultation is necessary for all displaced fractures. Closed reduction is often adequate for both bone fractures in children. Open reduction and internal fixation is usually required for displaced fractures in adults and for Monteggia and Galeazzi fracture-dislocations.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 267, “Injuries to the Elbow and Forearm,” by Jason H. Bredenkamp, Brian P. Jokhy, and Dennis T. Uehara.

## **CHAPTER 172**

# **Shoulder and Humerus Injuries**

Sandra L. Najarian

## **STERNOCLAVICULAR           SPRAINS           AND DISLOCATIONS**

### **Clinical Features**

Patients with simple sprains have pain and tenderness localized to the joint, whereas patients with dislocations have severe pain, which is exacerbated by arm motion and lying supine. In anterior dislocations, the medial clavicle is visibly prominent and palpable anterior to the sternum. In posterior dislocations, the medial clavicle is less visible and often not palpable. Symptoms of hoarseness, dysphagia, dyspnea, upper extremity paresthesias, or weakness may indicate life-threatening injuries to mediastinal contents, such as pneumothorax or compression or laceration of surrounding great vessels, trachea, and esophagus.

### **Diagnosis and Differential**

CT is the imaging test of choice. IV contrast may be needed to detect injury to adjacent mediastinal structures. Consider septic arthritis in the nontraumatic patient, especially in injection drug users.

### **Emergency Department Care and Disposition**

Treatment for sternoclavicular sprains and uncomplicated anterior dislocations includes ice, analgesics, and sling immobilization. Attempted

closed reduction is not necessary as this injury is often unstable. Posterior dislocations require immediate orthopedic consultation for open reduction and internal fixation.

## **CLAVICLE AND SCAPULA FRACTURES**

### **Clinical Features**

Patients with clavicle fractures present with pain, swelling, and tenderness over the clavicle. The scapula is a well-protected bone; therefore, fractures usually occur in association with injuries to the ipsilateral lung, thoracic cage, and shoulder girdle. Patients have pain and localized tenderness over the scapula, hold their arm in adduction, and resist any arm movement.

### **Diagnosis and Differential**

Routine radiographs may miss some clavicle and scapular fractures. CT can confirm the diagnosis as well as identify any associated pathology.

### **Emergency Department and Disposition**

The majority of fractures can be managed conservatively with sling immobilization, ice, and analgesics. Early range-of-motion exercises are important for scapular fractures. Orthopedic consultation is warranted for clavicle fractures that are open, have neurovascular compromise, or have persistent skin tenting. Presence of a scapula fracture mandates investigation for associated intrathoracic injuries. Displaced glenoid articular fractures, angulated glenoid neck fractures, and certain acromial and coracoid fractures may require surgical intervention.

## **ACROMIOCLAVICULAR JOINT INJURIES**

## Clinical Features

Acromioclavicular joint injuries range from mild sprain to complete disruption of all ligaments that attach the scapula and clavicle. The classification of these injuries and their physical findings are described in [Table 172-1](#).

Type	Injury	Radiograph	Examination
I	Sprained acromioclavicular ligaments	Normal	Tenderness over acromioclavicular joint
II	Acromioclavicular ligaments ruptured; coracoclavicular ligaments sprained	Slight widening of acromioclavicular joint; clavicle elevated 25% to 50% above acromion; may be slight widening of the coracoclavicular interspace	Tenderness and mild step-off deformity of acromioclavicular joint
III	Acromioclavicular ligaments ruptured; coracoclavicular ligaments ruptured; deltoid and trapezius muscles detached	Acromioclavicular joint dislocated 100%; coracoclavicular interspace widened 25% to 100%	Distal end of clavicle prominent; shoulder droops
IV	Rupture of all supporting structures; clavicle displaced posteriorly in or through the trapezius	May appear similar to type II and III; axillary radiograph required to visualize posterior dislocation	Possible posterior displacement of clavicle
V	Rupture of all supporting structures (more severe form of type III injury)	Acromioclavicular joint dislocated; generally 200% to 300% disparity of coracoclavicular interspace compared to normal shoulder	More pain; gross deformity of clavicle
VI	Acromioclavicular ligaments disrupted; coracoclavicular ligaments may be disrupted; deltoid and trapezius muscles disrupted	Acromioclavicular joint dislocated; clavicle displaced inferiorly	Severe swelling; multiple associated injuries

**TABLE 172-1** Classification and Physical Findings in Acromioclavicular Joint Injuries

## Diagnosis and Differential

Acromioclavicular radiographs can help determine the severity of the injury and identify any associated fractures.

### **Emergency Department Care and Disposition**

Treatment for type I and type II injuries includes sling immobilization, rest, ice, and analgesics. Early range-of-motion exercises are recommended at 7 to 14 days post injury. Treatment for type III is controversial, but the trend favors conservative management with sling immobilization rather than operative management. Treatment for type IV through VI is operative.

## **GLENOHUMERAL JOINT DISLOCATION**

### **Clinical Features**

[Table 172-2](#) describes the various mechanisms of injury, physical findings, and associated injuries with each type of glenohumeral joint dislocation.

Type	Description/Mechanism of Injury	Associated Injuries
Anterior	<p>Patient presentation: Arm is held in abduction and slight external rotation with shoulder appearing "squared off."</p> <p>Mechanism of injury: Indirect blow with arm in abduction, extension, and external rotation.</p>	<p>Axillary nerve palsy Fracture of the greater tuberosity</p> <p>Fracture of the humeral neck Disruption of the glenoid rim (Bankart lesion)</p>
Subcoracoid	Humeral head is displaced anterior to the glenoid and inferior to the coracoid.	Axillary artery disruption
Subglenoid	Humeral head lies inferior and anterior to the glenoid fossa.	
Subclavicular	Humeral head is displaced medial to the coracoid below the clavicle.	
Intrathoracic	Humeral head lies between the ribs and thoracic cavity.	
Posterior	Patient presentation:	Fractures of the posterior glenoid rim
Subacromial	Arm is adducted and internally rotated.	Fractures of the humeral head (reversed fractures of the Hill-Sachs deformity)
Subglenoid	Anterior shoulder is flat and the posterior aspect full.	
Subspinous	Coracoid process is prominent.	Fractures of the humeral shaft
	Patient will not allow external rotation or abduction because of severe pain.	Fractures of the lesser tuberosity
	Mechanism of injury: Indirect force that produces forceful internal rotation and adduction.	
Inferior (luxatio erecta)	<p>Patient presentation: Patient is in severe pain. Humerus is fully abducted. The elbow is flexed. Patient's hand is on or behind the head. Humeral head can be palpated on the lateral chest wall.</p> <p>Mechanism of injury: Neck of the humerus is levered against the acromion and inferior capsule tears. Humeral head is forced out inferiorly.</p>	<p>Severe soft tissue injuries Fractures of the proximal humerus Rotator cuff tear Neurovascular compression injuries</p>

**TABLE 172-2** Classification and Physical Findings in Dislocations of the Glenohumeral Joint



## Diagnosis and Differential

AP and scapular “Y” view radiographs confirm the type of dislocation and identify any associated fractures. The presence of minor fractures, such as a Hill-Sachs lesion or Bankart fracture, does not change ED management. Consider omitting prerduction radiographs in patients with a history of recurrent shoulder dislocation who present with signs and symptoms of a recurrence in the absence of trauma.

## Emergency Department Care and Disposition

Reduction techniques include traction, leverage, and scapular manipulation.

1. **Modified hippocratic technique.** This method uses traction and countertraction. Place the patient supine with their arm abducted and the elbow flexed 90°. A sheet is placed across the thorax of the patient and tied around the waist of the assistant. Another sheet is placed around the patient’s flexed elbow and the clinician’s waist. Gradually apply traction while an assistant provides countertraction. Gentle internal and external rotation may aid reduction.
2. **Milch technique.** With the patient supine, slowly abduct and externally rotate the arm to the overhead position. Apply gentle traction with the elbow fully extended. If reduction is not achieved, attempt to manipulate the humeral head into the glenoid fossa with the free hand.
3. **Scapular manipulation technique.** Scapular manipulation accomplishes reduction by repositioning the glenoid fossa rather than the humeral head. The first step is to apply traction to the patient’s arm held in 90° of forward flexion. This can be accomplished in the prone position or in a seated position with an assistant applying traction. Position the arm in slight external rotation. Push the scapular tip as far medially as possible while stabilizing the superior aspect of the scapula with the other hand. A small amount of dorsal displacement of the scapula tip is recommended.
4. **External rotation technique.** Place the patient supine with the arm adducted to the side. With the patient’s elbow flexed to 90°, slowly and

gently externally rotate the arm. No traction is applied. Reduction is subtle and usually occurs before reaching the coronal plane.

5. **Aronen technique.** This technique is most useful when reduction is simple to achieve, as in recurrent dislocations or immediately after injury before muscle spasm and swelling have occurred. This technique can be taught to patients with recurrent dislocations, as self-reduction may be the only method available to them in certain situations (eg, solo sailing, crosscountry skiing, etc). In this technique, the patient is seated on a gurney with the ipsilateral leg and knee in flexion. Patients are instructed to clasp their hands around the ipsilateral knee and then relax the shoulder muscles, thereby allowing the weight of the lower limb to provide gentle inline traction. Countertraction is applied by the patients' upper body weight and their own paraspinous muscles. Taping the clasped hands together can aid reduction.

Procedural sedation is recommended; however, an intraarticular injection of 10 to 20 mL of 1% lidocaine can facilitate reduction and may obviate the need for sedation. Once reduced, assess neurovascular status and provide sling immobilization. Postreduction radiographs are useful for confirmation and documentation of successful reduction. Urgent orthopedic follow-up is necessary. Some studies show that immobilization in adduction and external rotation reduces recurrent dislocation. Early operative repair may decrease incidence of recurrence as well.

## **HUMERUS FRACTURES**

### **Clinical Features**

Patients with proximal humeral fractures have pain, swelling, tenderness, ecchymosis, and crepitus about the shoulder. Range of motion is severely limited; patients hold their arm closely against the chest wall. Patients with humeral shaft fractures present with pain, swelling, localized tenderness, limited mobility, and crepitus on palpation. Shortening of the arm can be seen in displaced fractures. A careful neurovascular exam is essential. Injuries to the axillary nerve and artery are common in proximal humerus

fractures. The radial nerve is most frequently injured in humeral shaft fractures.

## **Diagnosis and Differential**

Radiographs confirm the diagnosis. The Neer classification system divides the proximal humerus into four parts (articular surface of the humeral head, greater tubercle, lesser tubercle, and diaphysis of the humerus) and is used to guide treatment.

## **Emergency Department Care and Disposition**

Proximal humerus fractures that are nondisplaced or one-part fractures (displaced  $<1$  cm or angulated  $<45^\circ$ ) require sling immobilization, ice, analgesics, and orthopedic referral. Humeral shaft fractures that are nondisplaced require a coaptation splint (sugar tong), hanging cast, or functional bracing. Multipart proximal humeral fractures, significantly displaced or angulated shaft fractures, open fractures, or any fracture with neurovascular injuries require immediate orthopedic consultation.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 268, “Shoulder and Humerus Injuries,” by John P. Rudzinski, Laura M. Pittman, and Dennis T. Uehara.

## **CHAPTER 173**

# **Pelvis, Hip, and Femur Injuries**

Jeffrey G. Norvell

## **PELVIC INJURIES**

### **Clinical Features**

Signs and symptoms of pelvic injuries vary from local pain and tenderness to pelvic instability and severe shock. Examine the patient for pain, pelvic instability, deformities, lacerations, ecchymoses, and hematomas. Avoid excessive movement of unstable fractures as this could produce further injury and cause additional blood loss. Rectal examination may reveal displacement of prostate or rectal injury. Blood at the urethral meatus suggests urethral injury. A vaginal speculum examination may be indicated to detect lacerations that would suggest an open fracture. If a pelvic fracture is found, assume associated intraabdominal, retroperitoneal, gynecologic, or urologic injuries exist until proven otherwise.

### **Diagnosis and Differential**

In patients with a suspected pelvic fracture, obtain a standard anteroposterior (AP) pelvis radiograph to evaluate for bony injury. Other radiographic views include lateral views, AP views of hemipelvis, internal and external oblique views of the hemipelvis, or inlet and outlet views of the pelvis. In an unstable blunt trauma patient, use an AP pelvic radiograph to identify a pelvic fracture quickly, allowing for emergent stabilization maneuvers. Routine pelvic radiographs are not needed in stable trauma patients who will undergo an emergent CT of the abdomen and pelvis. CT

is superior to pelvic radiographs for identifying pelvic fractures and evaluating pelvic ring instability.

Pelvic fractures include those that involve a break in the pelvic ring, fractures of a single bone without a break in the pelvic ring, and acetabular fractures. Single bone fractures are described in [Table 173-1](#).

Fracture	Description/ Mechanism of Injury	Treatment	Disposition and Follow-Up
Iliac wing (Duverney) fracture	Direct trauma, usually lateral to medial	Analgesics, nonweightbearing until hip abductors pain-free, usually nonoperative	Discharge with orthopedic follow-up in 1 to 2 weeks; admit for open fracture or concerning abdominal examination
Single ramus of pubis or ischium	Fall or direct trauma in elderly; exercise-induced stress fracture in young or in pregnant women	Analgesics, crutches	Discharge with PCP or orthopedic follow-up in 1 to 2 weeks
Ischium body	External trauma or from fall in sitting position; least common pelvic fracture	Analgesics, bed rest, donut-ring cushion, crutches	Discharge with orthopedic follow-up in 1 to 2 weeks
Sacral fracture	Transverse fractures from direct anteroposterior trauma; upper transverse fractures from fall in flexed position	Analgesics, bed rest, surgery may be needed for displaced fractures or neurologic injury	Discharge with orthopedic follow-up 1 to 2 weeks; orthopedic consultation for displaced fractures or neurologic deficits

Coccyx fracture	Fall in sitting position; more common in women	Analgesics, bed rest, stool softeners, sitz baths, donut-ring cushion	PCP or orthopedic follow-up in 2 to 3 weeks; surgical excision of fracture fragment if chronic pain
Anterior superior iliac spine	Forceful sartorius muscle contraction (eg, adolescent sprinters)	Analgesics, bed rest for 3 to 4 weeks with hip flexed and abducted, crutches	Discharge with orthopedic follow-up in 1 to 2 weeks
Anterior inferior iliac spine	Forceful rectus femoris muscle contraction (eg, adolescent soccer players)	Analgesics, bed rest for 3 to 4 weeks with hip flexed, crutches	Discharge with orthopedic follow-up in 1 to 2 weeks
Ischial tuberosity	Forceful contraction of hamstrings	Analgesics, bed rest for 3 to 4 weeks in extension, external rotation, crutches	Discharge with orthopedic follow-up in 1 to 2 weeks

**TABLE 173-1** Avulsion and Single Bone Fractures

Acetabular fractures are commonly associated with hip dislocations and can be diagnosed with pelvis radiographs and Judet views. CT is more sensitive than radiography in detecting acetabular injury and is helpful in preoperative planning.

### **Emergency Department Care and Disposition**

1. Due to associated hemorrhage and other injuries, patients with pelvic fractures may need resuscitation with crystalloid, blood, and blood products.
2. Stabilize the pelvis with a bed sheet or other pelvic binding device to stabilize fracture ends.
3. In hemodynamically unstable patients, evaluate for other locations of bleeding such as the thorax and the peritoneal cavity using a chest radiograph and the focused assessment with sonography for trauma (FAST) examination.
4. After excluding other sources of hemorrhage, treatment for ongoing hemodynamic instability in patients with pelvic fractures includes angiography with embolization and external fixation.

5. With the exception of lateral compression type I and AP compression type I injuries, all other pelvic ring fractures require open reduction and internal fixation (ORIF).
6. Acetabular fractures require orthopedic consultation and hospital admission. Nondisplaced fractures may be treated with analgesia and bed rest. Displaced fractures are treated with early ORIF.
7. The treatment and disposition of single bone fractures are listed in [Table 173-1](#).

## **HIP FRACTURES**

### **Clinical Features**

The vast majority of hip fractures occur in older patients with osteoporosis or other bony pathology who present after a fall ([Table 173-2](#)). The affected leg is classically shortened and externally rotated. Patients with hip fractures may complain of pain at the site of injury or in the groin and knee. After performing a primary survey and stabilizing the patient, examine the patient for pain, shortening, rotation, deformities, pelvic instability, and neurovascular status. If no significant abnormalities are found, carefully evaluate range of motion. A history of fall or significant trauma should prompt the examiner to evaluate for other injuries.

### **Diagnosis and Differential**

Fracture	Incidence/Demographics	Mechanism	Clinical Findings
Femoral head	Isolated fracture rare; seen in 6% to 16% of hip dislocations; usually result of high-energy trauma; dashboard to flexed knee most common	Superior aspect or impaction fracture in anterior dislocation; inferior aspect in posterior dislocation	Limb shortened and externally rotated (anterior dislocation); shortened, flexed, and internally rotated (posterior dislocation)
Femoral neck	Common in older patients with osteoporosis; rarely seen in younger patients	Low-impact falls or torsion in elderly; high-energy trauma or stress fractures in young	Ranges from pain with weightbearing to inability to ambulate; limb may be shortened and externally rotated
Greater trochanteric	Uncommon; older patients or adolescents	Direct trauma (older patients); avulsion due to contraction of gluteus medius (young patients)	Ambulatory; pain with palpation or abduction
Lesser trochanteric	Uncommon; adolescents (85%) > adults	Avulsion due to forceful contraction of iliopsoas (adolescents); avulsion of pathologic bone (older adults)	Usually ambulatory; pain with flexion or rotation
Intertrochanteric	Common in older patients with osteoporosis; rare in younger patients	Falls; high-energy trauma	Severe pain; swelling; limb shortened and externally rotated
Subtrochanteric	Similar to intertrochanteric; 15% of hip fractures	Falls; high-energy trauma; may also be pathologic	Severe pain; ecchymosis; limb shortened, abducted, and externally rotated

**TABLE 173-2** Proximal Femur and Hip Fractures

Radiographic evaluation of the hip includes AP and lateral views. Other radiographic views that may be helpful include an AP pelvis, which allows a comparison of both sides, and Judet views. Radiographs of the femur and knee may also be indicated. Significant pain with weightbearing or inability to bear weight in patients with normal radiographs should raise suspicion for occult fracture. MRI is very sensitive (nearly 100%) for identifying occult hip fractures and may identify other sources of pain. CT may be useful in identifying fractures not seen on radiographs, but it is not as sensitive as MRI for occult fracture. The differential diagnosis includes pelvic fracture, hip dislocation, femur fracture, sprains, and strains.



## Emergency Department Care and Disposition

1. The treatment of hip fractures is listed in [Table 173-3](#).

Fracture	ED Management	Disposition and Follow-Up
Femoral head	Immediate orthopedic consultation; emergent closed reduction of dislocation; ORIF if closed unsuccessful	Admission to orthopedic or trauma service
Femoral neck	Orthopedic consultation; ranges from nonoperative to total hip arthroplasty	Admission to orthopedic service
Greater trochanteric	Analgesics; protected weightbearing	Orthopedic follow-up 1 to 2 weeks; possible ORIF if displacement > 1 cm
Lesser trochanteric	Analgesics; weightbearing as tolerated; evaluate for possible pathologic fracture	Orthopedic or PCP follow-up in 1 to 2 weeks; admit or urgent follow-up for pathologic fracture
Intertrochanteric	Orthopedic consultation	Admit for eventual ORIF; may need preoperative testing and clearance by PCP or hospitalist
Subtrochanteric	Orthopedic consultation; Hare <sup>®</sup> or Sager <sup>®</sup> splint	Admit for ORIF

Key: AVN = avascular necrosis; DVT = deep venous thrombosis; ORIF = open reduction and internal fixation; PCP = primary care physician.

**TABLE 173-3** Proximal Femur and Hip Fractures Treatment Issues

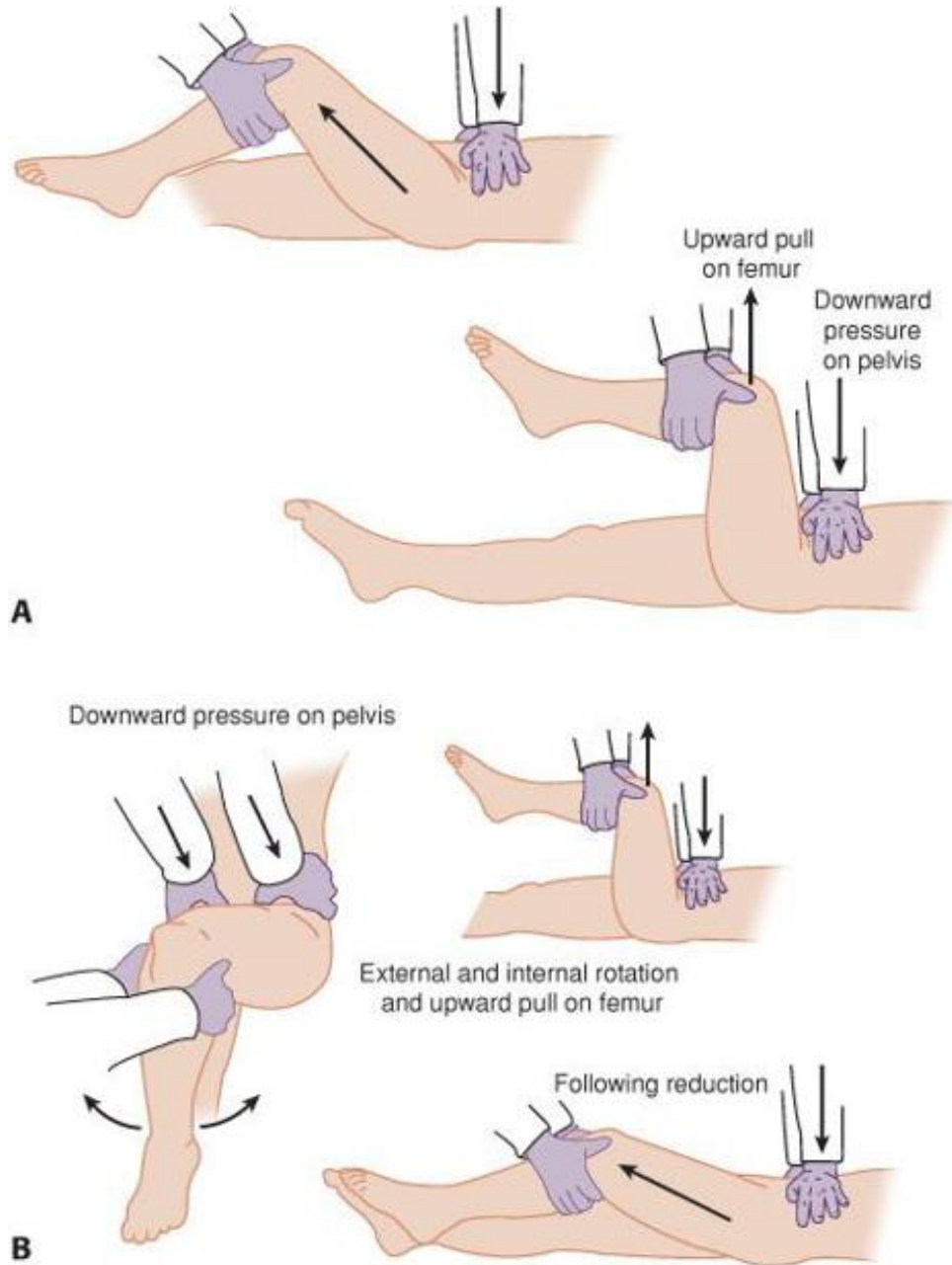
2. If clinical suspicion of an occult fracture is high, obtain either a CT or MRI scan. Alternatively, arrange urgent follow-up for imaging and have the patient remain nonweightbearing.
3. Traction devices may be used for immobilization of subtrochanteric fractures; however, they are contraindicated in femoral neck fractures.

## HIP DISLOCATIONS

Hip dislocations may be anterior or posterior, and commonly result from a high-speed motor vehicle crash. Ninety percent of hip dislocations are posterior, and they may be associated with acetabular fractures. On examination, the extremity is shortened, internally rotated, and adducted.

With anterior dislocations the extremity is held in abduction and external rotation. Assess the patient's neurovascular status. Radiographs of the hip and pelvis will evaluate for hip dislocation. Further assessment of the acetabulum and femur may be done with Judet views or CT.

Hip dislocations are true orthopedic emergencies and should be reduced within 6 hours because delays in reduction correspond with a higher incidence of avascular necrosis. One of the most common methods for hip reduction is described in [Fig. 173-1](#). Order postreduction radiographs or CT to confirm reduction and evaluate for injuries not apparent on initial radiographs.



**FIGURE 173-1. A and B. Allis maneuver for reduction of hip dislocation.**

## **FEMORAL SHAFT FRACTURES**

Fractures of the femoral shaft occur most commonly in younger patients secondary to high-energy trauma. Pathologic fractures can occur due to malignancies. Clinical features include shortening, pain, swelling, and deformity. Assess the patient for neurovascular status, signs of an open fracture, and other injuries. ED treatment includes splinting the extremity with a traction splint unless the patient has a sciatic nerve injury or a grossly contaminated open fracture. Open femur fractures require broad spectrum antibiotics and copious irrigation. Obtain emergent orthopedic consultation for further debridement and definitive management.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 269, "Pelvis Injuries," by Mark T. Steele and Jeffrey G. Norvell; and Chapter 270, "Hip and Femur Injuries," by Mark T. Steele and Amy M. Stubbs.

# CHAPTER 174

## Knee and Leg Injuries

Sandra L. Najarian

### FRACTURES

#### Clinical Features

Patients with patellar fractures present with focal tenderness and swelling, and usually with a loss of the extensor mechanism. Patients with femoral condyle fractures present with pain, swelling, deformity, rotation, shortening, and an inability to ambulate. Popliteal artery injury, deep peroneal nerve injury, ipsilateral hip dislocation or fracture, and quadriceps mechanism injury are associated with these fractures. Tibial spine fractures present with tenderness, swelling, inability to extend the knee, and a positive Lachman test. Patients with tibial plateau fractures have pain, swelling, and limited range of motion. Ligamentous instability is present in about one-third of these fractures. Patients with tibial shaft fractures present with pain, swelling, and crepitation. Distal tibial fractures involving the articular surface (tibial plafond or Pilon fracture) present with pain, swelling, and tenderness about the ankle. The risk of compartment syndrome is high with these types of tibial fractures and mandates a thorough neurovascular examination. Proximal fibular fractures may be associated with ankle injuries. Patients with isolated fibular shaft fractures may be able to bear weight.

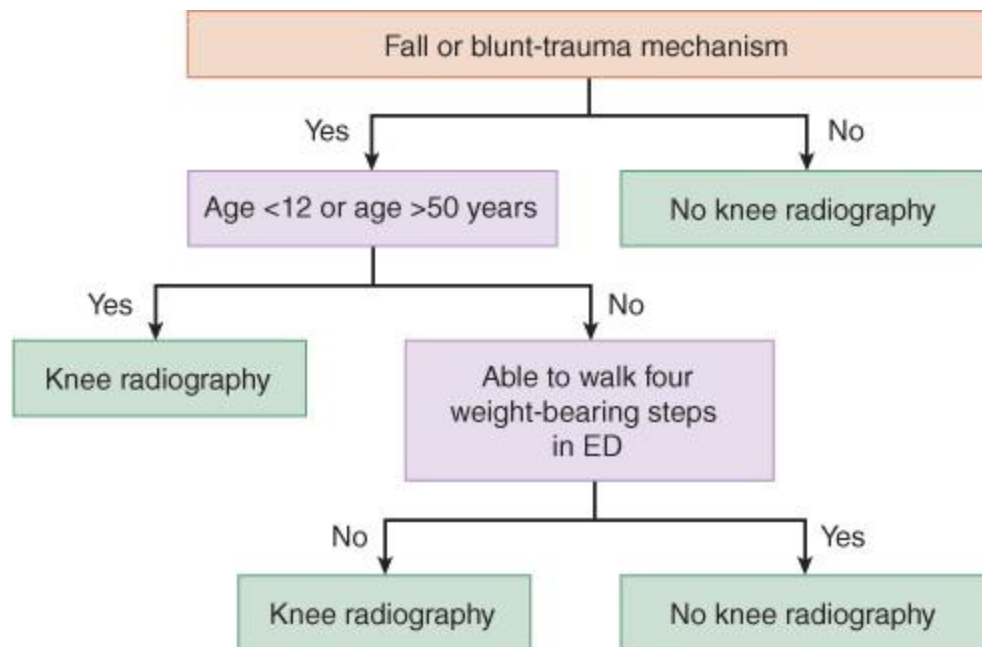
#### Diagnosis and Differential

The Ottawa Knee rules ([Table 174-1](#)) or the Pittsburgh Knee rules ([Fig. 174-1](#)) should be used to determine if radiography is needed for the knee.

These rules have been validated in both children and adults. In suspected tibial and fibular injuries, radiographs of the ankle and knee also may be necessary to exclude associated fractures.

Patient age >55 years (rules have been validated for children 2 to 16 years of age)
Tenderness at the head of the fibula
Isolated tenderness of the patella
Inability to flex knee to 90°
Inability to transfer weight for 4 steps both immediately after the injury and in the ED

**TABLE 174-1** Ottawa Knee Rules: Radiograph if 1 Criterion Is Met



**FIGURE 174-1.** Pittsburgh Knee rules for radiography. (Reproduced with permission from Seaberg DC, Yealy DM, Lukens T, et al: Multicenter comparison of two clinical decision rules for the use of radiography in acute, high-risk knee injuries. *Ann Emerg Med* 32: 8, 1998.)

## Emergency Department Care and Disposition

[Table 174-2](#) describes the mechanism and treatment for the various knee fractures. Most tibial fractures require emergent orthopedic consultation. Conditions for emergent operative repair include open fractures, vascular compromise, or compartment syndrome. Patients may be placed in long-leg immobilization and discharged home if they have a low-energy mechanism, have their pain well-controlled, and are not at risk for compartment syndrome. Treatment for isolated fibular shaft fractures includes splinting, ice, elevation, and orthopedic or primary care physician follow-up. Proximal fibular fractures associated with ankle injuries require surgical intervention and urgent orthopedic consult.

Fracture	Mechanism	Treatment
Patella	Direct blow (ie, fall, motor vehicle crash) or forceful contraction of quadriceps muscle	<p>Nondisplaced fracture with intact extensor mechanism: knee immobilizer, rest, ice, analgesia</p> <p>Displaced &gt;3 mm or with disruption of extensor mechanism: above treatment plus early referral for ORIF</p> <p>Severely comminuted fracture: surgical debridement of small fragments and suturing of quadriceps and patellar tendons</p> <p>Open fracture: irrigation and antistaphylococcal antibiotics in the ED; debridement and irrigation in the operating room</p>
Femoral condyles	Fall with axial load or a blow to the distal femur	<p>Incomplete or nondisplaced fractures in any age group or stable impacted fractures in the elderly: long leg splinting and orthopedic referral</p> <p>Displaced fractures or fractures with any degree of joint incongruity: splinting and orthopedic consult for ORIF</p>

Tibial spines and tuberosity	Force directed against flexed proximal tibia in an anterior or posterior direction (ie, motor vehicle crash, sporting injury)	Incomplete or nondisplaced fractures: immobilization in full extension (knee immobilizer) and orthopedic referral in 2 to 7 days Complete or displaced fracture: early orthopedic referral, often requires ORIF
Tibial tubercle	Sudden force to flexed knee with quadriceps contracted	Incomplete or small avulsion fracture: immobilization Complete avulsion: ORIF
Tibial plateau	Valgus or varus forces combined with axial load that drives the femoral condyle into the tibia (ie, fall, leg hit by car bumper)	Nondisplaced, unilateral fracture: knee immobilizer with nonweightbearing and orthopedic referral in 2 to 7 days Depression of articular surface: early orthopedic consult for ORIF

Key: ORIF = open reduction and internal fixation.

**TABLE 174-2** Mechanism of Knee Injury and Treatment

## DISLOCATIONS

### Clinical Features

Patella dislocation results in pain and deformity of the knee. Tearing of the medial knee joint capsule can occur. Knee dislocations result in significant ligamentous and capsular disruption. Multidirectional instability of the knee should raise the suspicion for a spontaneously reduced knee dislocation. A high incidence of associated injuries, such as popliteal artery injury and peroneal nerve injury, exists with knee dislocations.

### Diagnosis and Differential

Radiographs may help exclude associated fractures. Arteriography is recommended for all patients with confirmed knee dislocations by some orthopedists.

### Emergency Department Care and Disposition



Flex the hip and hyperextend the knee in order to reduce a patellar dislocation. Knee immobilization and orthopedic follow-up are necessary.

For knee dislocations, early reduction is essential along with documentation of pre-and postreduction neurovascular status. Immediate orthopedic and vascular surgery consultation is warranted for all knee dislocations, and admission is mandatory for observation of neurovascular status.

## **TENDON, LIGAMENTOUS, AND MENISCAL INJURIES**

### **Clinical Features**

Patients with quadriceps or patellar tendon rupture have pain and swelling about the knee and will not be able to extend the knee against resistance. A palpable defect is present above or below the knee depending on which tendon is involved. Most ligamentous injuries present with hemarthrosis, although serious ligamentous injuries may present with little pain and no hemarthrosis due to disruption of the capsule. Patients with anterior cruciate ligament (ACL) tears often describe a “pop” and significant swelling over the next several hours after injury. The Lachman test is the most sensitive test for ACL injuries. The anterior drawer and pivot shift test are also useful for diagnosis. Posterior cruciate ligament injuries may result in a positive posterior drawer test; the composite history and examination findings, however, are more accurate for diagnosis. Medial and lateral collateral ligament injuries are diagnosed with abduction (valgus) and adduction (varus) stress testing in 30° flexion. Laxity > 1 cm without a firm end point compared with the other knee is diagnostic for a complete medial or lateral collateral ligament rupture. Stress testing should be repeated in extension. If laxity is present in extension, then this indicates injury to the cruciate ligament and posterior or posterolateral capsule. Peroneal nerve injury may occur with lateral injuries. Symptoms of meniscal injury include painful locking of the knee; a popping, clicking or snapping sensation; a sense of

instability with activity; or joint swelling after activity. McMurray test and other tests for meniscal injury are not sensitive. Ligamentous injuries may be present along with meniscal injuries.

## **Diagnosis and Differential**

Diagnosis of these injuries is largely clinical. A high riding patella may be seen on the lateral radiograph of the knee with patellar tendon rupture. Radiographs are usually normal or show a joint effusion in ligamentous or meniscal injuries. An avulsion fracture at the site of the lateral capsular ligament on the lateral tibial condyle (Segond fracture) is associated with anterior cruciate ligament rupture. Outpatient MRI or arthroscopy provides definitive diagnosis.

## **Emergency Department Care and Disposition**

Treatment of patellar or quadriceps tendon rupture includes knee immobilization and orthopedic consultation for surgical repair, usually within the first 7 to 10 days after the injury. Treatment for ligamentous and meniscal injuries includes knee immobilization, ice, elevation, analgesics, and orthopedic referral.

## **OVERUSE INJURIES**

Patellar tendonitis (or “jumper knee”) presents with pain over the patellar tendon worsened by running up hills or standing up from a seated position. Treatment includes heat, NSAIDs, and quadriceps-strengthening exercises. Steroid injections should be avoided. Shin splints and stress fractures can present with pain over the anterior leg. Patients typically describe a change or sudden increase in their training pattern. Patients present with activity-induced pain that is relieved by rest; this may progress to constant pain. Radiographs are typically normal. Discontinuation of the activity is the

treatment for both shin splints and stress fractures. If a stress fracture is suspected, then an outpatient bone scan or MRI can confirm the diagnosis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 271, “Knee Injuries,” by Jeffrey N. Glaspy and Mark T. Steele; and Chapter 272, “Leg Injuries,” by Paul R. Heller.

# **CHAPTER 175**

## **Ankle and Foot Injuries**

Sarah Andrus Gaines

### **ANKLE INJURIES**

#### **Ligament and Tendon Injuries**

##### *Clinical Features*

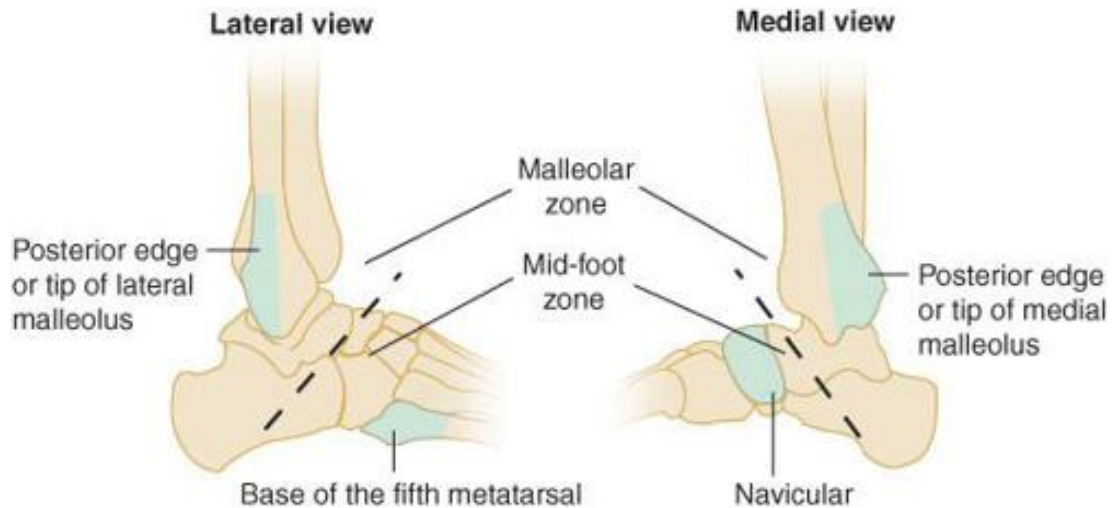
Tendon injuries typically result from either hyperdorsiflexion, when the peroneal tendon is injured, or sudden plantarflexion, which results in an Achilles tendon injury. Patients with an Achilles tendon rupture have severe pain and are unable to walk on their toes, run, or climb stairs. Ligamentous sprains tend to result from inversion and eversion injuries. The most common ankle sprain involves the anterior talofibular ligament. An isolated sprain of the medial deltoid ligament is rare, and an associated fibular fracture (Maisonneuve fracture) or syndesmotomic ligament injury may be present. Any injury with signs of neurovascular compromise requires immediate attention.

##### *Diagnosis and Differential*

Evaluate the ankle along with the joints above and below the injury. A positive Thompson test (with the patient lying prone and knee flexed at 90°, the foot fails to plantarflex when the calf is squeezed) is diagnostic of Achilles tendon rupture. Palpate the proximal fibula for tenderness resulting from a fracture or fibulotibialis ligament tear. Squeeze the fibula toward the tibia to evaluate for syndesmotomic ligament injury. If tenderness is isolated to

the posterior aspect of the lateral malleolus, then a peroneal tendon subluxation may be present.

The Ottawa Ankle Rules guide clinicians in determining when imaging studies are needed for suspected ankle injuries ([Fig. 175-1](#)).



**FIGURE 175-1.** Ottawa ankle rules for ankle and midfoot injuries. Ankle radiographs are required only if there is any pain in the malleolar zone or midfoot zone along with bony tenderness in any of these four locations or the inability to bear weight both immediately and in the ED.

Joint stability is the primary determinant of a treatment plan for a sprain. Instability is suspected based on physical examination and radiography. The examiner may perform the anterior drawer and talar tilt tests to assess stability. If the examiner is unable to perform reliable stress testing, the injury is considered potentially unstable. Any asymmetry in the gap between the talar dome and the malleoli on the talus x-ray view suggests joint instability.

### ***Emergency Department Care and Disposition***

1. If the patient has a stable joint and is able to bear weight, then **protection** (with an elastic bandage or ankle brace), **rest, ice, compression, and elevation (PRICE)** for up to 72 hours is indicated. Prescribe analgesics,

and add motion and strength exercises within 48 to 72 hours. The patient should follow-up in 1 week if the pain persists.

2. A patient with a stable joint who is unable to bear weight requires an **ankle brace** and orthopedic follow-up.
3. A patient with an unstable joint requires a **posterior splint** and referral to an orthopedist for definitive care.
4. Treatment of Achilles tendon rupture includes splinting in plantar flexion, nonweightbearing, and referral to an orthopedist for possible operative repair.

**Dislocations** Posterior dislocations are the most common ankle dislocation and occur with a backward force on the plantarflexed foot, usually resulting in rupture of the tibiofibular ligaments or a lateral malleolus fracture. Reduce ankle dislocations immediately if vascular compromise (absent pulses, a dusky foot, or skin tenting) is present. Grasp the heel and foot and apply downward traction, with analgesia and sedation as needed. Following successful reduction, apply a splint, assess postreduction neurovascular status, obtain postreduction radiographs, and consult orthopedics immediately.

**Fractures** Ankle fractures are classified as unimalleolar, bimalleolar, and trimalleolar. Bi- and trimalleolar fractures require open reduction and internal fixation (ORIF) by the orthopedist. ED care includes posterior splinting, elevation, ice application, and orthopedic consultation. Treat unimalleolar fractures with nonweight bearing and posterior splinting. Manage minimally displaced avulsion fractures of the fibula like ankle sprains. Ankle fractures can be occult and associated with other parts of the lower extremity ([Table 175-1](#)). Patients with open fractures require wet sterile dressing, splinting, tetanus toxoid as necessary, a first generation cephalosporin (eg, **cefazolin** 1 gram IV), and immediate orthopedic consultation.

Injury	Clinical Suspicion	Confirmatory Test
Important to identify in the ED		
Maisonneuve fracture	Examine proximal fibula and shaft, tenderness to palpation	Fibula radiograph
Peroneal tendon dislocation	Palpable anterior tendon dislocation or subluxation	Clinical examination
Usually identified in follow-up of ankle sprains		
Osteochondral injuries	Diffuse ankle swelling, passive plantar flexion	Ankle mortise view/CT
Syndesmosis tear	Significant ankle pain, positive squeeze test	Widened mortise with weightbearing
Anterior calcaneal process fracture	Tenderness more inferoanterior than a typical ankle sprain	Lateral ankle radiograph/CT
Lateral talar process fracture	Tenderness just distal to the tip of fibula	Ankle mortise view/CT
Os trigonum	Tenderness anterior to Achilles tendon	Lateral ankle radiograph

**TABLE 175-1** Associated and Occult Injuries of the Ankle

## FOOT INJURIES

### Clinical Features

The foot is divided into the hindfoot, midfoot, and forefoot. The Chopart joint separates the hindfoot and midfoot, and the Lisfranc joint separates the midfoot and forefoot.

### Diagnosis and Differential

Pay special attention on physical examination to the base of the fifth metatarsal and the area over the base of the second metatarsal. CT is indicated for suspected Lisfranc joint injuries.

### *Hindfoot Injuries*

Calcaneal injuries require a large force, and associated injuries are common. Measure Boehler angle (formed by the intersection of a line connecting the posterior tuberosity and apex of the posterior facet and a line from the posterior facet to the apex of the anterior facet, on the lateral radiograph view) if concerned for a calcaneal compression fracture. An angle of less than  $25^\circ$  is suggestive of a fracture. Treat with a posterior splint, elevation, analgesics, and orthopedic consultation. Manage small avulsion fractures of the talus with posterior splinting and orthopedic follow-up. Major fractures of the talar neck and body and subtalar dislocations require immediate orthopedic consultation.

### ***Midfoot Injuries***

Injuries around the tarsometatarsal joint and pain with torsion of the mid-foot is suspicious for a Lisfranc injury. Lisfranc joint injuries are often associated with a fracture, especially at the base of the second metatarsal. On x-ray, a gap greater than 1 mm between the bases of the first and second metatarsals is considered unstable. These injuries require CT and orthopedic consultation. Isolated navicular, cuboid, and cuneiform injuries are rare and treated conservatively.

### ***Forefoot***

Treat nondisplaced metatarsal shaft fractures with a posterior splint or orthopedic shoe. Fractures with  $> 3$  to 4 mm displacement require surgical reduction. Treat pseudo Jones fractures (nondisplaced avulsion fractures of the tuberosity of the fifth metatarsal) with a walking cast. Manage true Jones fractures (metaphyseal-diaphyseal junction fracture of the fifth metatarsal) with a nonweightbearing cast and orthopedic follow-up for potential surgery. Nondisplaced phalangeal fractures require buddy taping and a stiff-sole shoe. Treat displaced fractures and dislocations with a digital block, reduction by manual traction, and buddy taping. Recommended imaging and care for foot injuries can be found in [Table 175-2](#).



Fracture or Injury Type	ED Imaging	ED Care* (well-padded splints)	Orthopedic Referral (immediate: within 24 h; early: within 2 weeks)
Calcaneal, intra- and extraarticular	Plain films, Böhler angle; CT for subtle findings	Posterior splint	Intra: immediate Extra: early
Talus fx	CT	Posterior splint	Major: immediate Minor: early
Lisfranc	CT	Splint	Displaced: ortho in ED Nondisplaced: early
Navicular fx	Plain films or CT	Splint	Nondisplaced: early Displaced: immediate
Cuboid fx	Plain films or CT	Splint	Early
Cuneiforms fx	Plain films or CT	Splint	Early
Jones fx	Plain films; CT for athletes	Splint	Early
Metatarsals fx	Plain films	Posterior splint	Within 1 week for a cast
Stress fx	Clinical	—	—
Phalange fx	Plain films	Buddy or forefoot taping	—
Open fractures of any kind	Consider antibiotics, diphtheria-tetanus vaccine	Pain control	Ortho consult in ED

Key: fx = fracture; NWBS = nonweightbearing status; Ortho = orthopedic; RICE = rest, ice, compression, elevation.

\*All patients with fractures should receive adequate analgesia.

**TABLE 175-2** Summary of Emergent Care of Bony Foot Injuries

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 273, “Ankle Injuries,” by Daniel A. Handel and Sarah Andrus Gaines; and Chapter 274, “Foot Injuries,” by Sarah Andrus Gaines, Daniel A. Handel, and Peter N. Ramsey.

# CHAPTER 176

## Compartment Syndrome

Sandra L. Najarian

Elevated pressures within a confined muscle compartment can lead to functional and circulatory impairment of that limb. The most common compartments affected are in the leg and forearm. An increase in compartment size and volume or external compressive forces can lead to development of this syndrome ([Table 176-1](#)).

Orthopedic	Tibial fractures Forearm fractures
Vascular	Ischemic-reperfusion injury Hemorrhage
Iatrogenic	Vascular puncture in anticoagulated patients IV/intraarterial drug injection Constrictive casts
Soft tissue injury	Prolonged limb compression Crush injury Burns

**TABLE 176-1** Common Causes of Compartment Syndrome

## CLINICAL FEATURES

Severe and difficult to control pain, pain out of proportion to examination, and pain with passive stretch of the limb are the hallmark symptoms of this disease. Nerve dysfunction often accompanies the pain and is manifested by burning or dysesthesias in the sensory distribution of the nerve. Motor function can be impaired as well. On exam, the compartment is often swollen, firm, and tender to palpation. The 5 P's of compartment syndrome (pain, paresthesias, pallor, poikilothermia, and pulselessness) need not all be present to make the diagnosis. The affected limb can maintain temperature,

color, and detectable pulse until late in the disease process. Symptoms may begin within a few hours of the injury or up to 48 hours after the event.

## **DIAGNOSIS AND DIFFERENTIAL**

Maintain a high vigilance for this diagnosis, especially in patients with altered mental status or who are sedated. If the diagnosis is considered after the clinical assessment, then directly measure the compartment pressures. Several commercial devices are available to measure compartment pressures. Normal compartment pressure is <10 mm Hg. The exact pressure elevation at which cell death occurs is unclear. Traditionally, any pressure between 30 to 50 mm Hg was felt to be detrimental if left untreated for several hours. The “delta pressure,” the diastolic blood pressure minus the measured tissue pressure, better predicts potential for irreversible muscle damage. A delta pressure  $\leq 30$  mm Hg is most commonly used to diagnose acute compartment syndrome. Hypotensive patients do not tolerate elevated compartment pressures as well as normotensive patients. The differential diagnosis for compartment syndrome includes other causes of pain, such as fracture, hematoma, or infection, and other causes of neurologic or vascular compromise once symptoms progress beyond pain only.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Surgical fasciotomy is necessary once the diagnosis is confirmed. While definitive management is being arranged, administer supplemental oxygen, correct hypotension, remove constrictive casts or dressings, and place the affected limb at the level of the heart. Functional outcomes are favorable when diagnosis and treatment of compartment syndrome occurs within 6 hours of its onset.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 275, “Compartment Syndrome,” by Paul R. Haller.

# **SECTION 20 Nontraumatic Musculoskeletal Disorders**

## **CHAPTER 177**

# **Neck and Thoracolumbar Pain**

Amy M. Stubbs

Neck and back pain are common complaints seen in the ED and have a wide array of causes, ranging from benign to life threatening. Workup of these complaints requires a thorough history with careful attention to risk factors for serious pathology. A complete physical examination includes evaluation of specific nerve roots and dermatomes.

## **CLINICAL FEATURES**

Neck and back pain are often due to nonspecific musculoskeletal causes, but symptoms may also be attributable to spinal nerve roots (radiculopathy) or the spinal cord itself (myelopathy) ([Tables 177-1](#), [177-2](#), [177-3](#)). Thoracolumbar pain may be categorized by symptom duration: acute (<6 weeks), sub-acute (6 to 12 weeks), or chronic (> 12 weeks).

<b>Group 1: Cervical Problems Arising Mainly from Neck Joints and Associated Ligaments and Muscles</b>	<b>Group 2: Cervical Problems Involving the Cervical Nerve Roots or the Spinal Cord</b>
<p>Patients complain of pain and stiffness. Pain is a deep, dull aching sensation and often episodic. Patients have a history of excessive or unaccustomed activity or of sustaining an awkward posture. There is no history of specific injury. Ligament and muscle pain are localized and asymmetric. Pain from upper cervical segments is referred toward the head; pain from lower segments, to the upper limb girdle. Symptoms are aggravated by neck movement and relieved by rest.</p>	<p>Patients complain of significant root pain, typically sharp, intense, and may be described as "burning." Pain may radiate to the trapezial and periscapular areas or down the arm. Patients complain of numbness and motor weakness in a myotomal distribution. Headache may occur if the upper cervical roots are involved. Symptoms often become more severe with neck hyperextension (especially when the head is toward the affected extremity). Patients may experience gradual onset of shocklike sensations spreading down spine to extremities. Most common myelopathy at the level of the fifth cervical vertebra and affects shoulder abduction (deltoid) and external rotation (infraspinous).</p>

**TABLE 177-1** Symptoms and History Associated with Neck Pain

<b>Disk Space/ Nerve Root</b>	<b>Pain Complaint</b>	<b>Sensory Change</b>	<b>Motor Weakness/Altered Reflex</b>
C1-2/C1-2	Neck, scalp	Scalp	None/None
C4-5/C5	Neck, shoulder, upper arm	Shoulder, thumb	Infraspinatus, deltoid, biceps/Biceps reflex
C5-6/C6	Neck, shoulder, upper scapula, proximal forearm, thumb	Thumb, index finger, lateral forearm	Deltoid, biceps, pronator teres, wrist extensors/ Biceps and brachioradialis reflex
C6-7/C7	Neck, posterior arm, dorsal and proximal forearm, chest, medial scapula, middle finger	Middle finger, forearm	Triceps, pronator teres/ Triceps reflex
C7-T1/C8	Neck, posterior arm, proximal forearm, medial scapula, medial hand, ring and little fingers	Ring, little fingers	Triceps, flexor carpi ulnaris, hand intrinsic muscles/ Triceps

**TABLE 177-2** Symptoms and Signs of Cervical Radiculopathies

Disk Space	Nerve Root	Pain Complaint	Sensory Change	Motor Weakness	Altered Reflex
L2-3	L3	Medial thigh, knee	Medial thigh, knee	Hip flexors	None
L3-4	L4	Medial lower leg	Medial lower leg	Quadriceps	Knee jerk
L4-5	L5	Anterior tibia, great toe	Medial foot	Extensor hallucis longus	Biceps femoris
L5-S1	S1	Calf, little toe	Lateral foot	Foot plantar flexors	Achilles

**TABLE 177-3** Symptoms and Signs of Lumbar Radiculopathies

The history should include the pain's circumstances of onset, location (including any radiation), duration, and any exacerbating/alleviating factors. Neurologic complaints should also be sought. Concern for problems such as spinal cord compression should be high when complaints include weakness, paresthesia, or urinary or fecal problems (retention or incontinence). Risk for serious neurologic or bony pathology is higher when there are comorbidities (eg, cancer, rheumatologic disease, osteoporosis), constitutional symptoms such as fever or weight loss, or pain that is unremitting or occurs during the night.

Evaluation of mobility should include attention to presence of severe pain or gait disturbances. The structures of the head, neck, and back should be exposed and palpated for point tenderness, deformity, or signs of infection. A thorough neurologic examination, including assessment of specific nerve roots' strength, sensation, and reflexes, is vital ([Tables 177-2, 177-3](#)). Exam maneuvers should be performed as indicated, and may include straight-leg testing or assessment for the Lhermitte sign (electric shock-like pain in the spine and extremities occurring with neck flexion, suggesting cord compression). A rectal examination to evaluate for tone, sensation, masses, or infection should be performed when there are neurologic deficits or concerns for serious disease.

## **DIAGNOSIS AND DIFFERENTIAL**

The differentials for neck and back pain are broad but can generally be guided by the history and physical examination. The majority of patients

with neck and back pain will not require emergent imaging or diagnostic testing.

Imaging decision-making in patients with neck and back pain is informed by clinical suspicion as well as considerations of cost and radiation exposure. Plain films of the spine have low sensitivity but may be considered as an initial imaging step if clinical suspicions include tumor, fracture, or infection. **For patients with pain and neurologic deficits, MRI is the definitive test.** CT scan is helpful for identification of disorders of the bony skeleton (eg, fracture, osteomyelitis), but its sensitivity for nerve root or spinal cord disorders is poor. CT myelography can serve as an alternative to MRI, if the latter is contraindicated.

Three views of the cervical spine may be helpful in patients with chronic neck pain or those with a history of trauma, surgery, malignancy, or rheumatologic disease. Flexion-extension films may be considered if instability is suspected. If radiographs are normal, or show only spondylosis (ie, degenerative disease), and if the patient has a benign examination, no further imaging is required. For patients with back pain, if plain thoracic and/ or lumbar radiographs are indicated (as discussed above), anteroposterior and lateral views suffice. Compression fractures may be seen on thoracic spine films (eg, in osteoporotic patients). Advanced imaging is necessitated by presence of neurological deficits or abnormal plain radiographs.

Laboratory testing will not be useful in the majority of patients with neck and back pain. If serious pathology (eg, malignancy, infection) is suspected, a complete blood count (CBC), erythrocyte sedimentation rate (ESR), and urinalysis should be ordered. ESR has a sensitivity of 90% to 98% for infectious causes of back pain. Urinalysis is useful to rule out urinary or renal pathology as a pain source. Postvoid residual (PVR) assessment, with ultrasound or catheterization, should be performed on patients complaining of urinary retention. Elevated PVR is the most common finding in cauda equina syndrome, and PVR volume exceeding 100 mL may indicate overflow incontinence due to epidural compression.

Mechanical neck disorders, such as strain caused by trauma, are often characterized by delayed pain and paracervical pain and stiffness. National Emergency X-Radiography Utilization Study (NEXUS) criteria can be



utilized to determine the need for imaging (see [Chapter 161](#) “Spine and Spinal Cord Injuries”).

Cervical disc herniation, spondylosis, or stenosis can lead to radiculopathy or myelopathy. Signs and symptoms of a radiculopathy include pain and weakness in a dermatomal distribution ([Table 177-2](#)). Lower extremity hyperreflexia, a positive Babinski, or loss of sphincter tone are suspicious for myelopathy; any of these findings necessitate MRI. Metastatic cancer may also cause a radiculopathy or myelopathy and should be included in the differential for chronic pain. Osteomyelitis, epidural abscess or hematoma (consider in patients with coagulopathy), and transverse myelitis may also cause neck pain with neurologic deficits; these disorders are more commonly observed in the thoracic or lumbar spine.

Neck pain can be caused by a variety of other disorders such as myofascial pain syndrome, temporal arteritis, ischemic heart disease, and neurodegenerative disorders. The distribution of symptoms in these disorders will typically not be dermatomal, and they will usually be distinguished by other historical features.

The majority of patients with back pain have nonspecific back pain; they have no radiculopathy or myelopathy and no specific etiology is found. These patients typically have benign examinations and their pain is often exacerbated by movement. Diagnostic evaluation is negative.

As with neck pain, disc herniation and degenerative changes of the thoracic or lumbar spine can be a cause of acute or chronic back pain. More than 95% of disc herniations will be at the L4-L5 or L5-S1 nerve roots, compression of which can cause sciatica. These patients may have leg pain, neurologic deficits localized to a unilateral nerve root, and a positive straight-leg test. Spinal nerve root compression can occur at any level in the thoracic or lumbar spine, with associated localizing symptoms ([Table 177-3](#)). Also occurring at any level are epidural compression syndromes which include spinal cord compression, cauda equina syndrome, and conus medullaris syndrome. All epidural compression syndromes may cause pain, neurologic deficits, or autonomic dysfunction at different cord levels. The thoracic spine is the site of most (70%) cases of metastatic epidural compression.

Infectious causes of back pain include osteomyelitis, discitis, and epidural abscess, all of which are often missed on initial presentation. A high level of suspicion should be maintained in patients with risk factors such as immunocompromise, recent back surgery, retained hardware, or intravenous drug use. Plain films may show bony destruction in patients with osteomyelitis, but x-rays are often initially normal in cases of infectious back pain. MRI is typically required for diagnosis. The ESR is will be elevated in the majority of cases.

Other causes of back pain may originate from the spine itself, or from nonspinal causes. Spinal stenosis can cause low back and leg pain that mimics claudication, worsening with walking and improving with rest. CT, MRI, and sometimes angiography are needed to make the diagnosis. Ankylosing spondylitis, an autoimmune arthritis, causes chronic back pain and is identifiable by a characteristic squaring of vertebral bodies on radiography. Transverse myelitis, an inflammatory disorder of the spinal cord, may mimic a compression syndrome. Ruptured abdominal aneurysm, pyelonephritis, pancreatitis, or renal infarction should also be considered in patients with nonspecific back pain accompanied by concerning history or exam findings.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. Patients with neck and back pain that have progressive neurologic deficits, myelopathy, or intractable pain should be imaged as indicated (typically MRI) and admitted to the appropriate service for further management. **Dexamethasone 10 milligrams IV should be given prior to imaging for suspected epidural compression.**
2. Patients with a stable or mild radiculopathy may be managed conservatively with pain medication, routine activity, and strict return precautions for worsening symptoms. Outpatient MRI and neurosurgical follow-up should be considered for patients who have failed conservative treatment.

3. Pain management may include NSAIDs (if no contraindication), acetaminophen, or muscle relaxants (eg, diazepam 5 to 10 milligrams PO tid), used singly or in combination; all have been shown to be effective with no agent proved superior. A short course of oral opioids may be prescribed for patients with moderate to severe pain, but long-term use is discouraged. Other treatments such as manipulation or corticosteroids have limited benefit and should not be prescribed in the ED.
4. The majority of patients with neck or back pain will have benign courses and improve with time. These patients may be prescribed pain medicine as appropriate and reassurance should be provided.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 276, "Neck and Back Pain," by William J. Frohna and David Della-Giustina.

# **CHAPTER 178**

## **Shoulder Pain**

Andrew D. Perron

Shoulder pain is a common musculoskeletal complaint, especially in patients older than 40 years. Occupational, recreational, and normal daily activities stress the shoulder joint and may result in pain from acute injury or, more commonly, chronic overuse conditions. Complicating the evaluation of shoulder pain is that the origin of pain may be from pathology intrinsic to the shoulder joint or from extrinsic disorders causing referred pain.

### **CLINICAL FEATURES**

The pain of musculoskeletal shoulder pathology often is described by patients as an aching sensation, particularly in the setting of a more chronic process. Nighttime pain is a common feature of intrinsic shoulder pathology. Specific motions may exacerbate it, and this history is helpful in making a specific diagnosis. Decreased range of motion, crepitus, weakness, or muscular atrophy may be associated with certain conditions. Any systemic symptoms (eg, shortness of breath, fever, or radiation of pain from the chest or abdomen) should raise suspicion for extrinsic and potentially life-threatening problems.

### **DIAGNOSIS AND DIFFERENTIAL**

The primary diagnostic maneuver is a thorough history and physical examination. Examination of the shoulder joint should include range of motion and muscle strength testing, palpation for local tenderness or other abnormality, and identification of any neurovascular deficit. Specific tests for impingement and individual tests of rotator cuff muscle function are

often helpful in intrinsic disease. Plain radiographic studies of the shoulder joint are rarely diagnostic but may be helpful to exclude bony abnormalities in selected patients or to evaluate for abnormal calcifications. In patients in whom extrinsic causes of shoulder pain are suspected, further diagnostic testing may be indicated, such as laboratory studies, additional radiographs, and an electrocardiogram.

The differential diagnosis includes a variety of intrinsic musculoskeletal disorders, and individual patients may exhibit considerable overlap in their symptoms manifesting a combination of specific conditions. *Impingement syndrome* is a term that has been adopted to encompass many painful shoulder syndromes that result most frequently from repetitive overhead use of the arm. The pathologic entities included in this syndrome are subacromial tendonitis and bursitis, supraspinatus tendonitis, rotator cuff tendonitis, and the painful arc syndrome. Impingement syndrome is a painful overuse condition characterized by positive findings with impingement testing and relief of pain with anesthetic injection of the subacromial space. *Subacromial bursitis* is generally seen in patients younger than 25 years and will present with positive impingement tests with different degrees of tenderness at the lateral proximal humerus or in the subacromial space. *Rotator cuff tendonitis* is distinguished by an incidence primarily in individuals 25 to 40 years of age and findings of tenderness of the rotator cuff with mild to moderate muscular weakness. In more chronic disease, crepitus, decreased range of motion, and osteophyte formation visible on plain radiograph also may be apparent. *Rotator cuff tears* occur primarily in patients older than 40 years and are associated with muscular weakness (especially with abduction and external rotation) and cuff tenderness. Ninety percent will be chronic tears with a history of minimal or no trauma; in severe disease, muscular atrophy may be present. Acute tears may occur in patients of any age and result from significant force producing a tearing sensation with immediate pain and disability. In patients between the ages of 30 and 50 years, abnormal calcifications on radiograph in the clinical setting of a painful shoulder with rotator cuff tenderness and often crepitus suggest the diagnosis of calcific tendonitis. *Osteoarthritis* is characteristically associated with degenerative disease in other joints (primary) or previous fracture or other underlying disorder (secondary). The hallmark of *adhesive capsulitis* is significantly painful and limited range of

motion often, but not always, associated with a period of immobilization. radiographs should be obtained to exclude posterior glenohumeral dislocation.

Other causes of shoulder pain that should be considered are a number of extrinsic conditions. *Pancoast tumor* may compress the brachial plexus and thus manifest itself as shoulder pain. *Degenerative disease of the cervical spine, brachial plexus disorders, and suprascapular nerve compression* are neurologic processes that should be sought in patient evaluation. Vascular pathology, notably *axillary artery thrombosis*, also may cause shoulder pain. Acute cardiac, aortic, pulmonary, and abdominal pathology may cause pain referred to the shoulder, and the clinician must remain alert to this possibility.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION

1. For intrinsic disease, the primary goals of emergency department care are to reduce pain and inflammation and prevent progression of disease. For most conditions, this translates to relative rest of the joint assisted by use of a sling (full immobilization is not suggested), **nonsteroidal anti-inflammatory drugs**, opioid analgesics as needed, and the application of cold packs. Range-of-motion exercises should be encouraged as soon as pain allows to prevent loss of flexibility and to maintain strength.
2. **Joint space injection** with glucocorticoids (eg, triamcinolone 20 to 40 milligrams) with or without a local anesthetic such as lidocaine should be used judiciously in view of the potential deleterious effects on soft tissues, tendon rupture with direct injection, and a recommended limitation of 3 injections into a single area. For all intrinsic disorders, follow-up with a primary care physician with expertise in joint disease or orthopedic referral is suggested within 7 to 14 days. Physical therapy referral for stretching and strengthening also may be valuable.
3. In extrinsic disease, the treatment and referral pattern will depend on the diagnosis. Neurologic problems will require analgesia and anti-inflammatory medications and may require neurology or neurosurgical

follow-up. Vascular causes of shoulder pain must be evaluated carefully and, with axillary artery thrombosis, immediate consultation made to initiate thrombolysis. Treatment of other extrinsic conditions depends on the specific diagnosis.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 277, "Shoulder Pain," by David Della-Giustina and Benjamin Harrison.

# CHAPTER 179

## Hip and Knee Pain

Jeffrey L. Hackman

Hip and knee pain are common among all people. Athletes are at increased risk due to excess forces on their joints. Knee pain is usually due to local pathology and is not commonly referred to other sites. Hip pathology commonly causes referred pain in the buttocks and lower extremity, and pain felt in the hip may be due to extraarticular pathology. The differential diagnosis for hip or knee pain is broad but a focused history and physical examination will often lead to the diagnosis ([Table 179-1](#)).

Determine the location of the pain to narrow down the potential diagnosis.  
Determine the activities that bring on the pain.  
The knee "giving out" or "buckling" generally is due to pain and reflex muscle inhibition rather than an acute neurologic emergency. This complaint may also represent patellar subluxation or ligamentous injury and joint instability.  
Poor conditioning or quadriceps weakness generally causes anterior knee pain of the patellofemoral syndrome; therapy should address this weakness.  
Locking of the knee suggests a meniscal injury, which may be chronic.  
A popping sensation or sound at the onset of pain is reliable for a ligamentous injury.  
A recurrent effusion after activity suggests a meniscal injury.  
Pain at the joint line suggests a meniscal injury.

**TABLE 179-1** Suggested Clues for the Differential Diagnosis of Hip and Knee Pain

## REGIONAL NERVE ENTRAPMENT SYNDROMES

**Meralgia paresthetica** is a compressive inflammation of the lateral femoral cutaneous nerve causing pain in the hip, thigh, or groin, burning or tingling paresthesias, and hypersensitivity to light touch. ED treatment includes addressing the source of nerve irritation (eg, obesity, pregnancy, tight pants belt) and provision of NSAIDs. **Obturator nerve entrapment** usually



occurs after pelvic fractures or in athletes with a fascial band at the distal obturator canal, which causes pain in the groin and down the inner thigh. Surgery may be needed for pain relief. **Ilioinguinal nerve entrapment** is associated with pregnancy or hypertrophy of the abdominal wall musculature. **Piriformis syndrome**, irritation of the sciatic nerve from the piriformis muscle, manifests as pain in the buttocks and hamstring muscles that is worsened by sitting, climbing stairs, or squatting. ED treatment is conservative for all of these nerve entrapment syndromes.

## **PSOAS ABSCESS**

Abscess of the psoas muscle may present with abdominal pain radiating to the hip or flank, fever, and limp. The diagnosis is made by CT. Treatment includes antibiotics and surgical drainage.

## **BURSAL SYNDROMES OF THE HIP AND KNEE**

Hip and knee bursae may cause localized pain due to inflammation, infection, rheumatologic disorders (psoriatic arthritis, rheumatoid arthritis, ankylosing spondylitis) or crystalline disease (gout, pseudogout). Infection may be difficult to distinguish clinically from more benign disorders. ([Table 179-2](#)) Treatment is directed at the underlying cause. NSAIDs, rest, heat, and time are the basis of treatment for inflammatory conditions. Steroid injections into readily accessible bursa may be useful if infection has been excluded. Care should be taken to avoid injecting steroids into tendons. For infections, treat with antibiotics (see [Chapter 180](#) “Disorders of the Joints and Bursa,” for specific recommendations). Immunocompromised patients with suspected infections should be admitted for IV antibiotics and orthopedic surgery consultation.

	Location of Pain	Associated Symptoms	Common Populations
Trochanteric bursitis	Posterolateral hip	Pain with walking and climbing stairs	Female runners, older women
Iliopsoas bursitis	Groin	Pain with hip extension, tenderness over the middle third of the inguinal ligament	
Ischial or ischio-gluteal bursitis	Ischial prominence	Pain with sitting on a hard surface for long periods	Sedentary people
Iliopectineal bursitis	Anterior hip, pelvis/groin	Pain improves with the hip flexed and externally rotated	
Pes anserine bursitis	Anterior medial knee		Obese women, runners, others with overuse
Prepatellar bursitis	Anterior to the patella	Significant swelling of the bursa	People who repetitively kneel on hard surfaces

**TABLE 179-2** Characteristics of Bursal Syndromes of the Hip and Knee

# MYOFASCIAL SYNDROMES                      SYNDROMES/OVERUSE

Repetitive microtrauma that outpaces the body’s ability to heal results in overuse syndromes ([Table 179-3](#)). Treatment generally consists of NSAIDs, heat, and rest, followed by gradual resumption of activities, physical therapy, and strengthening where appropriate.

	Location of Pain	Associated Symptoms	Common Populations
Snapping hip syndrome	Posterior lateral hip	Snapping sound and popping sensation with hip flexion/extension	Athletes, young women
Fascia lata syndrome	Lateral thigh/ anterior groin		Athletes
Patellofemoral syndrome	Anterior knee	Pain exacerbated by prolonged knee flexion	Females
Iliotibial band syndrome	Lateral knee	Localized tenderness over lateral epicondyles	Distance runners, cyclists
Popliteus tendinitis	Posterior lateral knee	Pain exacerbated by running downhill	Athletes
Patellar tendinitis	Anterior superior knee	Pain exacerbated by running uphill	Jumpers
Infrapatellar fat pad syndrome	Anterior inferior knee	Commonly associated with patellar tendinitis	
Quadriceps tendinitis	Anterior superior knee		Athletes on hard playing surfaces
Semimembranous tendinitis	Posteromedial knee		Younger patients (athletes and overuse), older patients (degenerative changes)

**TABLE 179-3** Characteristics of Myofascial/Overuse Syndromes

## **BONE/ARTICULAR DERANGEMENTS**

**Osteonecrosis** (also known as aseptic necrosis, ischemic necrosis, avascular necrosis) is bone infarction caused by a lack of blood supply. It may be an idiopathic or primary disorder, secondary to a systemic condition, or following trauma. Conditions associated with avascular necrosis of the femoral head include: femoral neck fracture, hip dislocation, occult or minor trauma, sickle cell disease, collagen vascular diseases, alcohol abuse, renal transplant, systemic lupus erythematosus, dysbarism, chronic pancreatitis, exogenous steroid administration, Cushing disease, caisson disease, Gaucher disease, and renal osteodystrophy. Osteonecrosis of the hip may cause pain anywhere from the buttock to the knee. **Osteomyelitis** is an infection of the bone, resulting in bony destruction. Patients have local

pain and may have associated warmth, swelling, and erythema. Acutely ill patients should receive high-dose, broad spectrum, parenteral antibiotics based on the patient’s risk factors and most likely organisms. ([Table 179-4](#)) **Osteitis pubis** occurs following pregnancy, in athletes due to overuse of the adductors and gracilis muscles, and after bladder and prostate surgery. It causes pain in the region of the pubis and generally resolves over a period of months with rest and NSAIDs. **Myositis ossificans** (also known as heterotopic calcification) is the deposition of bone in abnormal sites after direct trauma. Pain and a palpable mass will be present. Pain or physical obstruction may limit motion in the affected muscle or joint. Surgery may be required.

Risk Factor	Likely Infecting Organism	Recommended Initial Empiric Antibiotic Therapy*
Elderly, hematogenous spread	<i>Staphylococcus aureus</i> , including MRSA, gram-negative bacteria	Vancomycin, 1 gram IV, plus piperacillin-tazobactam, 3.375 grams IV or imipenem, 500 milligrams IV
Sickle cell disease	<i>Salmonella</i> , gram-negative bacteria, ( <i>S aureus</i> becoming more common)	Ciprofloxacin, 400 milligrams, consider vancomycin, 1 gram IV
Diabetes mellitus, or vascular insufficiency	Polymicrobial: <i>S aureus</i> , <i>Streptococcus agalactiae</i> , and <i>S pyogenes</i> plus coliforms and anaerobes	Vancomycin, 1 gram IV, plus piperacillin-tazobactam, 3.375 grams IV, or imipenem, 500 milligrams IV
Injection drug user	<i>S aureus</i> including MRSA, and <i>Pseudomonas</i>	Vancomycin, 1 gram IV
Developing nations	<i>Mycobacterium tuberculosis</i>	See Chapter 70, Tuberculosis

Newborn	<i>S aureus</i> including MRSA, gram-negative bacteria, group B <i>Streptococcus</i>	Vancomycin, 15 milligrams/kilogram load, then reduce dose, <i>plus</i> ceftazidime, 30 milligrams/kilogram IV every 12 h
Children	<i>S aureus</i> including MRSA	Vancomycin, 10 milligrams/kilogram every 6 h, <i>plus</i> ceftazidime, 50 milligrams/kilogram every 8 h
Postoperative with or without retained orthopedic hardware	<i>S aureus</i> and coagulase-negative staphylococci	Vancomycin, 1 gram IV
Human bite	Streptococci or anaerobic bacteria	Piperacillin-tazobactam, 3.375 grams IV, or imipenem, 500 milligrams IV
Animal bite	<i>Pasteurella multocida</i> , <i>Eikenella corrodens</i>	Cefuroxime, 500 milligrams IV if known <i>P multocida</i> , piperacillin-tazobactam, 3.375 grams IV or imipenem, 500 milligrams IV

Key: MRSA = methicillin-resistant *Staphylococcus aureus*.

\*All patients require bone biopsy and debridement of infected/dead bone.

**TABLE 179-4** Risk Factors, Likely Infecting Organism, and Recommended Initial Empiric Antibiotic Therapy for Osteomyelitis

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 278, “Hip and Knee Pain,” by Kelly P. O’Keefe and Tracy G Sanson.

## **CHAPTER 180**

# **Acute Disorders of the Joints and Bursae**

Andrew D. Perron

Acute disorders of the joints and bursae are common emergency conditions that involve a wide spectrum of ages, acuities, and etiologies. Mismanagement of certain pathologic entities can lead to significant morbidity for the patient.

## **CLINICAL FEATURES**

Multiple pathways can cause disruption of the normal joint milieu leading to acute joint complaints. These pathways include degeneration of articular cartilage (osteoarthritis), deposition of immune complexes (rheumatoid arthritis), crystal-induced inflammation (gout and pseudogout), seronegative spondyloarthropathies (ankylosing spondylitis and Reiter syndrome), and bacterial and viral invasion (septic arthritis). These pathologic events invariably lead to pain, the most common complaint of patients with a joint problem. Important historical factors to elicit include a determination of previous joint or bursal disease; presence of constitutional symptoms; and whether the pain is acute, chronic, or acute on chronic. Determining the number and distribution of joints affected as can help narrow the differential diagnosis ([Table 180-1](#)). Systemic lupus erythematosus may present with a migratory pattern of joint pain, while migratory pain is characteristic of the following infectious etiologies: gonococcal arthritis, acute rheumatic fever, Lyme disease, and viral arthritis.

Number of Joints	Differential Considerations
1 = Monoarthritis	Trauma-induced arthritis Nongonococcal septic arthritis Gonococcal septic arthritis Crystal-induced (gout, pseudogout) Osteoarthritis (acute) Lyme disease Avascular necrosis Tumor
2 to 3 = Oligoarthritis	Lyme disease Reactive arthritis (Reiter syndrome) Ankylosing spondylitis Gonococcal arthritis Rheumatic fever
>3 = Polyarthritis	Rheumatoid arthritis Systemic lupus erythematosus Viral arthritis Osteoarthritis (chronic) Serum sickness Serum sickness–like reactions

**TABLE 180-1** Classification of Arthritis by Number of Affected Joints

On physical examination, arthritis should be distinguished from more focal periarticular inflammatory processes such as cellulitis, bursitis, and tendonitis. True arthritis produces joint pain exacerbated by active and passive motions.

## DIAGNOSIS AND DIFFERENTIAL

With the exception of recent joint surgery or cellulitis overlying a prosthetic knee or hip, history, physical examination, and routine blood tests do not distinguish acute septic arthritis from other forms of arthritis. Clinicians who suspect septic arthritis based on the patient's presentation should perform arthrocentesis. Synovial fluid should be sent for culture, Gram stain, cell count, and crystal evaluation ([Table 180-2](#)). Except in pediatric septic arthritis, where the erythrocyte sedimentation rate has been shown to have a 90% sensitivity, the serum white blood cell count and erythrocyte sedimentation rate lack the sensitivity and specificity to be reliable discriminators in disorders of the joints and bursae. Adults with risks for

sexually transmitted disease and migratory symptoms and or tenosynovitis should be evaluated for gonococcal arthritis.

	Normal	Noninflammatory	Inflammatory	Septic
Clarity	Transparent	Transparent	Cloudy	Cloudy
Color	Clear	Yellow	Yellow	Yellow
WBC/ $\mu$ L	<200	<200 to 2000	200 to 50 000	>50 000
PMNs (%) <sup>*</sup>	<25	<25	>50%	>50%
Culture	Negative	Negative	Negative	>50% positive <sup>†</sup>
Crystals	None	None	Multiple or none	None
Associated conditions		Osteoarthritis, trauma, rheumatic fever	Gout, pseudogout, RA, Lyme disease, SLE	Septic arthritis

Key: RA = rheumatoid arthritis, SLE systemic lupus erythematosus, PMNs = polymorphonuclear neutrophils.

<sup>\*</sup>The WBC and PMNs are affected by a number of factors, including disease progression, affecting organism, and host immune status. The joint aspirate WBC and %PMNs should be considered part of a continuum for each disease, particularly septic arthritis, and should be correlated with other clinical information.

<sup>†</sup>Gonococcal arthritis is frequently culture negative.

**TABLE 180-2** Examination of Synovial Fluid

Radiographs should be obtained when the differential diagnosis includes trauma, tumor, osteomyelitis, ankylosing spondylitis, or avascular necrosis. More sophisticated modalities such as computed tomography, magnetic resonance imaging, and radioisotope scanning are used in isolated cases.

## EMERGENCY DEPARTMENT CARE AND DISPOSITION OF SPECIFIC CONDITIONS

*Septic arthritis* is a condition that can rapidly lead to irreversible joint destruction if inadequately treated. It typically presents as a monoarticular arthritis and may be associated with fever, chills, or malaise, although absence of these symptoms does not exclude the diagnosis. The synovial fluid confirms the diagnosis. Therapy requires admission for parenteral antibiotics and repeated needle aspiration, arthroscopy, or open surgical



drainage. Specific patient demographics can help guide empiric antibiotic therapy in septic arthritis ([Table 180-3](#)).

Patient/Condition	Expected Organisms	Antibiotic Considerations
Older children and healthy adults, or patients with risk factors for <i>Neisseria gonorrhoeae</i>	<i>Staphylococcus</i> , <i>N gonorrhoeae</i> , <i>Streptococcus</i> , gram-negative bacteria	Vancomycin, 15 milligrams/kilogram IV load—if Gram stain reveals gram-positive organisms in clusters; ceftriaxone, 1 gram IV, or imipenem, 500 milligrams IV, should be used/added if either gram-negative organisms are present or no organisms present on Gram stain and <i>N gonorrhoeae</i> suspected (also culture urethra, cervix, or anal canal as indicated).
Adults with comorbid disease (rheumatoid arthritis, human immunodeficiency virus, cancer) or injection drug users	<i>Staphylococcus</i> , gram-negative bacilli	Vancomycin, 15 milligrams/kilogram IV load, <i>plus</i> ceftriaxone, 1 gram IV, <i>or</i> ciprofloxacin, 400 milligrams IV; imipenem, 500 milligrams IV, may be used as an alternative agent.
Sickle cell patients	<i>Salmonella</i> (increasingly <i>Staphylococcus</i> )	Vancomycin, 15 milligrams/kilogram IV load, <i>plus</i> ciprofloxacin, 400 milligrams IV; imipenem, 500 milligrams IV, may be used as an alternative agent.

\*Recommendations differ from the 2006 British Society of Rheumatology treatment guidelines primarily due to the current need to empirically treat methicillin-resistant *Staphylococcus aureus*, which has been shown to be an increasing cause of bacterial arthritis, the most common in some regions.

**TABLE 180-3** Commonly Encountered Organisms in Septic Arthritis in Adolescents and Adults\*

*Traumatic hemarthrosis* is associated with intraarticular fracture or ligamentous injury. Aspiration of large effusions may decrease pain and increase range of motion. Treatment is supportive. Spontaneous hemarthrosis may be associated with coagulopathies requiring specific clotting factor replacement. It is usually not recommended to aspirate spontaneous hemarthroses.

*Crystal-induced synovitis* generally affects middle-age to elderly patients. Gout (uric acid crystals) typically affects the great toe, tarsal joints, and knee, whereas pseudogout (calcium pyrophosphate crystals) typically affects the knee, wrist, ankle, and elbow. Pain with gout usually evolves over hours, whereas the pain associated with pseudogout occurs over a day or more. Either condition may be precipitated by trauma, surgery, significant illness, dietary or alcohol indiscretions, or certain medications. The synovial fluid is inflammatory with negative birefringent needle-shaped (gout) or weakly positive birefringent rhomboid (pseudogout) crystals. Treatment is with nonsteroidal anti-inflammatory drugs (eg, oral indomethacin 50 mg 3 times daily for 3 to 5 days) and opioid analgesics. Although not routinely necessary, colchicine (0.6 milligram/h PO until resolution of symptoms or intolerable gastrointestinal side effects) may be used as a complementary therapy.

*Osteoarthritis* is a chronic, symmetric, polyarticular destruction of joints (including the distal interphalangeal) distinguished by a lack of constitutional symptoms. Patients may present with acute monoarticular exacerbations with small, noninflammatory synovial fluid collections and characteristic joint space narrowing on radiographs. Treatment involves rest and analgesics.

*Lyme arthritis* is a monoarticular or symmetric oligoarticular arthritis (especially of the large joints) with brief exacerbations followed by complete remission occurring weeks to years after the primary infection. Synovial fluid is inflammatory with usually negative cultures. Treatment with appropriate antibiotics (doxycycline, erythromycin, or amoxicillin) for 3 to 4 weeks is effective.

*Gonococcal arthritis* is an immune-mediated infectious arthritis that typically affects adolescents and young adults. Fever, chills, and a migratory tenosynovitis or arthralgias typically precede mono- or oligoarthritis. Vesiculopustular lesions on the distal extremities are characteristic. Synovial fluid is usually inflammatory and often culture negative; cultures of the pharynx, urethra, cervix, and rectum increase the culture yield. The patient should be admitted for pain control and parenteral antibiotic therapy. Orthopedic consultation is advised.

*Reiter syndrome* is a seronegative reactive spondyloarthropathy characterized by acute asymmetric oligoarthritis (especially of the lower

extremities) preceded 2 to 6 weeks by an infectious illness such as urethritis (*Ureaplasma* or *Chlamydia*) or enteric infection (*Salmonella* or *Shigella*). The classic triad of arthritis, conjunctivitis, and urethritis is not required for diagnosis. Synovial fluid is inflammatory. Treatment is symptomatic, and antibiotics have not been found to be useful.

*Ankylosing spondylitis* is a seronegative spondyloarthropathy that primarily affects the spine and pelvis and may be associated with morning stiffness and constitutional symptoms such as fatigue and weakness. Hereditary predilection (HLA-B27 antigen or absence of rheumatoid factor) is significant. Radiographic findings include sacroiliitis and squaring of the vertebral bodies (bamboo spine). Treatment is symptomatic.

*Rheumatoid arthritis* is a chronic, symmetric, polyarticular joint disease (with sparing of the distal interphalangeal) associated with morning stiffness, depression, fatigue, and generalized myalgias. Pericarditis, myocarditis, pleural effusion, pneumonitis, and mononeuritis multiplex may occur. Synovial fluid is inflammatory. Treatment of an acute exacerbation involves immobilization, nonsteroidal anti-inflammatory drugs, and, occasionally, corticosteroids. Antimalarials, gold, and methotrexate are used for long-term therapy.

*Bursitis* refers to an inflammatory process involving any of the more than 150 bursae throughout the human body and may be caused by infection, trauma, rheumatologic diseases, or crystal deposition. Certain repetitive activities also may precipitate bursitis: “carpet layer’s knee” (prepatellar bursitis) or “student’s elbow” (olecranon bursitis). A suspicion for septic bursitis, especially in olecranon bursitis, necessitates aspiration of bursal fluid. Septic bursal fluid characteristically is purulent in appearance, with more than  $1500 \text{ mm}^3$  (mean 75,000, typically  $> 30,000$  leukocytes/ $\text{mm}^3$ ) and positive culture. Treatment principles include drainage, rest, compressive dressing, analgesics, and antibiotics for septic bursitis. Septic bursitis generally responds well to oral antibiotics, with emphasis on coverage of *Staphylococcus* (including MRSA) and *Streptococcus* species.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 281, “Acute Disorders of the Joint and Bursae,” by John H. Burton.

# CHAPTER 181

## Emergencies in Systemic Rheumatic Diseases

Michael P. Kefer

Morbidity and mortality in rheumatic disease usually involves multiple organ systems and results from the disease, its complications, and/or its treatment ([Table 181-1](#)).

Disorder	Common Clinical Features and Complications
Anti-phospholipid syndrome	Multiple and recurrent venous and arterial thromboses, recurrent abortions. Secondary form is associated with SLE, RA, systemic sclerosis, and Sjögren syndrome. Thrombophlebitis and DVT, pulmonary embolism, thrombocytopenia, hemolytic anemia, livedo reticularis, stroke, transient ischemic attack, eye vascular complications. Coronary, renal, mesenteric and stroke, ARDS.
Ankylosing spondylitis	Chronic inflammatory disease of the axial skeleton, with progressive stiffness of the spine. Young adults (peak at 20 and 30 years). Back pain (improves with exercise), buttock, hip, or shoulder pain, systemic complaints (fever, malaise, fatigue, weight loss, myalgias), uveitis, restrictive pulmonary failure due to costovertebral rigidity, renal impairment, fracture of the ankylosed spine, acute spinal cord compression.
Adult still disease	Inflammatory disorder. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), arthritis, myalgia, evanescent rash, pharyngitis, lymphadenopathy, splenomegaly, anemia, thrombocytopenia. Pericarditis, myocarditis, pleurisy, ARDS, arrhythmias, heart and liver failure.
Behçet disease	Chronic, relapsing, inflammatory disease. Systemic vasculitis involving arteries and veins of all sizes (carotid, pulmonary, aortic, and inferior extremities vessels are most commonly involved, with aneurysm, dissection, rupture, or thrombosis). Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), recurrent painful skin and mucosal lesions; asymmetric, nondeforming arthritis of the medium and large joints; thrombophlebitis and DVT; ocular complications. Neuropsychiatric manifestations. Pericarditis, myocarditis, bowel perforation.

<b>Churg-Strauss syndrome</b>	Vasculitis with a multisystemic involvement. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), allergic rhinitis, recurrent sinusitis, asthma, and peripheral blood eosinophilia. Systemic hypertension, pericarditis, abdominal pain, peripheral neuropathy; skin lesions, AMI, bowel perforation.
<b>Dermatomyositis/polymyositis</b>	Idiopathic inflammatory myopathies. Muscle weakness, myalgia and muscle tenderness. Elevated serum CK. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), Raynaud phenomenon, nonerosive inflammatory polyarthritis, esophageal dysfunction, respiratory failure, aspiration lung infections, conduction disturbances.
<b>Giant cell arteritis (temporal arteritis)</b>	Chronic vasculitis of large and medium sized vessels. Elderly (mean age at diagnosis: 70 years). Localized headache of new onset, tenderness of the temporal artery, and biopsy revealing a necrotizing arteritis. Temporal artery may be normal on clinical examination. Gradual onset, systemic complaints, jaw or tongue claudication, eye complaints and visual loss. Aortic regurgitation and aortic arch syndrome. Neurologic complications due to carotid and vertebrobasilar vasculitis.
<b>Henoch-Schönlein purpura</b>	Systemic vasculitis associated with IgA deposition, generally in children. Palpable purpura (in patients with neither thrombocytopenia nor coagulopathy), arthritis/arthralgia, abdominal pain, GI bleeding, and renal impairment (adult).
<b>Microscopic polyangiitis</b>	Small-vessel systemic vasculitis, characterized by rapidly progressive glomerulonephritis and pulmonary involvement. Lung complications differentiate microscopic polyangiitis from polyarteritis nodosa. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), arthralgias, skin lesions, hemoptysis, abdominal pain, renal impairment, systemic hypertension.
<b>Polyarteritis nodosa</b>	Systemic necrotizing vasculitis of the medium-sized muscular arteries. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), arthralgias, skin lesions, abdominal pain, renal impairment, systemic hypertension, peripheral mononeuropathy typically with both motor and sensory deficits, eye complications, leucocytosis and anemia, stroke, mesenteric ischemia, acute scrotum.
<b>Rheumatoid arthritis</b>	Chronic, systemic, inflammatory disorder. Symmetrical and potentially destructive arthritis. Systemic symptoms (fever, malaise, fatigue, weight loss, myalgia), skin lesions, splenomegaly. Cervical spine involvement, pleuritis, pericarditis, myocarditis, and aortitis. Cricoarytenoid arthritis with potential for airway obstruction. Ocular involvement. Peripheral artery disease, Sjögren syndrome, vasculitis, and renal impairment. Abdominal pain. Anemia, leucopenia, thrombocytosis, and Felty syndrome. ACS, respiratory failure.
<b>Polymyalgia rheumatica</b>	Immune-mediated condition. Ears (violaceous and erythematous auricula), nose (saddle nose deformity), and other cartilaginous structures inflammation (especially eyes, joints, and respiratory tract). One-third of cases associated with another SRD. Sternoclavicular, costochondral, and manubriosternal arthritis, upper airway involvement, aortic or mitral valvular regurgitation, pericarditis, renal impairment, peripheral neuropathies, ocular complications.

<b>Systemic lupus erythematosus</b>	Systemic autoimmune disease, characterized by relapses and remissions, and affecting virtually every organ. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), symmetric and polyarticular arthritis (small joints of the hands, the wrists and the knees), butterfly rash, mucocutaneous manifestations, oral and/or nasal ulcers, Raynaud phenomenon. Neuropsychiatric manifestations, pleurisy, lupus pneumonitis, shrinking or vanishing lung syndrome, and pulmonary hypertension. Libman-Sacks endocarditis, pericarditis, myocarditis, endocarditis. GI unspecific complaints. Renal impairment, leucopenia, mild anemia, and thrombocytopenia. Ocular complications, ACS.
<b>Sjögren syndrome</b>	Autoimmune disease. May be primary; secondary form is mostly associated with RA, SLE, polymyositis, or dermatomyositis. Systemic symptoms, arthralgia, skin lesions, Raynaud phenomenon. Pulmonary hypertension, pericarditis, neuropsychiatric manifestations, peripheral neuropathy, hepatic abnormalities, renal impairment, hypokalemic respiratory arrest, stroke, pulmonary embolism, transverse myelitis.
<b>Systemic sclerosis (scleroderma)</b>	Inappropriate and excessive accumulation of collagen in a variety of tissues; widespread vascular lesions with vascular spasm, thickening of the vascular wall and narrowing of the lumen. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), skin lesions (fingers, hands and face), carpal tunnel syndrome, Raynaud phenomenon. Renal impairment, GI dysmotility, gastroesophageal reflux (aspiration pneumonitis), chronic esophagitis and stricture formation. Vascular ectasia in the stomach ("watermelon stomach"). Alveolar hemorrhage, ARDS, arrhythmias, scleroderma renal crisis.
<b>Takayasu arteritis</b>	Chronic vasculitis, young women, predominantly Asians. Systemic complaints (fever, malaise, fatigue, weight loss, myalgia), arthralgias, skin lesions, abdominal pain and diarrhea. Aorta and its primary branches, and pulmonary artery involvement. Neurologic manifestations, syncope, subclavian steal syndrome, extremities ischemia. Normochromic, normocytic anemia, ACS, bowel ischemia and perforation, stroke.
<b>Wegener granulomatosis</b>	Multiple organ system vasculitis and necrotizing granulomas. Respiratory tract manifestations in approximately 100% cases, with nose, oral cavity, upper trachea, external and middle ear, and orbits, inflammations. Upper airway and pulmonary manifestations. Constitutional symptoms, arthralgias, glomerulonephritis and small vessel vasculitis (scleritis and episcleritis, palpable purpura or cutaneous nodules, peripheral neuropathy, deafness). Pericarditis, myocarditis. Renal impairment. Anemia, leukocytosis and thrombocytosis, ACS.

Key: ACS = acute coronary syndrome; AMI = acute myocardial infarction; AH = alveolar hemorrhage; ARDS = adult respiratory distress syndrome; DVT = deep vein thrombosis; GI = gastrointestinal; RA = rheumatoid arthritis; SLE = systemic lupus erythematosus; SRD = systemic rheumatic disease.

**TABLE 181-1** Common Features and Complications of Systemic Rheumatic Diseases

## AIRWAY

Relapsing polychondritis involves the tracheobronchial cartilage in approximately 50% of cases. Hoarseness and throat tenderness over the cartilage are noted. Repeated attacks can lead to airway collapse. Rheumatoid arthritis (RA) may involve the cricoarytenoid joints causing pain with speaking, hoarseness, or stridor. The cricoarytenoid joints may fix in a closed position which may mandate emergency tracheostomy. Anticipate difficult endotracheal intubation from temporomandibular joint dysfunction, atlantoaxial instability, or cervical ankylosis.

## **RESPIRATORY MUSCLE**

Dermatomyositis and polymyositis may lead to respiratory failure from respiratory muscle involvement in poorly controlled disease.

## **LUNG**

Alveolar hemorrhage complicates Goodpasture disease, systemic lupus erythematosus (SLE), Wegener granulomatosis, and other vasculitic conditions. Pulmonary fibrosis complicates ankylosing spondylitis, scleroderma, and other conditions. Pleural effusion complicates RA and SLE.

## **HEART**

Pericarditis occurs in RA and SLE. Myocardial infarction may occur from coronary artery involvement in Kawasaki disease or polyarteritis nodosa. Pancarditis occurs in acute rheumatic fever. Valvular heart disease occurs in ankylosing spondylitis, relapsing polychondritis, and rheumatic fever. Involvement may extend into the conduction system causing arrhythmias.

## **NERVOUS SYSTEM**

Patients with rheumatologic involvement of the cervical spine may be at high risk for serious cervical spine or spinal cord injury from otherwise trivial trauma, such as manipulation during endotracheal intubation, if extreme caution is not exercised. Ligamentous destruction of the transverse ligament of C-2, with resultant symptoms of cord compression, may complicate RA. Cervical spine inflexibility from ankylosing spondylitis predisposes to injury out of proportion to the mechanism. Vasculitis, aortic dissection, or thromboembolism may result in anterior spinal artery syndrome.

## **EYE**

Symptoms range from mild irritation to complete blindness. Temporal arteritis is a cause of sudden blindness and should be considered in any patient older than 50 years who presents with new onset headache, visual change, or jaw claudication. Dry eyes (and dry mouth) from Sjögren syndrome may occur alone or in combination with many rheumatologic conditions. Scleritis occurs in patients with RA and presents with marked ocular tenderness. Episcleritis, keratitis, and uveitis may occur.

## **KIDNEY**

Nephritis is a common complication of SLE, Wegener granulomatosis, and systemic vasculitis. Renal dysfunction can result from malignant hypertension as occurs with scleroderma, from rhabdomyolysis in the patient with florid myositis, or from nonsteroidal anti-inflammatory drugs used for treatment. Nephrotic syndrome in patients with SLE predisposes to renal vein thrombosis.

## **HYPERTENSION**



Hypertension can complicate any rheumatologic condition that affects the kidneys directly, as in polyarteritis nodosa, scleroderma, or SLE, or indirectly from nephrotoxic drugs used to treat the underlying disorder.

## **ADRENAL GLANDS**

Patients with steroid dependent disease are at risk for acute adrenal insufficiency from unexpected stressors or abrupt withdrawal.

## **BLOOD**

Anemia and thrombocytopenia are common. Many medications used for treatment are potent immunosuppressants.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 279, “Emergencies in Systemic Rheumatic Diseases,” by Gemma C. Morabito and Bruno Tartaglino.

# CHAPTER 182

## Infectious and Noninfectious Disorders of the Hand

Michael P. Kefer

Rest, elevation, and draining any pus are the mainstays of treatment for many conditions of the hand. This helps to decrease inflammation, avoid secondary injury, and prevent spread of any existing infection. The optimal position for splinting is the position of function: wrist in 15° extension, metacarpal-phalangeal (MCP) joint in 50° to 90° flexion, proximal interphalangeal (PIP) joint in 10° to 15° flexion, and distal interphalangeal (DIP) joint in 10° to 15° flexion.

### HAND INFECTIONS

**Cellulitis** is a superficial infection presenting with localized warmth, erythema, and edema. The absence of tenderness on deep palpation and range of motion helps exclude deep space involvement.

**Flexor tenosynovitis** is a surgical emergency diagnosed on examination ([Table 182-1](#)).

Percussion tenderness	Tenderness over the entire length of the flexor tendon sheath
Uniform swelling	Symmetric finger swelling along the length of the tendon sheath
Intense pain	Intense pain with passive extension
Flexion posture	Flexed posture of the involved digit at rest to minimize pain

**TABLE 182-1** Kanavel 4 Cardinal Signs of Flexor Tenosynovitis

**Deep space infections** involve the web or midpalmar space. Web space infection presents as dorsal and volar swelling of the web space causing separation of the affected digits. Midpalmar space infection occurs from spread of a flexor tenosynovitis or penetrating wound to the palm causing infection of the radial or ulnar bursa of the hand.

**Closed fist injury** is essentially a human bite wound to the MCP joint that results from punching an individual in the teeth. Risk of infection spreading along the extensor tendons is high. Wounds penetrating the skin should be explored, irrigated, and allowed to heal by secondary intention. When inspecting for extensor tendon injury, it is essential to consider the position of the hand at the time of injury. Extensor tendon repair is delayed until the risk of infection has passed.

**Paronychia** is an infection of the lateral nail fold. If there is no pus, treat with warm soaks, elevation, and antibiotics if warranted. A simple paronychia is drained by lifting the nail fold with a needle or number 11 blade to drain the abscess. If pus is seen beneath the nail, a portion of the nail may have to be removed and packing placed for adequate drainage. Avoid injury to the nail bed. Recheck the wound in 24 to 48 hours, pull the packing, and begin warm soaks.

**Felon** is an infection of the pulp space of the fingertip. Incision and drainage are by the lateral approach to protect the neurovascular bundle. The incision should remain within the borders of the DIP joint crease proximally and the base of the phalangeal tuft distally. Incise deep enough to extend across the entire finger pad to divide the septae at the bony insertions. Unless there is a pointing abscess, the radial aspect of the index and middle fingers and the ulnar aspect of the thumb and small finger, should be avoided. If there is a pointing volar abscess, a longitudinal volar incision is used. Pack the wound. Splint the hand in the position of function. Recheck in 24 to 48 hours, pull the packing, and begin warm soaks.

**Herpetic whitlow** is a viral infection of the fingertip. Clinically, it may present similar to a felon, but vesicles are present. Immobilize, elevate, and protect with a dry dressing to prevent transmission. Antiviral agents may shorten the duration.

[Table 182-2](#) summarizes recommended antibiotic therapy for common hand infections.

Infection	Initial Antimicrobial Agent(s)/Comments
Cellulitis	<p><i>For mild to moderate cellulitis:</i> TMP/SMX double strength, 1 to 2 tablets twice per day PO for 7 to 10 days.  <i>Plus/minus</i> cephalexin, 500 milligrams PO 4 times per day for 7 to 10 days, <i>or</i> dicloxacillin, 500 milligrams PO 4 times daily for 7 to 10 days. <i>Clindamycin is an option depending on local resistance.</i></p> <p><i>For severe cellulitis/injection drug abusers:</i> vancomycin, 1 gram IV every 12 h.</p>
Felon/paronychia	<p>TMP/SMX double strength, 1 to 2 tablets twice per day PO for 7 to 10 days.  <i>Plus/minus</i> cephalexin, 500 milligrams PO 4 times per day for 7 to 10 days, <i>or</i> dicloxacillin, 500 milligrams PO 4 times daily for 7 to 10 days.  <i>Consider</i> addition of clindamycin or amoxicillin-clavulanate to TMP/SMX (rather than cephalexin) if anaerobic bacteria are suspected.</p>
Flexor tenosynovitis	<p>Ampicillin-sulbactam, 1.5 grams IV every 6 h, <i>or</i> ceftiofur, 2 grams IV every 8 h, <i>or</i> piperacillin/tazobactam, 3.375 grams IV every 6 h.  <i>Plus:</i> vancomycin, 1 gram IV every 12 h, if MRSA is prevalent in community.</p>
Deep space infection	<p>Ampicillin-sulbactam, 1.5 grams IV every 6 h, <i>or</i> ceftiofur, 2 grams IV every 8 h, <i>or</i> piperacillin/tazobactam, 3.375 grams IV every 6 h.  <i>Plus:</i> vancomycin, 1 gram IV every 12 h, if MRSA is prevalent in community.</p>
Animal bites (including human)	<p>If no visible signs of infection: amoxicillin-clavulanate, 875/125 milligrams PO twice daily for 5 days.  For signs of infection: ampicillin-sulbactam, 1.5 grams IV every 6 h, <i>or</i> ceftiofur, 2 grams IV every 8 h, <i>or</i> piperacillin/tazobactam, 3.375 grams every 6 h.  For penicillin allergy, use clindamycin plus ciprofloxacin.</p>
Herpetic whitlow	<p>Acyclovir, 400 milligrams PO thrice daily for 10 days. (No surgical drainage is indicated.)</p>

Key: TMP/SMX = trimethoprim-sulfamethoxazole.

**TABLE 182-2** Initial Antibiotic Coverage for Common Hand Infections

## NONINFECTIOUS CONDITIONS

**Tendonitis and tenosynovitis** are usually due to overuse. Examination reveals tenderness over the involved tendon. Treat with immobilization and nonsteroidal anti-inflammatory drugs (NSAIDs).

**Trigger finger** is a tenosynovitis of the flexor sheath where inflammation or scarring results in impingement and snap release of the tendon. This occurs as the finger is extended from a flexed position. Steroid injection may be effective in early stages. Definitive treatment is surgery.

**DeQuervain tenosynovitis** involves the extensor pollicis brevis and abductor pollicis tendons. Pain occurs at the radial aspect of the wrist and radiates into the forearm. Finkelstein test is diagnostic: the patient grasps the thumb in the fist and deviates the hand ulnarly, reproducing the pain. Treat with a thumb spica splint, NSAIDs, and referral.

**Carpal tunnel syndrome** results from compression of the median nerve by the transverse carpal ligament. The cause is usually edema secondary to overuse, pregnancy, or congestive heart failure. Pain in the median nerve distribution tends to be worse at night. On examination, pain may be reproduced by tapping over the nerve at the wrist (Tinel sign) or by holding the wrist flexed maximally for > 1 min (Phelan sign). Treat with a wrist splint, NSAIDs, and referral.

**Dupuytren contracture** results from fibrous changes in the subcutaneous tissues of the palm, which may lead to tethering and joint contractures. Refer to a hand surgeon.

**Ganglion cyst** is a cystic collection of synovial fluid from a joint capsule or tendon sheath. Treat with NSAIDs and referral.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 280, “Nontraumatic Disorders of the Hand,” by Carl A. Germann and Mark W. Foure.

## **CHAPTER 183**

# **Soft Tissue Problems of the Foot**

Robert L. Cloutier

## **CORNS AND CALLUSES**

Calluses represent a dermatologic reaction to focal pressure. Ongoing pressure results in calluses developing into corns. Corns can be differentiated from warts when incised; warts will bleed, corns will not. The differential diagnosis includes syphilis, psoriasis, lichen planus, rosacea, arsenic poisoning, basal cell nevus syndrome, and malignancy. Treatment for corns is paring with a No. 15 blade to include removal of central keratin plug.

## **PLANTAR WARTS**

Plantar warts are common, contagious, and caused by the human papillomavirus. The diagnosis is clinical and the differential diagnosis includes corns and undiagnosed melanoma. Topical treatment with 15% to 20% salicylic acid is most effective. Nonhealing lesions should be referred to a dermatologist or podiatrist.

## **ONYCHOCRYPTOSIS (INGROWN TOENAIL)**

Onychocryptosis is characterized by increased inflammation or infection of the lateral or medial aspects of the toenail. This occurs when the nail plate penetrates the nail sulcus and subcutaneous tissue (usually in the great toe). Patients with underlying diabetes, arterial insufficiency, cellulitis, ulceration, or necrosis are at risk for amputation if treatment is delayed.

Treatment depends on the type of inflammation. If toenail is uninfected, sufficient results will often be obtained with elevation of the nail with a wisp of cotton between the nail plate and the skin, daily foot soaks, and avoidance of pressure on the area. A second option (requiring digital block) is to remove a spicule of the nail and debride the nail groove. If granulation tissue or infection is present, partial removal of the nail is indicated. If the toenail is infected perform digital block and cut one-fourth or less of the nail with a longitudinal incision (including beneath the cuticle). A nonadherent bulky dressing should be placed, and the wound should be checked in 24 to 48 hours.

## **BURSITIS**

Pathologic bursae of the foot are categorized as follows: (1) noninflammatory, (2) inflammatory, (3) suppurative, and (4) calcified. Noninflammatory bursae become painful as a result of direct pressure, whereas inflammatory bursitis results from gout, syphilis, or rheumatoid arthritis. Suppurative bursitis results from spread of pyogenic organisms (often *Staphylococcus aureus*) from adjacent wounds. Complications include hygroma, calcified bursae, fistula, and ulcer formation. Treatment for severe septic bursitis includes intravenous antibiotics such as nafcillin 500 milligrams qid or oxacillin 500 milligrams qid. For further discussion, see [Chapter 180](#) Acute Disorders of the Joints and Bursae.

## **PLANTAR FASCIITIS**

The plantar fascia is connective tissue anchoring the plantar skin to the bone protecting the arch of the foot. Plantar fasciitis is the most common cause of heel pain due to overuse. Patients have point tenderness over the anterior–medial calcaneus, that is, worse on arising and after activity. The differential diagnosis includes abnormal joint mechanics, poorly cushioned shoes, Achilles tendon pathology, and rheumatoid disease. Treatment includes rest, ice, and nonsteroidal anti-inflammatory drugs (NSAIDs).

Most cases are self-limited. Glucocorticoid injections are *not* indicated in the ED. Severe cases may require a short-leg walking cast and should be referred to a podiatrist or orthopedist.

## **NERVE ENTRAPMENT SYNDROMES**

### **Tarsal Tunnel Syndromes**

Tarsal tunnel syndrome involves heel and foot pain due to compression of the posterior tibial nerve as it courses inferior to the medial malleolus. Causes include running, restrictive footwear, edema of pregnancy, posttraumatic fibrosis, ganglion cysts, osteophytes, and tumors. Pain is worse at night and located at the medial malleolus, the heel, the sole, and the distal calf.

The differential diagnosis includes plantar fasciitis and Achilles tendonitis. Tinel sign is positive, and eversion and dorsiflexion worsen symptoms. The pain of tarsal tunnel syndrome involves the more medial heel and arch and worsens with activity. Ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) may aid in diagnosis. Treatment includes NSAIDs, rest, and possible orthopedic referral.

### **Deep Peroneal Nerve Entrapment**

Entrapment of the deep peroneal nerve occurs most frequently where it courses beneath the extensor retinaculum. Recurrent ankle sprains, soft tissue masses, and restrictive footwear represent the most common causes. Symptoms include dorsal and medial foot pain as well as sensory hypoesthesia at the first web space.

Pain and tenderness can be elicited by plantar flexion in inversion of the foot. Plantar fasciitis should also be considered. Ultrasound, CT, and MRI may aid in diagnosis. Treatment includes NSAIDs, rest, and possible orthopedic referral.



# GANGLIONS

A ganglion is a benign synovial cyst attached to a joint capsule or tendon sheath near the anterolateral ankle. Typically a firm, nontender, cystic lesion is found on examination. The diagnosis is clinical, but MRI or ultra-sound can be used if in doubt. Treatment includes aspiration and injection of glucocorticoids, but most require surgical excision.

# TENDON LESIONS

**Tenosynovitis and Tendinitis:** Tenosynovitis or tendonitis are usually due to overuse, and present with pain over the involved tendon. Treatment includes ice, rest, and NSAIDs.

**Tendon Lacerations:** Tendon lacerations should be explored and repaired if the ends are visible in the wound. Due to the high complication rate, specialty consultation is recommended. After repair, extensor tendons are immobilized in dorsiflexion and flexor tendons in equinus.

**Tendon Ruptures:** *Achilles tendon* rupture presents with pain and a palpable defect in the area of the tendon. Patients have an inability to stand on tiptoes, and an absence of plantar flexion with squeezing of the calf (Thompson test). Treatment is generally surgical in younger patients and conservative (casting in equinus) in the elderly. *Anterior tibialis tendon* rupture results in a palpable defect and mild foot drop. *Posterior tibialis tendon* rupture is usually chronic and presents with a flattened arch and swelling over the medial ankle. Examination may show weakness on inversion, a palpable defect, and inability to stand on tiptoes. *Flexor hallucis longus* rupture presents with loss of plantar flexion of the great toe and must be surgically repaired in athletes. *Disruption of the peroneal retinaculum* occurs after a direct blow during dorsiflexion, and causes localized pain behind the lateral malleolus; there is clicking during walking, as the tendon is subluxed. Treatment is surgical.

## **PLANTAR INTERDIGITAL NEUROMA (MORTON NEUROMA)**

Neuromas are thought to occur from entrapment of the plantar digital nerve due to tight-fitting shoes; the third interspace is most commonly affected. Patients often present with burning, cramping, or aching over the affected metatarsal head. Diagnosis is clinical, but ultrasound or MRI may be helpful. Conservative treatment includes wide shoes and glucocorticoid injections. Local glucocorticoid injections may be curative. Surgical neurolysis is occasionally required.

## **COMPARTMENT SYNDROMES OF THE FOOT**

The foot has 9 compartments. Compartment syndromes in the foot are associated with high-energy crush injuries. Other causes include bleeding disorders and postischemic swelling after arterial injury, ankle fractures, burns, and chronic overuse. Patients should be considered at-risk if there is increasingly severe pain exacerbated with active and passive motion, coupled with paresthesias or neurovascular deficits.

At-risk patients must have compartment pressures checked. Any difference of less than 30 mm Hg between the Stryker STIC Device (Stryker, Kalamazoo, MI) and diastolic blood pressure is considered positive. Prompt consideration of emergent fasciotomy.

## **PLANTAR FIBROMATOSIS**

Plantar fibromatosis (Dupuytren contracture of the plantar fascia) involves small, asymptomatic, palpable, slowly growing, firm masses on the plantar surface of a (nonweightbearing) foot. MRI may be helpful for diagnosis.

Toe contractures do not occur. Lesions tend to reabsorb spontaneously, and treatment is conservative.

## **MALIGNANT MELANOMA**

Melanoma of the foot, which accounts for 15% of all cutaneous melanomas, may present as atypical nonpigmented or pigmented lesions; the nail may be included. Vigilance is key as these lesions often mimic more benign conditions. The differential diagnosis includes fungal infections, plantar warts, and foot ulcers. All atypical or nonhealing lesions should be sent for biopsy.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 282, “Soft Tissue Problems of the Foot,” by Franz R. Melio.

## **SECTION 21 Psychosocial Disorders**

## **CHAPTER 184**

# **Clinical Features of Behavioral Disorders**

Lance H. Hoffman

## **PSYCHIATRIC SYNDROMES (AXIS I DISORDERS)**

### **Dementia**

Dementia is a disorder consisting of a pervasive disturbance in cognition that impairs memory, abstraction, judgment, personality, and higher critical functions such as language. Its onset is typically gradual, and the patient's normal level of consciousness is maintained. The presence of global cognitive impairment can be detected by using a bedside screening test such as the Mini-Mental State Exam. Potentially reversible causes of dementia include metabolic and endocrine disorders, adverse drug effects and interactions, and depression.

### **Delirium**

Delirium is characterized by acute development of impairment in cognitive function, diminished level of consciousness, inattention, and sensory misperceptions that fluctuate over the course of hours. Visual hallucinations are common. The causes of delirium are frequently treatable and include infection, electrolyte abnormalities, toxic ingestions, and head injury. Treatment is directed toward correcting the underlying cause.

### **Amnestic Disorders**

Amnesic patients cannot learn new information or recall previously learned information. Amnesia may be due to brain trauma, stroke, anoxic brain injury, substance abuse, and chronic nutritional deficiencies.

## **Substance-Induced Disorders**

Intoxication is an exogenous substance-induced syndrome that results in maladaptive behavior and impaired cognitive functioning and psychomotor activity. Judgment, perception, attention, and emotional control may be affected. Repeated use (substance abuse) may lead to physical or psychological dependence. Substance withdrawal symptoms may develop when the amount ingested is reduced or stopped. Symptoms and timing of withdrawal depend on the substance of abuse.

## **Schizophrenia and Other Psychotic Disorders**

Schizophrenia is a chronic disease characterized by functional deterioration. Clinical features include “positive symptoms” such as hallucinations, delusions, disorganized speech or behavior, or catatonic behavior, “negative symptoms” such as blunted affect, emotional withdrawal, lack of spontaneity, anhedonia, or impaired attention, as well as cognitive impairment with loose associations or incoherence and the relative absence of a mood disorder. Patients may present to the emergency department for worsening psychosis, suicidal ideations, bizarre or violent behavior, or adverse medication events. Older antipsychotic medications, such as haloperidol, effectively treat the “positive symptoms,” and newer antipsychotic medications, such as aripiprazole, quetiapine, olanzapine, risperidone, ziprasidone, and clozapine, effectively treat “positive” and “negative” symptoms. The diagnosis of schizophreniform disorder is made when an individual experiences symptoms and demonstrates signs consistent with schizophrenia for less than 6 months. A brief psychotic disorder is a psychosis that lasts less than 4 weeks in response to a

traumatic life experience, such as sexual assault, natural disaster, combat, or death of a loved one.

## **Mood Disorders**

### ***Major Depression***

Major depression is characterized by a persistent dysphoric mood or a pervasive loss of interest and pleasure in usual activities (anhedonia) that lasts longer than 2 weeks. Associated psychological symptoms include feelings of guilt over past events, self-reproach, worthlessness, hopelessness, and recurrent thoughts of death or suicide. Physiologic symptoms include loss of appetite and weight, sleep disturbances, fatigue, inability to concentrate, and psychomotor agitation or retardation. The diagnosis should be entertained in any patient presenting with multiple vague complaints. The lifetime risk of suicide in patients with this disorder is 15%. Consequently, all patients suspected of having major depression should be questioned about suicidal thoughts. An appropriate psychiatric consultation and referral are needed for those with suicidal thoughts.

### ***Bipolar Disorder***

Bipolar disorder is characterized by recurrent, cyclic episodes of manic and depressive symptoms, with depressive episodes being more common than manic episodes. Manic individuals experience an elated mood that can quickly deteriorate to irritability and hostility should their expectations not be met. They appear energetic and expansive, with a decreased need for sleep, poor impulse control, racing thoughts, and pressured speech. They have grandiose ideas. Complications include suicide, substance abuse, and marital and occupational disruptions. Valproic acid and lithium salts are commonly prescribed as chronic mood stabilizers.

## **Anxiety Disorders**

Panic disorder consists of recurrent episodes of severe anxiety and sudden, extreme autonomic symptoms. It is a diagnosis of exclusion as these symptoms can also occur in life-threatening cardiovascular and pulmonary disorders. Benzodiazepines, such as alprazolam and lorazepam, are effective in treating acute episodes of anxiety. The diagnosis of generalized anxiety disorder can be made when a patient experiences persistent worry or tension without discrete panic attacks for at least 6 months. Phobias consist of symptoms of anxiety, recognized as excessive by the person, prompted by the exposure to, or the anticipated exposure to, a specific stimulus. Posttraumatic stress disorder is an anxiety reaction to a severe, psychosocial stressor, typically perceived as life threatening. The individual experiences repetitive, intrusive memories of the event. Nightmares, feelings of guilt and depression, and substance abuse are common. Individuals with obsessive-compulsive disorder experience intrusive thoughts or images that create anxiety (obsessions). To control these thoughts and anxiety, the individual engages in repetitive behaviors or rituals (compulsions).

## **PERSONALITY DISORDERS (AXIS II DISORDERS)**

Individuals with a personality disorder exhibit a lifelong pattern of maladaptive behavior that is not limited to periods of illness. Personality disorders include paranoid, schizoid, schizotypal, antisocial, borderline, histrionic, narcissistic, avoidant, dependent, and obsessive-compulsive.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 284, "Behavioral Disorders: Diagnostic Criteria," by Leslie Zun.



## **CHAPTER 185**

# **Emergency Assessment and Stabilization of Behavioral Disorders**

Lance H. Hoffman

In the last 2 decades, mental health-related visits to emergency departments (ED) in the United States increased 38% to 23.6 per 1000 population.

## **CLINICAL FEATURES**

Suicidal patients are often forthcoming about their intentions for self-harm. The patient's intentions may be more difficult to infer after a nontraumatic suicide attempt and if the patient has an altered level of consciousness than after a self-inflicted traumatic injury. Patients with multiple seemingly unrelated somatic complaints may be depressed. High-and low-risk profiles for suicide risk are summarized in [Table 185-1](#). Medication overdose is the most common type of suicide attempt.

<b>Demographic, Health, and Social Profile</b>	<b>High Risk</b>	<b>Lower Risk</b>
Gender	Male	Female
Marital status	Separated, divorced, or widowed	Married
Family history	Chaotic, conflictual Family history of suicide	Stable No family history of suicide
Job	Recently unemployed	Employed
Relationships	Recent conflict or loss of a relationship	Stable relationships
School	In disciplinary trouble	No disciplinary problems
Religion	Weak or no suicide taboo	Strong taboo against suicide
Health		
Physical	Acute or chronic, progressive illness Excessive drug or alcohol use	Good health Little or no drug or alcohol use
Mental	Depression (SIG E CAPS + MOOD)* History of schizophrenia or bipolar disorder Panic disorder Antisocial or disruptive behavior Feelings of helplessness or hopelessness Few, weak reasons for living Unstable, inappropriate affect	No depression No psychosis Minimal anxiety Directable, oriented Has hope, optimism Good, strong reasons for living Appropriate affect

Suicidal ideation	Frequent, intense, prolonged, pervasive	Infrequent, low intensity, transient
Suicide attempts	Repeated attempts Realistic plan, including access to means Previous attempt(s) planned Rescue unlikely Lethal method Guilt  Unambiguous or continuing wish to die	No prior attempts No plan, lacks access to means  Previous attempt(s) impulsive High likelihood of rescue Method of low lethality Embarrassment about suicide ideation  No previous or continuing wish to die; large appeal component
Relationship with health professional	Lacks insight Poor rapport	Insight Good rapport
Social support	Unsupportive family, friends Socially isolated	Concerned family, friends Socially integrated

\*SIG E CAPS + MOOD is a mnemonic for the eight symptoms of depression plus depressed mood: S = sleep disturbance; I = loss of interest in usual pleasurable activities; G = guilt; E = loss of energy; C = inability to concentrate; A = loss of appetite; P = psychomotor slowing; S = suicidal thoughts, MOOD = depressed mood (ie, "Have you felt blue, down, or depressed most of the day for most days in the last 2 weeks?"). Fulfillment of 5 or more of the 8 items from the list of 8 symptoms indicates the presence of major depression. Symptoms must be present nearly every day for 2 weeks and must include depressed mood or loss of interest or pleasure in activities. Symptoms must represent a change from previous functioning resulting in social, occupational, or other life impairment, and they cannot be the direct result of substance use, a medical condition, or bereavement.

**TABLE 185-1** Evaluation of Suicide Risk in Adults and Adolescents

Homicidal or violent patients tend to pose little diagnostic dilemma to the clinician. They may openly assert their harmful intentions. Their language may contain profanity, escalate in volume, and be rapid or pressured. Mannerisms suggestive of a potentially violent patient include restlessness, pacing in the examination room, clenched fists, acts of violence directed toward inanimate objects in the room, and hypervigilance.

## **DIAGNOSIS AND DIFFERENTIAL**

Differentiating medical (organic) and psychiatric etiologies for abnormal behavior is essential. Evaluation includes a detailed history of present illness, past medical and psychiatric history, medication history, social history, and a physical examination, including a thorough neurologic and mental status examination. Important historical information includes the presence of previous psychiatric illness, fever, head trauma, infections, ingestion of medications or legal and illegal substances, disorientation or

confusion, impaired speech, syncope or loss of consciousness, headaches, and difficulty performing routine tasks. Inquire about suicidal or homicidal intent. Obtain a third-party account of the patient's behavior, changes in behavior and normal level of function.

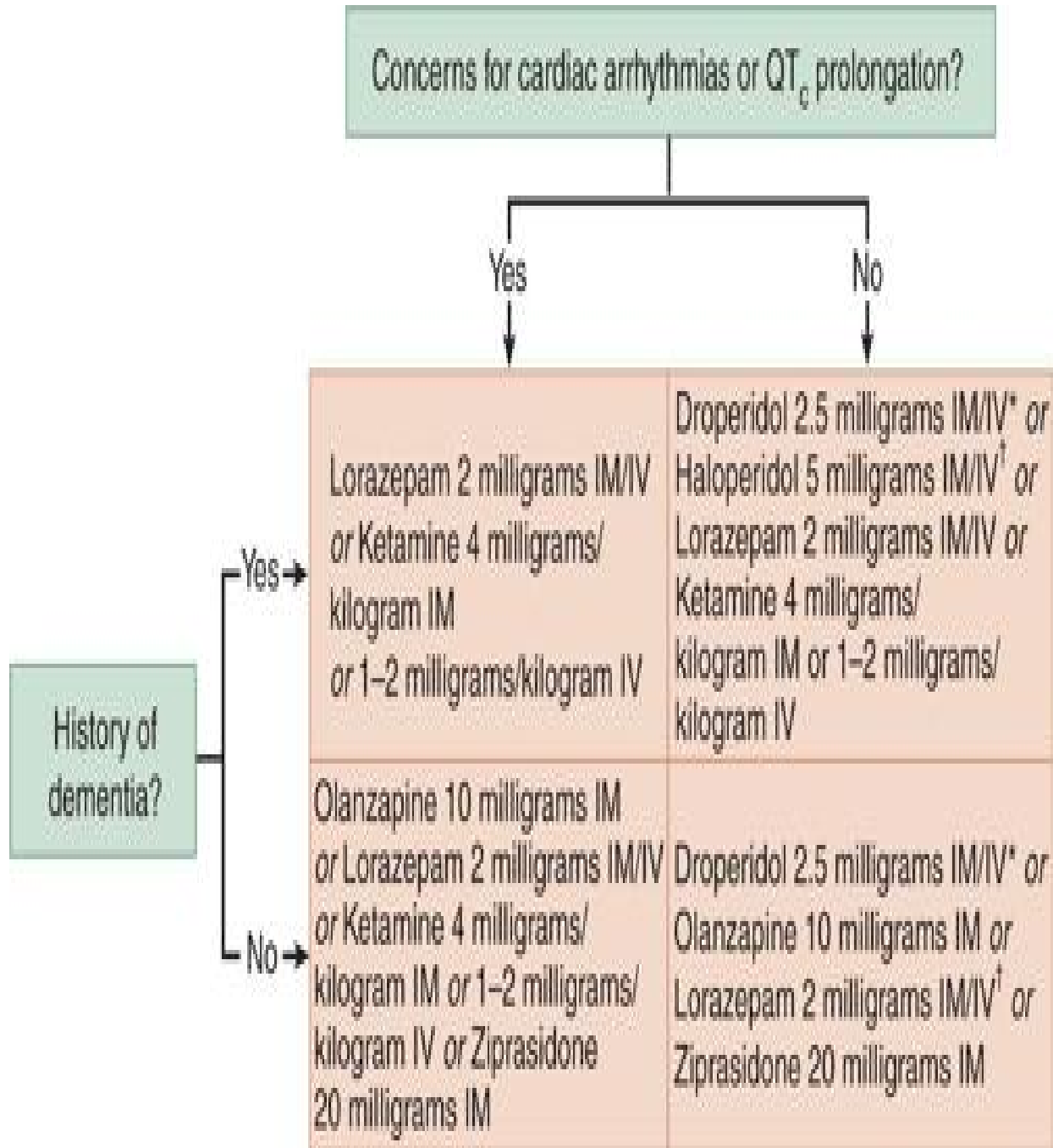
Mental status examination should note physical appearance, affect, orientation, speech pattern, behavior, level of consciousness, attention, language, memory, judgment, thought content, and perceptual abnormalities. Findings such as abnormal vital signs, disorientation with clouded consciousness, abnormal findings on mental status exam, recent memory loss, age > 40 without prior history of psychiatric disease, focal neurologic signs, visual hallucinations, and psychomotor retardation may indicate an organic etiology for the behavior and serve as a guide for further evaluation. Visual hallucinations are more suggestive of a medical etiology, whereas auditory hallucinations support a psychiatric etiology.

Multiple reversible medical conditions can result in the acute onset of a behavioral abnormality. Look for signs of trauma, infection, substance abuse, intoxication or withdrawal, metabolic and endocrine disorders, and disorders of the central nervous system. The patient's laboratory evaluation should be directed toward discovering or confirming suspected abnormalities. Oxygen saturation on room air and glucose levels should be quickly assessed. Additional tests that can be useful depending on the clinical situation include a complete blood count, serum electrolytes, creatinine, hepatic enzymes, T4 level, TSH, ethanol, urinalysis, urine drug screen, pregnancy test, arterial blood gas analysis, cerebrospinal fluid analysis, electrocardiogram, and computed tomography or magnetic resonance imaging of the brain. Salicylate and acetaminophen levels also are useful in the suicidal patient.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

1. The first priority is safety and stabilization, including attention to airway, breathing, and circulation. Contain violent and dangerous psychotic patients to ensure their safety and the safety of everyone else in the ED.

Agitated and violent patients may require sedation and/or restraints to avoid self-injury and harm to nearby individuals. [Figure 185-1](#) suggests an algorithmic approach for safe chemical sedation.



**FIGURE 185-1.** Suggested algorithm for the ED management of patients with acute undifferentiated agitation. \*Droperidol dosing may be repeated if

clinically indicated. Consider reduced dosing in the elderly; lorazepam, 1 milligram IM, haloperidol, 2 milligrams IM, and ketamine, 2 milligrams/kilogram IM.

2. Suicidal, homicidal or violent patients should be disrobed, gowned and searched for potentially dangerous items.
3. Approach the violent patient with a nonthreatening voice, avoid excessive eye contact, keep the exit from the room accessible, and enforce acceptable limits of behavior.
4. Determine if the patient needs to be detained for emergency evaluation.
5. Proceed with medical and psychiatric evaluation. Treat medical conditions as indicated.
6. Assess severity of psychiatric disease and determine the need for psychiatric consultation.
7. Patients judged to be at high risk of harming themselves or others or those who are unable to care for themselves require admission to a psychiatric facility.
8. Disposition decisions for moderate-risk patients are usually made in consultation with a psychiatric consultant. Ensure close psychiatric follow-up and good social support, including someone to stay with the patient, prior to discharge.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 283, “Behavioral Disorders: Emergency Assessment,” by Gregory Luke Larkin and Annette L. Beautrais; and Chapter 285, “Psychotropic Medications and Rapid Tranquilization,” by Marc L. Martel and Michelle H. Biros.

## **CHAPTER 186**

# **Panic and Conversion Disorders**

Lance H. Hoffman

## **PANIC DISORDER**

### **Clinical Features**

The cardinal features of panic disorder are recurrent acute episodes of intense anxiety and fear and persistent worry about having another such episode. Symptoms include palpitations, sweating, shortness of breath, trembling or shaking, choking sensation, chest pain or discomfort, dizziness or light-headedness, paresthesias, chills or hot flashes, fear of losing control, fear of dying, derealization, and depersonalization. Episodes begin unexpectedly; severity peaks within 10 min and symptoms last for up to 1 hour.

### **Diagnosis and Differential**

Panic disorder is a diagnosis of exclusion because its symptoms and signs mimic those of many potentially life-threatening disorders. A thorough history and physical examination, and when indicated other tests, help to rule out these life-threatening disorders. The differential diagnosis of panic attacks is listed in [Table 186-1](#).

Cardiovascular	Neurologic	Drug withdrawal
Angina	Migraine headache	Alcohol
Myocardial infarction	Mènière disease	Barbiturates
Mitral valve prolapse	Complex partial seizures	Benzodiazepines
Congestive heart failure	Transient ischemic attacks	Opiates
Tachyarrhythmias: premature atrial contractions, supraventricular tachycardia	Drug induced	β-Antagonists
Pulmonary	Caffeine	Psychiatric
Hyperventilation	Cocaine	Post-traumatic stress disorder
Asthma	Sympathomimetics	Depressive disorders
Pulmonary embolus	Theophylline	Other anxiety disorders
Endocrine	Thyroid preparations	Psychosocial
Hyperthyroidism	Selective serotonin reuptake inhibitors	Partner violence
Hypoglycemia	Cannabis	Sexual abuse or assault
Hyponatremia	Corticosteroids	Other situational stressors
Pheochromocytoma	β-Agonists	
Carcinoid syndrome	Triptans	
Cushing syndrome	Nicotine	
	Yohimbine	
	Hallucinogens	
	Anticholinergics	

**TABLE 186-1** Medical Differential Diagnosis of Panic Attacks

## Emergency Department Care and Disposition

1. After excluding life-threatening causes of symptoms, educate and reassure patients that panic disorder is an illness that can be treated effectively.
2. Benzodiazepines, such as **alprazolam** 0.25 to 0.5 milligrams PO or **lorazepam** 1 to 2 PO/IV are used to control acute symptoms. Antidepressants, such as selective serotonin reuptake inhibitors and serotonin/ norepinephrine reuptake inhibitors, are preferred for maintenance therapy.
3. Ask patients about suicidal thoughts. Patients who are suicidal or so incapacitated that they cannot care for themselves require psychiatric consultation and hospitalization.
4. Most patients can be discharged. Refer to a psychiatrist for outpatient cognitive behavioral therapy and initiation of pharmacotherapy.

## CONVERSION DISORDER



## Clinical Features

Patients with conversion disorder unconsciously develop symptoms that suggest a physical or neurologic disorder. The symptoms are not consciously produced by the patient, are usually in response to a stressor or conflict, are not limited to pain or sexual dysfunction, and are not explained by a known organic etiology or culturally sanctioned response pattern. Organic disease may be concurrently present.

## Diagnosis and Differential

An organic explanation for the patient's symptoms must be excluded before the diagnosis of conversion disorder can be made. The differential diagnosis is broad and includes stroke, multiple sclerosis, polymyositis, infectious disorders, as well as drug ingestions or poisonings. The examination techniques listed in [Table 186-2](#) may help test for true neurologic deficits.

Function	Technique
Sensation	
Yes/no test	Patient closes eyes and responds <i>yes</i> or <i>no</i> to touch stimulus. <i>No</i> response in numb area favors conversion disorder.
Bowls and Currier test	Patient extends and then crosses the arms, with thumbs pointed down and palms facing together. Fingers (but not thumbs) are then interlocked, and then the hands are rotated inward toward chest. Sharp stimuli are applied to each finger in turn and the patient is asked to indicate normal or abnormal sensation in each digit. Patients with conversion disorder make mistakes and are inconsistent with responses.
Strength test	Patient closes eyes. Test "strength" by touching finger to be moved. True lack of sensation would not allow patient to ascertain finger to be moved.
Pain	
Gray test	With abdominal pain due to psychological factors, the patient will close eyes during palpation. In pain of organic basis, the patient is more likely to watch the examiner's hand to anticipate pain.

<b>Motor</b>	
Drop test	When a patient with paralysis of nonorganic etiology lifts a thumb, the affected limb will drop more slowly or fall with exaggerated speed as compared with the unaffected limb. In addition, an extremity dropped from above the face will miss it.
Thigh adductor test	Examiner places hands against both inner thighs of the patient who is told to adduct the "good" leg against resistance. With pseudoparalysis, the adductor muscles of "bad" leg will also adduct.
Hoover test	Examiner's hands cup both heels of the patient who is asked to elevate the "good" leg. With pseudoparalysis the "bad" leg will push downward. When the patient is asked to lift the "bad" leg, if there is no downward pressure in the "good" leg, the patient is not trying.
Sternocleidomastoid test	Contraction of normal sternocleidomastoid muscle causes face to rotate away from side of the contracted muscle. Patient with conversion hemiplegia cannot turn head to the weak side.
<b>Coma</b>	
Corneal reflex	Corneal reflexes remain intact in an awake patient.
Bell phenomenon	Eyes divert upward when lids are opened, whereas eyes remain in neutral position in true coma.
Lid closing	In true coma, lids when opened close rapidly initially and then more slowly as lids descend. Awake patients will have lids stay open, snap shut, or flutter.
<b>Seizures</b>	
Corneal reflex	Usually intact in pseudoseizure.
Abdominal musculature	Palpation of abdominal musculature reveals lack of contractions with pseudoseizure.
<b>Blindness</b>	
Opticokinetic drum	Rotating drum with alternating black and white stripes or piece of tape with alternating black and white sections pulled laterally in front of a patient's open eyes will produce nystagmus in a patient with intact vision.

**TABLE 186-2** Testing Techniques for Conversion Disorder

**Emergency Department Care and Disposition**

1. Direct confrontation stating that symptoms have no organic etiology may worsen the condition. After excluding an organic cause for symptoms, gently reassure the patient that a serious medical illness has not been identified.
2. Suggest to the patient that symptoms often spontaneously.
3. Consult with a neurologist or psychiatrist if symptoms do not resolve and preclude discharge. Otherwise, refer for outpatient psychiatric treatment as repetitive reassurance may be needed before full function returns.

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 287, “Panic Disorder,” by Linda M. Nicholas and Elizabeth Shumann; and see Chapter 288, “Conversion Disorder,” by Gregory P. Moore and Kenneth C. Jackimczyk.

## **SECTION 22 Abuse and Assault**

# **CHAPTER 187**

## **Child and Elderly Abuse**

Jonathan Glauser

### **CHILD ABUSE**

Child maltreatment includes physical abuse, sexual abuse, emotional abuse, neglect, parental substance abuse, and Munchausen syndrome by proxy.

#### **Clinical Features**

Child neglect in early infancy results in the syndrome of failure to thrive. These children are frequently brought to the ED because of other medical problems, such as skin infections, severe diaper dermatitis, or acute gastroenteritis. Infants may have little subcutaneous tissue, protruding ribs, or occipital alopecia from lying on their back all day. They are wide eyed, wary, and difficult to console. They may have increased muscle tone in their lower extremities. Weight gain in the hospital is thought to be diagnostic of failure to thrive.

Children over the age of 2 with environmental neglect are termed psychosocial dwarfs. They exhibit short stature, bizarre and voracious appetites are hyperactive with unintelligible or delayed speech.

Physical abuse is suggested by a history that is inconsistent with the nature of the injuries. The history of the event given by the caretaker may keep changing, or may be different from that given by the child.

The following findings suggest physical abuse: bruises over multiple areas, bites with an intercanine diameter  $>3$  cm, lacerations of the frenulum or oral mucosa from force-feeding, burns of an entire hand or foot, or burns of the buttocks or genitalia from toilet training punishment, cigarette burns, spiral fractures caused by twisting of long bones, metaphyseal chip

fractures, periosteal elevation from new bone formation at sites of previous microfractures, multiple fractures at different stages of healing, fractures at unusual sites such as lateral clavicle, ribs, sternum, vomiting, irritability, seizures, change in mental status, apnea or retinal hemorrhages from intracranial hemorrhage (shaken baby syndrome). Vomiting, abdominal pain and tenderness with diminished bowel sounds or abdominal distention may be due to a duodenal hematoma, as evidenced by a “double-bubble” sign on abdominal x-ray films.

Munchausen syndrome by proxy is a synonym for medical child abuse. A parent fabricates illness in a child in order to secure prolonged contact with health care providers. Complaints may be numerous and agents such as ipecac or warfarin may have been given to precipitate these complaints. Parents typically encourage more diagnostic tests, and are happy if they are positive.

Sexual abuse is suggested with complaints referable to the anogenital area, such as bleeding, discharge, or the presence of a sexually transmitted disease. Clefts or concavities in the hymen typically present in the 6 o'clock position. Victims of child abuse may be overly compliant with painful medical procedures, overly protective of the abusing parent, or overly affectionate to medical staff.

## **Diagnosis and Differential**

Any serious injury in a child under the age of 5 should be viewed with suspicion. Parents and caregivers may appear to be under the influence of drugs or alcohol and refuse diagnostic studies. Victims of neglect may appear dirty, improperly clothed, and may be unimmunized.

A skeletal survey of the long bones will help detect evidence of physical abuse. See [Chapter 157](#) “Trauma in Children,” for further workup of traumatic injuries.

Inspect the genital area carefully for injury. Speculum examination in the preadolescent is not needed unless perforating vaginal trauma is suspected. Absence of physical findings does not rule out abuse.

Laboratory testing for sexual abuse should include cultures of the throat, vagina, and rectum for gonorrhea and chlamydia. Rapid antigen assays are

**not** reliable forensic evidence in prepubescent children. Perform testing for syphilis testing if clinical concern exists. Test for HIV if clinically concerned and appropriate counseling is available.

Rarely, conditions such as leukemia, aplastic anemia, and osteogenesis imperfecta can mimic physical abuse.

## **Emergency Department Care and Disposition**

Address all medical issues and injuries. Infants suspected of suffering from failure to thrive and children with suspected Munchausen syndrome by proxy should be admitted to the hospital. Involve social services during the ED visit. Ensure a safe environment for each child. Every state requires reporting of suspected cases of child abuse. The law protects physicians from legal retaliation from parents.

## **ELDER ABUSE**

Elder abuse is an act or omission resulting in harm to the health or welfare of an elderly person.

### **Clinical Features**

Physical abuse is the most easily recognized form of elder abuse, although chemical restraint such as intentional overmedication may be subtle. *Caregiver neglect*, defined as failure of a caregiver to provide basic care, goods and services such as food, clothing and shelter, accounts for the majority of cases of elder abuse. Financial abuse is the second commonest form of abuse, and occurs when family members take control of or steal assets, checks, or pensions for personal gain.

Emotional abuse entails inflicting anguish, emotional pain, or distress. Verbal threats, social isolation, and harassment can contribute to depression and other mental health problems. Self-neglect includes those behaviors of

an elderly person that threaten his or her own safety: failure to provide adequate food, medical care, hygiene, clothing or shelter.

Caretakers may give a conflicting report of an injury or illness. The patient may be fearful of his or her companion. The caretaker may seem indifferent or angry toward the patient, or may be overly concerned with costs of treatment needed by the patient.

## **Diagnosis and Differential**

Risk factors for elder abuse may be associated with caregivers, perpetrators, or with the elders. Patient characteristics include: (a) cognitive impairment, (b) female sex, (c) physical dependency, (d) alcohol abuse, (e) developmental disability, (f) special medical or psychiatric needs, (g) lack of social support, and (h) limited experience managing finances.

Risk factors for perpetrators of abuse include (a) history of violence within or outside of the family, (b) excessive dependence on the elder for financial support, (c) history of mental illness or substance abuse.

Patients should be interviewed in private. Screening questions for elder abuse are available, areas of concern include whether anyone has touched or hurt them, forced them to do things, taken something of theirs without asking, threatened them, or made them feel afraid.

The following findings on physical examination are suggestive of abuse: bruising or trauma, poor general appearance and hygiene inappropriate or soiled clothing, malnutrition and dehydration, contusions and lacerations to normally protected areas of the body: inner thighs, mastoid, palms, soles, buttocks, unusual burns or multiple burns in different stages of healing, rope or restraint marks on ankles or wrists, spiral fractures of long bones, midshaft ulnar (nightstick) fractures from attempts to shield blows, multiple deep/ uncared for ulcers.

## **Emergency Department Care and Disposition**

Elder abuse is widely under-reported and under-recognized. Treatment entails 3 key components:



1. Address medical and psychosocial needs.
2. Ensure patient safety.
3. Adhere to local reporting requirements.

Medical problems and injuries may be best managed with hospital admission. If neglect is unintentional, educate the caregiver. All 50 states have reporting requirements for elder abuse and neglect. Requirements for reporting within one's practice area are available at [www.nceaaoa.gov](http://www.nceaaoa.gov).

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 290, "Child Abuse and Neglect," by Carol D. Berkowitz, and Chapter 293, "Abuse of the Elderly and Impaired," by Frederic M. Hustey and Jonathan Glauser.

## **CHAPTER 188**

# **Sexual Assault and Intimate Partner Violence and Abuse**

Sara Laskey

Not all individuals who are sexually assaulted sustain an injury. Lack of injury does not mean that an assault did not occur. Often the perpetrator is known to the assault survivor.

Intimate partner violence and abuse is defined as a pattern of assaultive behavior that may include physical injury, sexual assault, psychological abuse, stalking, deprivation, intimidation, and threats. Intimate partner violence and abuse occurs in every race, ethnicity, culture, geographic region, and religious affiliation and occurs in gay, lesbian, and heterosexual relationships.

## **CLINICAL FEATURES**

Elements of the sexual assault history are listed in [Table 188-1](#). The history for intimate partner violence and abuse can be more difficult to obtain. Risk factors for intimate partner violence and abuse include female sex, age between 20 and 24 years, low socioeconomic status, separated relationship status, and residence in rental housing. Injuries inconsistent with the patient's history, multiple injuries in various stages of healing, delay in the time of injury occurrence and presentation, a visit for vague complaints without evidence of injury, or suicide attempts should trigger suspicions of intimate partner violence and abuse. Patients may complain initially of chronic pain syndromes, gynecologic or psychiatric difficulties, and alcohol and substance abuse. The patient also may appear frightened when the partner is present or the partner may be hostile, defensive, aggressive, or overly solicitous. Recent and remote abuse, including dates, locations, details of abuse, and witnesses, should be documented. Patients need to be

asked about any suicidal or homicidal ideation and plans and get appropriate, immediate evaluation.

<p><b>Who?</b>          Did the assault survivor know the assailant?          Was it a single assailant or multiple assailants?          What were the assailant's identity and race? (Document in the medical records.)</p>
<p><b>What happened?</b>          Was the patient physically assaulted?          With what (eg, gun, bat, or fist) and where?          Was there actual or attempted vaginal, anal, or oral penetration?          Did ejaculation occur? If so, where?          Was a foreign object used?          Was a condom used?</p>
<p><b>When?</b>          When did the assault occur?          (Emergency contraception is most effective when started within 72 h of the assault.)</p>
<p><b>Where?</b>          Where did the assault occur?          (Corroborating evidence may be found based on the location of the assault.)</p>
<p><b>Suspicion of drug-facilitated rape</b>          Was there a period of amnesia?          Is there a history of being out drinking and then suddenly feeling very intoxicated?          Is there a history of waking up naked or with genital soreness?</p>
<p><b>Douche, shower, or change of clothing</b>          Did the patient douche, shower, or change clothing after the assault? (Performing any of these activities prior to seeking medical attention may decrease the probability of sperm or acid phosphatase recovery, as well as recovery of other bits of trace evidence.)</p>

**TABLE 188-1** Assault History

## Physical Examination

Perform a general medical examination including vital signs, appearance and demeanor. Focus head-to-toe inspection on defensive injury areas, such as the extremities, and potential areas of injury, such as the oral cavity, breasts, thighs, and buttocks. Record all signs of trauma, new and old, in detail using a body map. Speculum examination should note any trauma, discharge, abrasions, including on the cervix. If anal penetration is reported, the rectum should be examined for abrasions and lacerations. Use of anoscopy in male patients increases detection of trauma.

Characteristic injuries of intimate partner violence and abuse include fingernail scratches, bite marks, cigarette burns, rope burns, and forearm bruising or nightstick fractures, suggesting a defensive posture. Abdominal injuries are common in the pregnant intimate partner violence and abuse patient ([Table 188-2](#)).

Findings	Comments
Injuries characteristic of violence	Fingernail scratches, broken fingernails, bite marks, dental injuries, cigarette burns, bruises suggesting strangulation or restraint, and rope burns or ligature marks may be seen.
Injuries suggesting a defensive posture	Forearm bruises or fractures may be sustained when individuals try to fend off blows to the face or chest.
Injuries during pregnancy	Up to 45% of women report abuse or assault during pregnancy. Preterm labor, placental abruption, direct fetal injury, stillbirth can occur.
Central pattern of injury	Injuries to the head, neck, face, and thorax, and abdominal injuries in pregnant women may suggest violence.
Extent or type of injury inconsistent with the patient's explanation	Multiple injuries may be seen at different anatomic sites inconsistent with the described mechanism of injury. The most common explanation of injury is a "fall." Embarrassment, evasiveness, or lack of concern with the injuries may be noted.
Multiple injuries in various stages of healing	These may be reported as "accidents" or "clumsiness."
Delay between the time of injury and the presentation for treatment	Victims may wait several days before seeking medical care for injuries. Victims may seek care for minor or resolving injuries.
Visits for vague or minor complaints without evidence of physiologic abnormality	This pattern may include frequent ED visits for a variety of injuries or illnesses, including chronic pelvic pain and other chronic pain syndromes.
Suicide attempts	Women who attempt or commit suicide often have a history of intimate partner violence.

**TABLE 188-2** Signs Suggestive of Intimate Partner Violence

## Evidence Examination

Evidence collection in sexual assault is performed only within the first 72 hours after the assault. Informed consent if required. Most hospitals have a

prepackaged rape kit. Chain of custody of the evidence must be maintained and the kit should never be left unattended. If > 72 hours have elapsed or the patient declines an evidentiary examination, perform a history, physical examination, document injuries, and provide prophylaxis for pregnancy and sexually transmitted infections. Label evidence clearly with the patient's name, type and source of evidence, date and time, and name of the examiner collecting the evidence.

## **DIAGNOSIS AND DIFFERENTIAL**

Sexual assault is a legal determination, not a medical diagnosis. The legal definition contains 3 elements: carnal knowledge, nonconsent, and compulsion or fear of harm. Because of the legal considerations, careful documentation and evidence collection are important.

Many experts recommend routine screening for intimate partner violence and abuse for all adolescent and adult women who present to the ED and for mothers of children. Providers educated about the dynamics of intimate partner violence and abuse should conduct screening in a safe and private environment. Document all findings, interventions, referrals and required reporting.

## **EMERGENCY DEPARTMENT CARE AND DISPOSITION**

Address life-threatening injuries and the psychological needs. Treating critical injuries are the initial priority of the ED physician. A social worker or trained advocate should counsel the patient in the ED. Ensure the safety of the patient and any children involved while they are in the ED. Be familiar with reporting laws in your state.

## **SEXUAL ASSAULT**

### **Emergency Contraception**

1. Obtain a pregnancy test.
2. Offer emergency contraception. **Levonorgesterol** only, 1.5 milligrams PO in a single dose or 2 doses 0.75 milligrams PO 12 hours apart OR, **combined estrogen-progestin** 2 doses **ethinyl estradiol**, 100 micrograms PO plus **levonorgestrel**, 0.5 milligrams PO, 12 hours apart.

## **STD Prophylaxis**

Recommended regimens for infection prophylaxis include a single dose of **ceftriaxone**, 250 milligrams IM PLUS a single dose of **metronidazole**, 2 grams PO PLUS a single dose of **azithromycin**, 1 gram PO, OR **doxycycline**, 100 milligrams PO twice a day for 7 days.

## **STD Treatment**

1. Gonorrhea: **Ceftriaxone** 250 milligrams IM single dose or **cefixime**, 400 milligrams PO single dose.
2. Chlamydia: **azithromycin** 1 gram PO as single dose OR **doxycycline** 100 milligrams PO twice per day for 7 days (do not use doxycycline during pregnancy).
3. Trichomonas and bacterial vaginosis: **metronidazole** 2 grams PO in single dose (do not use during first trimester of pregnancy).
4. Syphilis: **penicillin G benzathine** 2.4 million IU IM. Use **erythromycin** 500 milligrams PO 4 times per day for 15 days if penicillin allergic.

## **Hepatitis Prophylaxis**

Administer vaccine at time of initial exam if patient has not been previously vaccinated. Follow-up doses of vaccine should be administered 1 to 2 months and 4 to 6 months after the first dose

## **HIV: Prophylaxis and counseling**

Rates of human immunodeficiency virus (HIV) seroconversion are low but have occurred from sexual assault or sexual abuse. Circumstances should guide the decision to administer postexposure prophylaxis. An assailant known to be infected with HIV, high viral load exposure, vaginal trauma, and ejaculate on membranes increase the risk for HIV seroconversion. Post-HIV exposure recommendations are posted on the Centers for Disease Control and Prevention (CDC) website ([www.cdc.gov](http://www.cdc.gov)). Routine prophylaxis is not recommended, and counseling and follow-up should be provided.

## **INTIMATE PARTNER VIOLENCE AND ABUSE**

1. An attempt to leave an abusive relationship is often the most dangerous time.
2. Assess for potentially lethal situations. These include increasing frequency or severity of violence, the threat or use of weapons, obsession with the patient, taking hostages, stalking, homicidal or suicidal threats, and substance abuse by the assailant, especially with crack cocaine or amphetamines.
3. Hospital admission is an option in high-risk situations if a safe location cannot be established before discharge.

### **Follow-up Care**

Follow-up care often requires the coordinated efforts of physicians, law enforcement, survival advocates, and counselors. Some communities and hospitals use the Sexual Assault Nurse Examiner (SANE) programs, others have rape crisis centers, and 24-hours safe rooms. National hotlines are available ([Table 188-3](#)). Know the resources available in your community.

<b>National Domestic Violence Hotline:</b> 24 h; links caller to help in her (or his) area—emergency shelter, domestic violence shelters, legal advocacy and assistance programs, social services	800-799-SAFE (7233) 800-787-3224 (TTY)
<b>Rape, Abuse, and Incest National Network:</b> 24 h; automatically transfers caller to nearest rape crisis center anywhere in the nation	800-656-HOPE (4673) <a href="http://www.rainn.org">http://www.rainn.org</a>

**TABLE 188-3** Hotlines for Patients

For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 7th ed., see Chapter 291, “Female and Male Sexual Assault,” by Sheryl L. Heron and Debra E. Houry; and Chapter 292, “Intimate Partner Violence and Abuse,” by Mary Hancock.



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