26 Medical Emergencies

Rebecca Bavolek, Evan Schwarz, and Jason Wagner

AIRWAY EMERGENCIES

Emergent Airway Management

GENERAL PRINCIPLES

Recognition of the need to manage a patient's airway must be made in a timely and rapid fashion. Respiratory failure can range from immediate to insidious. Increasing evidence shows that inexperienced intubators frequently do more harm than good.

Etiology

The need to emergently manage an airway typically arises for one of three reasons.

- Loss of airway protective reflexes
- Respiratory failure
- Cardiopulmonary arrest

TREATMENT

- If you are not prepared to manage a definitive airway, there are several things that can be done to either temporarily support the airway or help maximize success.
- Inexperienced intubators should maintain the airway with the best device immediately available. Evidence suggests the following order from most effective to least.
  - High-flow nasal cannula oxygen at 15 L/min (apneic oxygenation increases desaturation time)
  - Non-rebreather oxygen mask (NRB) at 15 L/min
  - Bag-valve mask (BVM)
  - Supraglottic airway devices (laryngeal mask airway, Combitube, King Tube)
- While awaiting definitive airway expertise, there are steps that can be taken to improve success during intubation.
  - Place the patient upright to decrease dependent lung volume before intubation.
  - Place the patient on NRB for 3 minutes if possible.
  - If you cannot delay for 3 minutes, then deliver eight vital capacity breaths via BVM.
  - Place a positive end-expiratory pressure valve set to 5–20 cm H_2O on an NRB adding positive pressure to both bagging and passive oxygenation.

Performing the above steps will maximize your chances of success in nearly all airway situations, whether you are the one managing the airway or someone else.

Emergent Airway Adjuncts

- **Gum elastic bougie** is a flexible rubbery stick with a hockey stick tip. The bougie can be used blindly but is better suited for direct laryngoscopy where the intubator cannot visualize the cords. The goal is to obtain the best view possible and for the coude tip of the bougie to be distal and anterior. When the bougie is in the trachea, you can often feel the tracheal rings as you slide the bougie back and forth. Alternatively, you can push the bougie down the oropharynx as deep as possible without losing control of it. If in

the esophagus, the bougie will slide all the way down past the stomach with minimal resistance. If in the trachea, the bougie will quickly hit a bronchus and meet resistance. Once you are in the trachea, you can simply slide an endotracheal tube (ETT) over the bougie and verify placement as you normally would.

- **Laryngeal mask airway (LMA)** is an easy-to-use rescue device for nearly all airway events. It is an ETT with a balloon at the end that is inflated to cup the trachea while occluding the esophagus. Note that it should not be used in patients with upper airway obstruction that cannot be cleared or patients with excessive airway pressures such as with chronic obstructive pulmonary disease (COPD), asthma, or pregnancy. There are models of LMAs (which are preferred) that allow an ETT to be passed through them when a definitive airway is desired. Be cautious with excessive bagging because this can lead to emesis.
- **Supraglottic airway devices** are placed blindly in the oropharynx and inflated with air. An upper balloon obstructs the oropharynx while a lower balloon obstructs the esophagus, allowing ventilation in a similar fashion to an LMA with the same limitations. Note that you cannot intubate through a supraglottic device as you can with intubating LMAs.
- **Fiberoptic/digital airway devices** are considered by many the new standard of care. These devices allow the intubator to get a view of the vocal cords via a camera or fiberoptic scope without having to view it through the mouth, making intubation much easier. Excessive secretions or blood can obstruct the camera, so the person using these devices needs to be capable of direct laryngoscopy as well as indirect fiberoptic laryngoscopy.

Pneumothorax

GENERAL PRINCIPLES

- Pneumothorax may occur spontaneously or as a result of trauma.
- **Primary spontaneous pneumothorax** occurs without obvious underlying lung disease.
- **Secondary spontaneous pneumothorax** results from underlying parenchymal lung disease including COPD and emphysema, interstitial lung disease, necrotizing lung infections, *Pneumocystis jirovecii* pneumonia, TB, and cystic fibrosis.
- **Traumatic pneumothoraces** occur as a result of penetrating or blunt chest wounds.
- **Iatrogenic pneumothorax** occurs after thoracentesis, central line placement, transbronchial biopsy, transthoracic needle biopsy, and barotrauma from mechanical ventilation and resuscitation.
- **Tension pneumothorax** results from continued accumulation of air in the chest that is sufficient to shift mediastinal structures and impede venous return to the heart. This results in hypotension, abnormal gas exchange, and ultimately, cardiovascular collapse.
  - Causes include barotrauma due to mechanical ventilation, a chest wound that allows ingress but not egress of air, or a defect in the visceral pleura that behaves in the same way ("ball-valve" effect).
  - Suspect tension pneumothorax when a patient experiences hypotension and respiratory distress on mechanical ventilation or after any procedure in which the thorax is pierced by a needle.

DIAGNOSIS

Clinical Presentation

History

- Patients commonly complain of ipsilateral chest or shoulder pain, usually of acute onset. A history of recent chest trauma or medical procedure can suggest the diagnosis.
- Dyspnea is usually present.

Physical Examination

- Although examination of the patient with a small pneumothorax may be normal, classic findings include decreased breath sounds and a more resonant percussion note on the ipsilateral side.
- With a larger pneumothorax or with underlying lung disease, there may be tachypnea and respiratory distress. The affected hemithorax may be noticeably larger (due to decreased elastic recoil of the collapsed lung) and relatively immobile during respiration.
- If the pneumothorax is very large, and particularly if it is under tension, the patient may exhibit severe distress, diaphoresis, cyanosis, and hypotension. In addition, the patient's trachea may be shifted to the contralateral side.
- If the pneumothorax is the result of penetrating trauma or pneumomediastinum, subcutaneous emphysema may be felt.
- Clinical features alone do not predict the relative size of a pneumothorax, and in a stable patient, further diagnostic studies must be used in order to guide treatment strategy. However, tension pneumothorax remains a clinical diagnosis, and if suspected in the appropriate clinical scenario, immediate intervention should be undertaken prior to further testing.

Diagnostic Testing

Electrocardiography

An **ECG** may reveal diminished anterior QRS amplitude and an anterior axis shift. In extreme cases, tension pneumothorax may cause electromechanical dissociation.

Imaging

- A **CXR** will reveal a separation of the pleural shadow from the chest wall. If the posteroanterior radiograph is normal and pneumothorax is suspected, a lateral or decubitus film may aid in diagnosis (*Thorax 2003;58(suppl II):ii39*). Air travels to the highest point in a body cavity; thus, a pneumothorax in a supine patient may be detected as an unusually deep costophrenic sulcus and excessive lucency over the upper abdomen caused by the anterior thoracic air. This observation is particularly important in the critical care unit, where radiographs of the mechanically ventilated patient are often obtained with the patient in supine position.
- Although tension pneumothorax is a clinical diagnosis, radiographic correlates include mediastinal and tracheal shift toward contralateral side and depression of the ipsilateral diaphragm.
- **Ultrasonography** is a useful tool for bedside diagnosis of pneumothorax, especially on patients who must remain supine or who are too unstable to undergo CT scanning. Placement of the probe in the intercostal spaces provides information regarding the pleura and underlying lung parenchyma. During normal inspiration, the visceral and parietal pleura move along one another and produce a "sliding sign" phenomenon. In addition, the air-filled lung parenchyma below the pleura produces a ray-like opacity known as "comet tails." Presence of the sliding sign and comet tails on ultrasound during inspiration rule out a pneumothorax with high reliability at the point of probe placement. Conversely, absence of these signs is a highly reliable predictor for the presence of pneumothorax. Several places on the chest should be evaluated, including places that air is most likely to accumulate such as the anterior and lateral chest (*Chest 2012;141:1099*). Studies have shown that in the hands of an experienced clinician with ultrasound training, chest ultrasound is more sensitive than CXR (*Emerg Radiol 2013;20:131*).
- Chest CT is the gold standard for diagnosis and determining the size of pneumothorax. Although not always necessary, it may be particularly useful for differentiating pneumothorax from bullous disease in patients with underlying lung conditions (*Thorax 2003;58(suppl II):ii42*).

TREATMENT

Treatment depends on cause, size, and degree of physiologic derangement.

- **Primary pneumothorax**
  - A small, primary, spontaneous pneumothorax without a continued pleural air leak may resolve spontaneously. Air is resorbed from the pleural space at roughly 1.5% daily, and therefore, a small (approximately 15%) pneumothorax is expected to resolve without intervention in approximately 10 days.
  - Confirm that the pneumothorax is **not increasing in size** (repeat the CXR in 6 hours if there is no change in symptoms) and send the patient home if he or she is asymptomatic (apart from mild pleurisy). Obtain follow-up radiographs to confirm resolution of the pneumothorax in 7–10 days. Air travel is discouraged during the follow-up period, because a decrease in ambient barometric pressure results in a larger pneumothorax.
  - If the pneumothorax is **small but the patient is mildly symptomatic**, far from home, or unlikely to cooperate with follow-up, admit the patient and administer high-flow oxygen; the resulting nitrogen gradient will speed resorption.
  - If the patient is **more than mildly symptomatic or has a larger pneumothorax**, simple aspiration is a reasonable initial management strategy. However, aspiration may not be successful for very large pneumothoraces. In patients in whom aspiration fails, proceed with thoracostomy tube insertion (*Thorax 2003;58(suppl II):ii39*).
  - **Pleural sclerosis** to prevent recurrence is recommended by some experts but, in most cases, is not used after a first episode unless a persistent air leak is present.
- **Secondary pneumothorax**
  - Individuals with a secondary spontaneous pneumothorax are usually symptomatic and require lung reexpansion.
  - Often, a bronchopleural fistula persists and a larger thoracostomy tube and suction are required.
  - **Consult a pulmonologist** about pleural sclerosis for persistent air leak and to prevent recurrence.
  - **Surgery** may be required for persistent air leak and should be considered for high-risk patients for prevention of recurrence.
- **Iatrogenic pneumothorax**
  - Iatrogenic pneumothorax is generally caused either by introducing air into the pleural space through the parietal pleura (e.g., thoracentesis, central line placement) or by allowing intrapulmonary air to escape through breach of the visceral pleura (e.g., transbronchial biopsy). Often, no further air leak occurs after the initial event.
  - If the pneumothorax is small and the patient is minimally symptomatic, he or she can be managed conservatively. If the procedure that caused the pneumothorax required sedation, admit the patient, administer oxygen, and repeat the CXR in 6 hours to ensure the patient's stability. If the patient is completely alert and the CXR shows no change, the patient can be discharged.
  - If the patient is symptomatic or if the pneumothorax is too large for expectant care, a pneumothorax catheter with aspiration or a one-way valve is usually adequate and can often be removed the following day.
  - Iatrogenic pneumothorax due to barotrauma from mechanical ventilation almost always has a persistent air leak and should be managed with a chest tube and suction.
  - **Tension pneumothorax**
    - When the clinical situation and physical examination strongly suggest this diagnosis, decompress the affected hemithorax immediately with a 14-gauge needle. Place the needle in the second intercostal space, midclavicular line, just superior to the rib. Release of air with clinical improvement confirms the diagnosis.
    - Recognize that an obese patient or a patient with a large amount of breast tissue may not have resolution of tension with a standard angiocatheter due to inability to reach

the chest wall or weight of the tissue kinking off the air escape path. These patients may require a longer needle than a standard angiocatheter in order to reach the intrathoracic space for decompression or require insertion of a larger gauge reinforced catheter to stent open the pathway for air release.

- If long-needle decompression or reinforced catheter insertion is unsuccessful, and the diagnosis is highly probable in an unstable patient, surgical decompression can be performed by incision of the pleura in the fourth to fifth anterior axillary line above the rib in the same space in which thoracostomy tubes are inserted. This technique has been shown to be effective; however, safety and complication rates are not able to be determined due to lack of studies (*Resuscitation 2007;72(1):11*). The full technique for this procedure is beyond the scope of this book.
- Seal any chest wound with an occlusive dressing and arrange for placement of a thoracostomy tube.

HEAT-INDUCED INJURY

Heat Exhaustion

GENERAL PRINCIPLES

Heat exhaustion occurs through water or sodium depletion but is often a combination of both. Water depletion heat exhaustion often occurs in the elderly or persons working in hot environments with limited water replacement. Salt depletion occurs in unacclimatized individuals who replace fluid losses with large amounts of hypotonic solution.

DIAGNOSIS

- The patient presents with headache, nausea, vomiting, dizziness, weakness, irritability, and/or cramps.
- The patient may have postural hypotension, diaphoresis, and normal or minimally increased core temperature.

TREATMENT

- Treatment consists of resting the patient in a cool environment, accelerating heat loss by fan evaporation, and repleting fluids with salt-containing solutions.
- If the patient is not vomiting and has stable blood pressure, an oral, commercial, balanced salt solution is adequate.
- If the patient is vomiting or hemodynamically unstable, check electrolytes and give 1–2 L of 0.9% saline IV.
- The patient should avoid exercise in a hot environment for 2–3 additional days.

Heat Syncope

GENERAL PRINCIPLES

- Heat syncope is a variant of postural hypotension.
- Exercise in a hot environment results in peripheral vasodilation and pooling of blood, with subsequent loss of consciousness. The affected individual has normal body temperature and regains consciousness promptly when supine. These factors separate this syndrome from heat stroke.

TREATMENT

Treatment consists of resting in a cool environment and fluid repletion.

Heat Stroke

GENERAL PRINCIPLES

- Heat stroke occurs in two varieties, classic and exertional. Both varieties present with high core temperatures that result in direct thermal tissue injury. Secondary effects include acute renal failure from rhabdomyolysis. Even with rapid therapy, mortality rates can be very high for body temperatures above 41.1°C (106°F). The distinction between classic and exertional heat stroke is not important because the therapeutics goals are similar in both and a delay in cooling increases mortality rate.
- The cardinal features of heat stroke are **hyperthermia (>40°C [104°F]) and altered mental status**. Although patients presenting with classic heat stroke may have anhidrosis, this is not considered a diagnostic criterion, because 50% of patients are still diaphoretic at presentation.
- The central nervous system (CNS) is very vulnerable to heat stroke with the cerebellum being highly sensitive. Ataxia may be an early sign. Seizures are common. Neurologic injury is a function of maximum temperature and duration of exposure (*N Engl J Med 2002;346:1978*).

DIAGNOSIS

Diagnosis is based on the history of exposure or exercise, a core temperature usually of 40.6°C (105°F) or higher, and changes in mental status ranging from confusion to delirium and coma.

Differential Diagnosis

- Drug associated
  - Toxicity
    - Anticholinergic
    - Stimulant toxicity
    - Salicylate toxicity
  - Neuroleptic malignant syndrome (NMS) associated with antipsychotic drugs. It is worth noting that NMS and malignant hyperthermia are both accompanied by severe muscle rigidity.
  - Serotonin syndrome
  - Malignant hyperthermia
  - Drug withdrawal syndrome (ethanol withdrawal)
  - Drug fever
- Infections
  - Generalized infections (sepsis, malaria, etc.)
  - CNS infections (meningitis, encephalitis, brain abscess)
- Endocrine
  - Thyroid storm
  - Pheochromocytoma
- Hypothalamic dysfunction due to stroke or hemorrhage
  - Status epilepticus
  - Cerebral hemorrhage (*Emerg Med Clin North Am 2013;31(4):1097*)

Diagnostic Testing

Laboratories

- Laboratory studies should include complete blood count (CBC); partial thromboplastin time; prothrombin time; fibrin degradation products; electrolytes; blood urea nitrogen (BUN); creatinine, glucose, calcium, and creatine kinase levels; liver function tests (LFTs); arterial blood gases (ABGs); urinalysis; and ECG.
- If an infectious etiology is suspected, obtain appropriate cultures.

Imaging

If a CNS etiology is considered likely, CT imaging followed by spinal fluid examination is appropriate.

TREATMENT

- **Immediate cooling** is necessary.
  - The best method of cooling is controversial. A systematic review showed that ice water immersion decreases body temperature twice as quickly as passive cooling and is the procedure of choice when exertional heat stroke is anticipated (long-distance races, military training). If that cannot be achieved, continuous water spray accompanied by fanning has been shown to be adequate for most patients with exertional heat stroke (*Int J Sports Med 1998;19(suppl 2):S150*; *Ann Intern Med 2000;132:678*; *J Athl Train 2009;44(1):84*).
  - If response is insufficiently rapid, submerge the patient in ice water, recognizing that this may interfere with resuscitative efforts (*Am J Emerg Med 1996;14:355*).
  - Most emergency facilities that do not care for large numbers of heat illness cases are not equipped for this treatment. In this case, mist the patient continuously with tepid water (20–25°C [68–77°F]). Cool the patient with a large electric fan with maximum body surface exposure.
  - Ice packs should be placed at points of major heat transfer, such as the groin, axillae, and chest, to further speed cooling.
  - Antipyretics have no indication.
- **Dantrolene sodium** does not appear to be effective for the treatment of heat stroke (*Crit Care Med 1991;19:176*).
- Monitor core temperatures continuously by rectal probe because oral and tympanic membrane temperature may be inaccurate.
- Discontinue cooling measures when the core temperature reaches 39°C (102.2°F), which should ideally be achieved within 30 minutes. A temperature rebound may occur in 3–6 hours and should be retreated.
- **For hypotension, administer crystalloids:** If refractory, treat with vasopressors and monitor hemodynamics. Avoid pure α-adrenergic agents, because they cause vasoconstriction and impair cooling. Administer crystalloids cautiously to normotensive patients.

COMPLICATIONS

- **Rhabdomyolysis** may occur. Treat as described in Chapter 13, Renal Diseases.
- **Hypoxemia and acute respiratory distress syndrome** may occur. Treat as described in Chapter 8, Critical Care.
- Treat seizures with benzodiazepines and phenytoin.

MONITORING/FOLLOW-UP

Patients should be placed on telemetry.

COLD-INDUCED ILLNESS

Exposure to the cold may result in several different forms of injury. A risk factor is accelerated heat loss, which is promoted by exposure to high wind or by immersion. Extended cold exposure may result from alcohol or drug abuse, injury or immobilization, and mental impairment.

Chilblains

GENERAL PRINCIPLES

- Chilblains are among the mildest form of cold injury and result from exposure of bare skin to a cold, windy environment (0.6–15.6°C [33–60°F]).
- The ears, fingers, and tip of the nose typically are injured, with itchy, painful erythema on rewarming.

TREATMENT

Treatment involves rapid rewarming (see Frostnip section), moisturizing lotions, analgesics, and instructing the patient to avoid reexposure.

Immersion Injury (Trench Foot)

GENERAL PRINCIPLES

Immersion injury is caused by prolonged immersion (longer than 10–12 hours) at a temperature $<$10°C ($<$50°F).

TREATMENT

Treat by rewarming followed by dry dressings. Treat secondary infections with antibiotics.

Frostnip (Superficial Frostbite)

GENERAL PRINCIPLES

Superficial frostbite involves the skin and SC tissues.

DIAGNOSIS

Areas with first-degree involvement are white, waxy, and anesthetic; have poor capillary refill; and are painful on thawing. Second-degree involvement is manifested by clear or milky bullae.

TREATMENT

The **treatment of choice** is rapid rewarming. Immerse the affected body part for 15–30 minutes; hexachlorophene or povidone-iodine can be added to the water bath. Narcotic analgesics may be necessary for rewarming pain. Typically, no deep injury ensues and healing occurs in 3–4 weeks.

Deep Frostbite

GENERAL PRINCIPLES

- Deep frostbite involves death of skin, SC tissue, and muscle (third degree) or deep tendons and bones (fourth degree).
- Diabetes mellitus, peripheral vascular disease, an outdoor lifestyle, and high altitude are additional **risk factors**.

DIAGNOSIS

- The tissue appears frozen and hard.
- On rewarming, there is no capillary filling.
- Hemorrhagic blisters form, followed by eschars. Healing is very slow, and demarcation of tissue with autoamputation may occur.
- The majority of deep frostbite occurs at temperatures <6.7°C (44°F) with exposures longer than 7–10 hours.

TREATMENT

- The treatment is rapid rewarming as described earlier. **Rewarming should not be started until there is no chance of refreezing.**
- Administer analgesics (IV opioids) as needed.
- Early surgical intervention is not indicated.
- **Elevate** the affected extremity, prevent weight bearing, separate the affected digits with cotton wool, prevent tissue maceration by using a blanket cradle, and prohibit smoking.
- Update tetanus immunization.
- Intra-arterial vasodilators, heparin, dextran, prostaglandin inhibitors, thrombolytics, and sympathectomy are not routinely justified.
- Role of antibiotics is unclear (*Tintinalli's Emergency Medicine Manual. 7th ed. New York: McGraw-Hill, 2010: Chapter 202: Frostbite and Other Localized Cold Injuries*).
- Amputation is undertaken only after full demarcation has occurred.

Hypothermia

GENERAL PRINCIPLES

Definition

Hypothermia is defined as a core temperature of <35°C (95°F).

Classification

Classification of severity by temperature is not universal. One scheme defines hypothermia as mild at 34–35°C (93.2–95°F), moderate at 30–34°C (86–93.2°F), and severe at <30°C (86°F).

Etiology

- The most common cause of hypothermia in the United States is cold exposure due to alcohol intoxication.
- Another common cause is cold water immersion.

DIAGNOSIS

Clinical Presentation

Presentation varies with the temperature of the patient on arrival. All organ systems can be involved.

- **CNS effects**
  - At temperatures **below 32°C (89.6°F)**, mental processes are slowed and the affect is flattened.
  - At **32.2°C (90°F)**, the ability to shiver is lost, and deep tendon reflexes are diminished.
  - At **28°C (82.4°F)**, coma often supervenes.
  - **Below 18°C (64.4°F)**, the electroencephalogram (EEG) is flat. On rewarming from severe hypothermia, central pontine myelinolysis may develop.
- **Cardiovascular effects**
  - After an initial increased release of catecholamines, there is a decrease in cardiac output and heart rate with relatively preserved mean arterial pressure. ECG changes manifest initially as sinus bradycardia with T-wave inversion and QT interval prolongation and may manifest as atrial fibrillation at temperatures of <32°C (<89.6°F).
  - Osborne waves (J-point elevation) may be visible, particularly in leads II and V_6.
  - An increased susceptibility to ventricular arrhythmias occurs at temperatures **below 32°C (89.6°F)**.
  - At temperatures of **30°C (86°F)**, the susceptibility to ventricular fibrillation is increased significantly, and unnecessary manipulation or jostling of the patient should be avoided.
  - A decrease in mean arterial pressure may also occur, and at temperatures of **28°C (82.4°F)**, progressive bradycardia supervenes.
- **Respiratory effects**
  - After an initial increase in minute ventilation, respiratory rate and tidal volume decrease progressively with decreasing temperature.
  - ABGs measured with the machine set at 37°C (98.6°F) should serve as the basis for therapy without correction of pH and carbon dioxide tension (PCO_2) (*Ann Emerg Med 1989;18:72*; *Arch Intern Med 1998;148:1643*).
- **Renal manifestations:** Cold-induced diuresis and tubular concentrating defects may be seen.

Differential Diagnosis

- Cerebrovascular accident
- Drug overdose
- Diabetic ketoacidosis
- Hypoglycemia
- Uremia
- Adrenal insufficiency
- Myxedema

Diagnostic Testing

Laboratories

- Basic laboratory studies should include CBC, coagulation studies, LFTs, BUN, electrolytes, creatinine, glucose, creatine kinase, calcium, magnesium, amylase levels, urinalysis, ABGs, and ECG.
- Obtain toxicology screen if mental status alteration is more profound than expected for temperature decrease.
- Serum potassium is often increased.
- Elevated serum amylase may reflect underlying pancreatitis.

- Hyperglycemia may be noted but should not be treated because rebound hypoglycemia may occur with rewarming.
- Disseminated intravascular coagulation may also occur.

Imaging

Obtain chest, abdominal, and cervical spine radiographs to evaluate all patients with a history of trauma or immersion injury.

TREATMENT

Medications

- Administer supplemental oxygen.
- Give **thiamine** to most patients with cold exposure, because exposure due to alcohol intoxication is common.
- Administration of **antibiotics** is a controversial issue; many authorities recommend antibiotic administration for 72 hours, pending cultures. In general, the patients with hypothermia due to exposure and alcohol intoxication are less likely to have a serious underlying infection than those who are elderly or who have an underlying medical illness.

Other Nonpharmacologic Therapies

- **Rewarming:** The patient should be rewarmed with the goal of increasing the temperature by 0.5–2.0°C/h (32.9–35.6°F/h), although the rate of rewarming has not been shown to be related to the outcome.
- **Passive external rewarming**
  - This method depends on the patient's ability to shiver.
  - It is effective only at core temperatures of **32°C (89.6°F) or higher**.
  - Remove wet clothing, cover patient with blankets in a warm environment, and monitor.
- **Active external rewarming**
  - Application of heating blankets (40–45°C [104–113°F]) or warm bath immersion may cause paradoxical core acidosis, hyperkalemia, and decreased core temperature, as cold stagnant blood returns to the central vasculature (*J R Nav Med Serv 1991;77:139*), although Danish naval research supports arm and leg rewarming as effective and safe (*Aviat Space Environ Med 1999;70:1081*).
  - Pending further investigation, active rewarming is best reserved for young, previously healthy patients with acute hypothermia and minimal pathophysiologic derangement.
- **Active core rewarming is preferred for treatment of severe hypothermia**, although few data are available on outcomes (*Resuscitation 1998;36:101*).
  - **Heated oxygen** is the initial therapy of choice for the patient whose cardiovascular status is stable. This therapeutic maneuver can be expected to raise core temperatures by 0.5–1.2°C/h (32.9–34.2°F/h) (*Ann Emerg Med 1980;9:456*). Administration through an ETT results in more rapid rewarming than delivery via face mask. Administer heated oxygen through a cascade humidifier at a temperature of 45°C (113°F) or lower.
  - **IV fluids** can be heated in a microwave oven or delivered through a blood warmer; give fluids only through peripheral IV lines.
  - **Intravascular heat exchange via catheter** is a recent addition to the options on this list. Less invasive and more familiar to most physicians than the more extreme options, this option can increase body temperature up to 3°C/h. This is simply a central venous catheter placed by the familiar modified Seldinger technique. After placement, warm fluids are run through the catheter (not into the venous system) allowing for cool blood to rewarm as it passes.
  - **Heated nasogastric or bladder lavage** is of limited efficacy because of low-exposed surface area and is reserved for the patient with cardiovascular instability.
  - **Heated peritoneal lavage** with fluid warmed to 40–45°C (104–113°F) is more effective than heated aerosol inhalation, but it should be reserved for patients with cardiovascular

instability. Only those who are experienced in its use should perform heated peritoneal lavage, in combination with other modes of rewarming.
  - **Closed thoracic lavage** with heated fluid by thoracostomy tube has been recommended but is unproved (*Ann Emerg Med 1990;19:204*).
  - **Hemodialysis** can be used for the severely hypothermic, particularly when due to an overdose that is amenable to treatment in this way.
  - **Extracorporeal circulation** (cardiac bypass) is used only in hypothermic individuals who are in cardiac arrest; in these cases, it may be dramatically effective (*N Engl J Med 1997;337:1500*). Extracorporeal circulation may raise the temperature as rapidly as 10–25°C/h (50–77°F/h) but must be performed in an intensive care unit (ICU) or operating room.

Resuscitation

- Maintain airway and administer oxygen.
- If intubation is required, the most experienced operator should perform it (see Airway Management and Tracheal Intubation section in Chapter 8, Critical Care).
- Conduct **cardiopulmonary resuscitation (CPR)** in standard fashion. Perform simultaneous vigorous core rewarming; as long as the core temperature is severely decreased, it should not be assumed that the patient cannot be resuscitated. Reliable defibrillation requires a core temperature of 32°C (89.6°F) or higher; prolonged efforts (to a core temperature of 35°C [95°F]) may be justified because of the neuroprotective effects of hypothermia. **Do not begin CPR if an organized ECG rhythm is present**, because inability to detect peripheral pulses may be due to vasoconstriction, and CPR may precipitate ventricular fibrillation.
- Do not perform Swan-Ganz catheterization, because it may precipitate ventricular fibrillation.
- If ventricular fibrillation occurs, begin CPR as per the advanced cardiac life support (ACLS) protocol (Appendix C). Amiodarone may be administered as per the protocol, although there is no evidence to support its use or guide dosage; some experts suggest reducing the maximum cumulative dose by half. Avoid procainamide because it may precipitate ventricular fibrillation and increase the temperature that is necessary to defibrillate the patient. Rewarming is key.
- Monitor ECG rhythm, urine output, and, possibly, central venous pressure in all patients with an intact circulation.

Disposition

- Admit patients with an underlying disease, physiologic derangement, or core temperature <32°C (<89.6°F), preferably to an ICU.
- Discharge individuals with mild hypothermia (32–35°C [89.6–95°F]) and no predisposing medical conditions or complications when they are normothermic and an adequate home environment can be ensured.

MONITORING/FOLLOW-UP

- Monitor core temperature.
- A standard oral thermometer registers only to a lower limit of 35°C (95°F). Monitor the patient continuously with a rectal probe with a full range of 20–40°C (68–104°F).

OVERDOSES

Overdose, General

Below is a brief review of three of the most common toxicologic emergencies physicians encounter in the United States. For more information about these exposures and other toxicologic conditions, please refer to Chapter 28, Toxicology, an online chapter.

Acetaminophen

GENERAL PRINCIPLES

N-Acetyl-para-aminophenol (APAP) is available worldwide as an over-the-counter analgesic and antipyretic. It is the most common cause of toxicologic fatalities and liver failure in the United States (*Clin Toxicol (Phila) 2014;52(10):1032*; *Hepatology 2005;42(6):1364*).

DIAGNOSIS

Clinical Presentation

- Patients can initially present with nausea, vomiting, and abdominal pain. However, patients can be asymptomatic, even after potentially toxic ingestions.
- As toxicity progresses, patients develop transaminase elevation, metabolic acidosis, renal failure, and a coagulopathy.
- Patients may eventually develop fulminant hepatic failure, cerebral edema, and sepsis.
- Acetaminophen combination products (such as opioids, antihistamines) can cause additional symptoms such as opioid toxicity and anticholinergic delirium.

History

To predict the risk of hepatotoxicity after acute overdose and use the Rumack-Matthew nomogram, a reliable time of ingestion must be obtained from the patient or family/friends.

Physical Examination

Assess airway, breathing, and circulation (ABCs) and mental status. Especially in patients who are nauseated or vomiting, the assessment of mental status is crucial to prevent aspiration pneumonitis.

Diagnostic Criteria

- Obtain an APAP serum concentration at 4 hours or later after an acute ingestion.
- Plot the APAP concentration on the Rumack-Matthew nomogram (APAP serum concentration vs. time after ingestion) to assess the possibility of hepatic toxicity. Note: The nomogram can only be used for acute ingestions.
- In general, an APAP dose of 150 mg/kg is the potentially toxic limit that requires therapeutic intervention. This limit includes an added 25% safety margin that was added by the US Food and Drug Administration (*BMJ 1998;316(7146):1724*).
- If the time of ingestion is unknown or the ingestion occurred over multiple days or hours, the Rumack-Matthew nomogram cannot be used. The clinician must use the history and laboratory results to determine if the patient is at risk for hepatic injury.

Diagnostic Testing

- **APAP serum level at 4 hours** after ingestion or later (see above).
- **LFT, international normalized ratio, coagulation tests**—aspartate aminotransferase (AST) is a relatively sensitive nonprognostic marker for hepatic injury.

TREATMENT

- ***N*-Acetylcysteine (NAC):** NAC is the antidote to prevent APAP-related hepatotoxicity (*Toxicol Sci 2004;80(2):343*). It should be administered early (i.e., within 8 hours after ingestion) to prevent liver injury but still offers some protection if its administration is delayed (*N Engl J Med 1988;319(24):1557*).
- NAC can be administered either orally or IV.

- **Oral dosing:** Loading dose of 140 mg/kg PO, then 70 mg/kg PO every 4 hours for a total of 17 doses.
- **IV dosing:** Per the package insert, NAC is administered using a three-bag approach: bag 1: 150 mg/kg over 1 hour; bag 2: 50 mg/kg administered over 4 hours; bag 3: 100 mg/kg over 16 hours. The infusion is then continued if there are signs of toxicity. To simplify the approach, hospitals have developed their own protocols. At Barnes-Jewish Hospital, the protocol is to administer the loading dose over 1 hour followed by a continuous infusion at 14 mg/kg/h for the next 20 hours.
- **NAC indications:** NAC treatment should be started in the following:
  - Any patient after acute poisoning with a toxic APAP level according to the nomogram.
  - Patients who present beyond 8 hours after acute ingestion. Start NAC therapy while awaiting the initial APAP serum concentration and LFTs. Continue treatment if the serum concentration is in the toxic range per nomogram or the LFTs are elevated.
  - Patients who present more than 24 hours after acute ingestion and still have a detectable serum APAP level or elevated AST.
  - Patients with chronic APAP exposure (i.e., >4 g/d in adults, >120 mg/kg/d in children) who present with elevated acetaminophen concentrations and transaminases or a concerning history.
  - Patients with signs of fulminant hepatic failure. NAC treatment should be started immediately and transfer to a transplant center arranged without fail. NAC is shown to improve survival of patients in fulminant hepatic failure (*Lancet 1990;335(8705):1572*; *N Engl J Med 1991;324(26):1852*; *BMJ 1991;303(6809):1026*).

Opioids

DIAGNOSIS

Clinical Presentation

Symptoms of opioid overdose are respiratory depression, a depressed level of consciousness, and **miosis**. However, the pupils may be dilated with acidosis or hypoxia or following an overdose with meperidine, propoxyphene, or dextromethorphan.

Diagnostic Testing

Laboratories

Drug concentrations and other standard laboratory tests are of little use. Urine drug screens are associated with multiple false positives and negatives. Opioid intoxication is a clinical diagnosis.

Imaging

A CXR should be obtained if pulmonary symptoms are present or there is concern for aspiration.

TREATMENT

- Treatment includes airway maintenance, ventilatory support, and naloxone, an opioid antagonist.
- Limit use of whole-bowel irrigation to body packers. Body packers rarely require surgery, except in cases of intestinal obstruction. This should only be done in consultation with a poison center or medical toxicologist.

Medications

- **Naloxone hydrochloride** is indicated for opioid-induced respiratory depression. It should not be used to reverse decreased mental status.

- The lowest effective dose should be used. The goal of treatment is adequate spontaneous respiration and not necessarily alertness. The initial dose is 0.04–2 mg IV, although the lowest effective dose should be used.
- Larger doses (up to 10 mg IV) may be required to reverse the effects of methadone.
- If multiple doses of naloxone are required, an IV infusion should be initiated. The infusion should be started at two-thirds of the dose required to reverse respiratory depression.
- In the absence of an IV line, naloxone can be administered sublingually (*Ann Emerg Med 1987;16:572*), intranasally (*Emerg Med J 2006;23:221*), or IM. Isolated opioid overdose is unlikely if there is no response to a total of 10 mg of naloxone.
- **Disposition**
  - Patients should be observed for at least an hour following naloxone administration.
  - Patients requiring a naloxone infusion should be admitted to an ICU.
  - Body packers should be admitted to an ICU for close monitoring of the respiratory rate and level of consciousness and remain in the ICU until all packets have passed, as documented by CT.

Salicylates

GENERAL PRINCIPLES

Definition

- Salicylate toxicity may result from **acute or chronic** ingestion of acetylsalicylic acid (aspirin is a generic name in the United States, but a brand name in the rest of the world).
- Toxicity from chronic ingestion typically occurs in elderly patients with chronic underlying medical conditions. They can present similarly to patients with sepsis.

DIAGNOSIS

Clinical Presentation

- Nausea, vomiting, tinnitus or hearing changes, tachypnea, tachycardia, diaphoresis, hyperpnea, and malaise are common in acute toxicity.
- Severe intoxications may include lethargy, noncardiogenic pulmonary edema, seizures, and coma, which may result from cerebral edema and energy depletion in the CNS.

Diagnostic Testing

- Obtain electrolytes, BUN, creatinine, and glucose.
- Obtain either ABGs or venous blood gases.
- Obtain a serum salicylate concentration. Patients generally require treatment for concentrations >30 mg/dL. **Note: Units may be different at other institutions. For the purposes of this chapter, salicylate concentrations are in mg/dL.**
- Salicylate concentrations >100 mg/dL are very serious and often fatal.
- Salicylate concentrations of 10–30 mg/dL often do not require treatment. However, patients should receive serial evaluations to make sure that the concentration is appropriately decreasing.
- Chronic ingestion can cause toxicity at lower salicylate concentrations than acute ingestions.

TREATMENT

Medications

- **Multidose charcoal** may be useful in severe overdose (*Pediatrics 1990;85:594*) or in cases in which salicylate concentrations fail to decline as absorption tends to be delayed due to bezoar formation and pylorospasm.

- Patients are often volume depleted and require 1–2 L of normal saline.
- **Urine alkalinization** is indicated for patients with salicylate concentrations >30 mg/dL.
- Administer 150 mEq (three ampules) sodium bicarbonate in 1000 mL 5% dextrose in water (D5W) at 1.5–2 times maintenance.
- Maintain alkalinization and titrate to a goal urine pH of 7.5–8. Patients with hypokalemia cannot effectively have their urine alkalinized.
- **Use caution in patients who cannot handle large volumes of fluid such as the elderly, patients with renal failure, patients with heart failure, or patients with cerebral or pulmonary edema.**
- Alkalinization can be stopped once the serum concentration is <30 mg/dL. The patient should have a repeat salicylate concentration drawn 4–6 hours after all treatment is stopped. If it is declining appropriately (approximately half of previous concentration), the patient does not require further treatment.
- **Hyperventilate any patient requiring endotracheal intubation.** Intubation should be avoided if at all possible in these patients, because they require complex ventilatory settings. The ventilator should be set at their maximal respiratory rate. Any worsening of their acidosis due to improper ventilator settings can result in rapid deterioration and death.
- **Treat altered mental status with IV dextrose** even with a normal blood glucose.
- Treat **seizures** with a **benzodiazepine**. Standard antiepileptics will not be effective.

Hemodialysis

Indications include:

- Salicylate concentrations >100 mg/dL in acute toxicity
- Salicylate concentrations >80 mg/dL or rising despite treatment
- Salicylate concentrations >60 mg/dL in chronic toxicity
- Patients with pulmonary edema, cerebral edema, or seizures
- Patients requiring intubation
- Patients who cannot receive large amounts of fluid and have potentially toxic ingestions