Emergency Medicine

7

Jo-Ann O. Nesiama, Jennifer McConnell, and Kenneth Yen

RESPIRATORY DISTRESS

Definitions

- Respiratory distress
  - Interruption of the respiratory tract or the systems that control respiration
  - One of the most common pediatric complaints in the emergency room
- Respiratory failure
  - Inability of the respiratory system to meet metabolic demand for oxygenation or ventilation
  - Prominent cause of pediatric deaths, particularly in infants
  - Respiratory failure is a primary cause of pediatric cardiac arrest

Special Considerations in the Anatomy of the Pediatric Airway

- Large occiput
  - Causes flexion of airway when supine
- Large tongue
  - May obstruct posterior pharynx when supine
- Obligate nose breathers (particularly < 4 months)
  - Nasal suctioning can significantly relieve respiratory distress due to nasal congestion
- Omega-shaped and floppy epiglottis
  - Structures easily collapse, and are not as rigid as adult airway structures
- Anterior airway with higher glottic opening at C2–C3 (versus C4–C5 in adults)
  - More difficult to visualize larynx during intubation
- Significant anatomic variation with age
  - Shorter trachea
    - Technically difficult intubation
    - Easy to insert endotracheal tube too far (right mainstem intubation)
    - Easy to lose artificial airway (dislodged tube, esophageal intubation)
- Smaller tracheal diameter:
  - Small increase in tracheal thickness causes disproportionately larger obstruction
    - Poiseuille's law: Resistance varies inversely with fourth power of the radius
      - Example: 1 mm thickening of trachea decreases tracheal diameter by 20% in an adult and 80% in a small child
- Narrowest portion of airway at cricoid ring (versus at vocal cords in adults)
  - Uncuffed endotracheal tubes may provide adequate seal at "natural" cuff

J.-A. O. Nesiama · J. McConnell · K. Yen (✉)
Division of Pediatric Emergency Medicine, Department of Pediatrics, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Ken.yen@utsouthwestern.edu

O. I. Naga (ed.), *Pediatric Board Study Guide*, https://doi.org/10.1007/978-3-030-21267-4_7

- Small cricothyroid membrane
  - Needle cricothyrotomy difficult
  - Surgical cricothyrotomy impossible in small children

Localizing physical findings

- A careful physical can help rapidly localize the etiology of distress (Table 7.1)

Common causes of respiratory distress by anatomical location

- Many causes of respiratory distress in pediatric patients are commonly encountered and have well-defined clinical syndromes

Upper airway

- Upper airway obstruction

Table 7.1 Physical exam findings seen in pediatric patients in respiratory distress

Physical finding	Description	Physiology	Important notes
Retractions	Accessory muscle usage (supraclavicular, intercostal, abdominal)	Counteract high negative intrathoracic pressure via increased respiratory effort	Generalized finding of respiratory distress
Head bobbing	Flexion–extension movement of head and neck during inspiration and expiration	Emerges due to severe accessory muscle use, seen particularly in small children and infants	Indicative of severe respiratory distress, potential impending respiratory failure
Flaring	Widening of the lateral nares	Increases the upper airway diameter as an attempt to help relieve obstruction	Often a late finding of respiratory distress seen in small children and infants
Stertor	Low pitched, loud, rumbling, snoring sound	Upper airway obstruction: may be due to large tongue, tonsils, or adenoids; poor muscle tone; or altered mental status	Can improve with repositioning ("sniffing position") or jaw thrust
"Hot potato" voice	Muffled, soft voice	Obstruction of upper airway, typically oropharynx or pharynx	May be seen in retropharyngeal abscess or peritonsillar abscess
Hoarseness	Rough or harsh characteristic to voice	Obstruction or abnormality of vocal cords or larynx	Can indicate benign/minor pathology such as viral URI or concerning pathology such as injury to vagus or recurrent laryngeal nerve
Barky cough	Loud, "seal-" or "dog-like" hacking cough	Pathology or obstruction of subglottic area	Commonly seen in acute viral croup
Stridor	Harsh multiphonic, high-pitched upper airway noise, "noisy breathing"	Obstruction of upper airway results in turbulence and subsequent noise; inspiratory stridor most commonly glottic/subglottic in origin, expiratory stridor more likely lower airway, e.g., carina	Can indicate acute viral croup, or other concerning pathology such as aspirated foreign body or epiglottitis
Grunting	Soft, quick "puffing" expiratory noise	Expiration against partly closed glottis, attempt to maintain lung volume and prevent atelectasis via "auto-PEEP"	Often late finding of respiratory distress, seen in small children and infants
Wheezing	Musical, continuous noise	Expiratory wheezing indicates bronchi and bronchiolar obstruction	Expiratory wheezing commonly heard in asthma and bronchiolitis
Rales (fine crackles)	Inspiratory, high-pitched, "Velcro-like" sounds	Opening of collapsed alveoli filled with secretions, indicate pathology at lung tissue level	Typically will not clear with repositioning and coughing
Rhonchi (coarse crackles)	Inspiratory, mid- to low-pitched "popping" sounds	Turbulence and secretions from secretions or inflammation within bronchi and bronchioles	More likely to clear with repositioning and coughing

URI upper respiratory infection

  - Anaphylaxis (hives, angioedema, atopy, history of exposure to antigen, stridor, hoarseness)
  - Nasal congestion/obstruction (congestion, rhinorrhea, concurrent upper respiratory infection [URI])
  - Foreign body (history of coughing or choking event, stridor, drooling)
  - Congenital/developmental airway anomalies (choanal atresia, adenotonsillar hypertrophy, laryngotracheomalacia, subglottic stenosis/web/hemangioma, branchial cleft abnormalities)
- Upper airway infection
  - Croup (URI symptoms, barky cough, fever, inspiratory stridor)
  - Epiglottitis (toxic appearance, fever, dysphagia, drooling, inspiratory stridor)
  - Peritonsillar abscess (fever, sore throat, trismus, dysphagia, drooling, vocal changes, uvular displacement)
  - Retropharyngeal abscess (fever, dysphagia, drooling, vocal changes, neck stiffness/pain with extension, torticollis)
  - Tracheitis (toxic appearance, fever, stridor—similar appearance to epiglottitis, usually will have risk factors)

Lower airway

- Lower airway obstruction
  - Anaphylaxis (hives, angioedema, atopy, history of exposure to antigen, wheezing)
  - Asthma/reactive airway disease (atopy, history of bronchodilator use, expiratory wheezing, prolonged inspiratory to expiratory ratio)
  - Bronchiolitis (previously healthy, no prior wheezing, concurrent URI symptoms)
  - Foreign body
  - Tracheobronchomalacia (recurrent stridor or noisy breathing, acute or chronic exacerbations with concurrent URI)
- Lower airway infection
  - Pneumonia (cough, tachypnea, fever)

Extrapulmonary

- Mediastinal masses (orthopnea, B symptoms, hoarseness, hemoptysis, lymphadenopathy)
- Pericardial tamponade (history of trauma, orthopnea, hypotension, jugular vein distention, pulsus paradoxus, muffled heart sounds)
- Pleural effusion (risk factors such as pneumonia/chemotherapy/autoimmune disorders, orthopnea, pleuritic pain)
- Pneumothorax/tension pneumothorax (possible history of trauma or spontaneous sudden onset, unilateral absent breath sounds, possible hypotension, deviated trachea with mediastinal shift if tension is present)

An approach to the differential diagnosis by clinical syndrome

Respiratory distress with signs of upper airway obstruction (stridor, stertor, vocal change, dysphagia, drooling):

- If acute onset with fever, consider infection
  - Croup, peritonsillar abscess, retropharyngeal abscess, tracheitis, epiglottitis
- If acute onset without fever, consider
  - Anaphylaxis, foreign body
- If chronic, consider
  - Masses, congenital/developmental abnormalities such as tonsillar hypertrophy, vocal cord dysfunction, laryngotracheomalacia, psychogenic causes

Respiratory distress with signs of lower airway pathology (wheezes, rales):

- If acute onset with presence of fever, consider infection/inflammation
  - Bronchiolitis, pneumonia, subacute foreign body, myocarditis
- If acute onset without fever, consider
  - Asthma, bronchiolitis, viral/atypical pneumonia, foreign body aspiration, anaphylaxis

Respiratory distress with no signs of airway obstruction, with the presence of tachypnea:

- If acute onset tachypnea with fever, broaden the differential to include a more systemic illness

  - Pneumonia, subacute foreign body, pulmonary embolism, myocarditis, pericarditis, sepsis
- If acute-onset tachypnea without fever and with concern for cardiac abnormality (arrhythmia, rubs, gallops, new murmurs, hepatomegaly, poor perfusion), consider
  - Congenital heart disease, myocarditis, pericarditis, pericardial effusion/tamponade, congestive heart failure, pleural effusion
- If acute-onset tachypnea without fever and no concern for cardiac abnormality:
  - Very large differential diagnosis, obtain thorough history and physical
    - Respiratory disorder (pneumonia, atelectasis, pulmonary embolism, pulmonary deformity or mass)
    - Metabolic (acidosis, hyperammonemia, hyperglycemia, hepatic/renal disease)
    - Toxic (ingestions, methemoglobinemia)
    - CNS disorder (seizure, mass, encephalopathy, neuromuscular disease, anxiety, pain)
    - Intra-abdominal pathology (abdominal pain, distention, mass)
    - Hematologic (anemia, methemoglobinemia)

Initial Emergency Care for Patient in Acute Respiratory Distress

Airway management

- Leading cause of pediatric cardiac arrest is from respiratory failure
- Timely management of the pediatric airway is key to resuscitation of pediatric patients with acute respiratory distress

Categorize the airway

- Airway is clear: Airway is open and non-obstructed for normal breathing
  - Patient is able to vocalize clearly (speaking or loud crying)
- Airway is maintainable: Airway is obstructed but maintained with simple measures (Table 7.2)
  - Patient able to vocalize, but abnormally (stertor, stridor, choking, coughing, dysphonia, etc.)
- Airway is not maintainable: Airway is obstructed and requires advanced intervention such as intubation
  - Unable to speak or absence of cry (gurgling, gasping, cyanosis, loss of consciousness, extreme agitation, etc.)

Table 7.2 Initial simple airway maneuvers for patients in acute respiratory distress

Airway maneuvers	Description
Airway clearing	Suctioning oro-/nasopharynx to remove secretions or debris
	Nasopharyngeal airway
	Oral airway in patient with altered mental status
Airway positioning	Allow an awake child to assume position of comfort (e.g., tripoding)
	Head tilt and chin lift ("sniffing" position) to compensate for large occiput
	Jaw thrust/chin lift to open airway and bring tongue forward
	Shoulder roll to prevent forward flexion of cranium due to large occiput

Breathing management

- Pediatric patients are less tolerant of hypoxemia and hypercarbia
  - Higher metabolic rate means more metabolic demand

Support oxygenation and ventilation

- Provide supplemental oxygen for hypoxemia
- Provide ventilatory support for hypercarbia or inadequate/absent respiratory effort
- Make timely interventions for patient in acute respiratory distress (Table 7.3) (see Chap. 8 "Critical Care" for additional details)

Table 7.3 Initial interventions in resuscitation of patients in acute respiratory distress

Intervention	Common uses
Nasal cannula	Hypoxemia alone, will dry nasal mucosa over time
Simple face mask	Hypoxemia alone, significant room air entrainment and mixing
Non-rebreather face mask	Hypoxemia alone, allows for greater FIO2 via a reservoir
Heated high flow nasal cannula	Hypoxemia and increased respiratory effort, allows humidification and heating, higher oxygen flow rates
Noninvasive positive pressure ventilation	Hypoxemia and hypercarbia in select patients with respiratory failure but adequate airway protection and mentation; allows for positive pressure delivery via sealed mask
Bag–valve–mask ventilation	Hypoxemia and hypercarbia, insufficient/absent respiratory effort, a temporizing but life-saving measure while definitive airway/ventilatory support is being planned
Inhaled bronchodilators: albuterol and ipratropium	States of reversible bronchospasm (i.e., asthma exacerbation)
Nebulized racemic epinephrine	Suspected upper airway obstruction and edema with stridor (i.e., acute infectious croup)
Intramuscular epinephrine	Suspected anaphylaxis

Summary

- Correction of respiratory distress ultimately needs identification of the underlying cause
- Any disorder that causes respiratory distress has the potential to be life-threatening
- Remember the unique characteristics of the pediatric airway
- Initial resuscitation of the patient in acute respiratory distress requires attention to airway, breathing, and circulation

THE ACUTE ABDOMEN

The following section will focus on the presentation of the pediatric acute abdomen in the setting of emergency care, with a particular emphasis on the identification of surgical abdominal emergencies. Abdominal emergencies in the setting of pediatric abdominal trauma will be covered in the Trauma and Burns section.

The Pediatric Acute Abdomen

- Abdominal complaints in pediatric patients are common and often benign
- Surgical abdominal emergencies must not be missed, due to high morbidity and mortality, and require prompt recognition and timely surgical evaluation

Findings that may indicate a surgical abdomen

- Bilious emesis
  - Bilious emesis in infants is a surgical emergency
- Guarding
  - Active guarding
  - Passive guarding/rigidity
- Distention
- Bowel sounds
  - Absent bowel sounds may indicate ileus
- Tenderness
  - Rebound tenderness is indicative of peritonitis
- Associated symptoms
  - Fever can signify a systemic inflammatory response
  - Bloody diarrhea can signify bowel wall necrosis and malperfusion

Age-dependent presentation

The age of the presentation can significantly shape the differential diagnosis

- Infant: Maintain higher suspicion for congenital anomalies
  - Intussusception, malrotation with midgut volvulus, incarcerated hernias, Meckel diverticulum, pyloric stenosis
- School-aged child: Infectious causes become increasingly common
  - Acute appendicitis
- Adolescent:
  - Acute appendicitis, ectopic pregnancy, ovarian torsion, testicular torsion

General Principles of Management

- Definitive treatment must be targeted at the etiology of the acute abdomen
- There are general treatment principles that can be applicable in most cases

Initial resuscitation

- Attend to the ABCs (airway, breathing, circulation)
  - Be alert for signs of shock and poor perfusion
  - Restore intravascular volume as clinically appropriate
- Pain control
  - Use an appropriate regimen to treat escalating degrees of pain
  - If warranted, opioids can be safely used in pediatric patients
- *Nil* per os (NPO) status
  - Do not allow the patient to eat or drink
- For active bilious emesis and abdominal distention, consider decompression
  - Nasogastric tube
- Early surgical consultation
  - In the presence of suspicion for an acute surgical abdomen, prompt and early surgical consultation is important and should not be delayed
- Imaging options
  - Abdominal plain radiographs can be helpful in the evaluation of an acute abdominal obstruction, foreign body, bowel perforation, or constipation
    - Identification of free air
    - Air-fluid levels indicating ileus
    - Dilated loops of bowel in obstruction
    - Radiopaque ingested foreign body
    - Evaluation of stool burden
  - Ultrasonography is the preferred first line in many cases
    - Acute appendicitis, intussusception, ovarian torsion, pyloric stenosis, cholecystitis, pancreatitis, nephrolithiasis, pregnancy
  - Computed tomography (CT) imaging
    - CT of the abdomen/pelvis is the radiation exposure equivalent of more than 100 plain radiographs of the chest
    - CT of the abdomen/pelvis increases risk of radiation-induced solid cancers in children
    - Due to radiation exposure risks:
      - CT is not recommended in the routine evaluation of abdominal pain
      - Ultrasound should be considered first in the evaluation of acute appendicitis in children
  - Magnetic resonance imaging (MRI) will typically not be obtained in the emergency setting for the evaluation of abdominal pain

Select surgical abdominal emergencies (Table 7.4)

Nonsurgical Causes for an Acute Abdomen

- Due to the small size of pediatric patients and possibility for referred pain, many extraintestinal or nonsurgical diseases can present with an acute abdomen and acute abdominal pain
- Abdominal
  - Gastroenteritis, viral or bacterial
  - Constipation
  - Mesenteric adenitis
  - Gastritis/peptic ulcer disease
  - Pancreatitis
  - Gallbladder disease
  - Hepatitis
- Head, eyes, ears, nose, and throat (HEENT)
  - Streptococcal pharyngitis
  - Infectious mononucleosis
- Pulmonary
  - Pneumonia
- Cardiac
  - Pericarditis, myocarditis
- Renal
  - Spontaneous bacterial peritonitis (with peritoneal dialysis)

Table 7.4 Selected surgical abdominal emergencies, clinical features, and management

Features	Disease process	Clinical pearls	Clinical pitfalls	Initial management
Periumbilical pain migrating to the right lower quadrant at McBurney's point	Acute appendicitis	Most common abdominal emergency in children, with peak incidence between 9 and 12 years; many will have pain with walking or jumping; may also have anorexia, vomiting, diarrhea, and fever	The anatomic location of the appendix can vary, causing nonclassical sites of pain	Fluid resuscitation, pain control, appendix ultrasound, surgical consultation
Suspected acute appendicitis followed by sudden relief of pain, then development of generalized peritonitis	Perforated appendicitis	More likely to occur if appendicitis present for > 72 h, will often present with increasing signs of peritonitis and toxicity	Although appendicitis in infants is rare, the young child is more likely to present with perforation due to difficulty in the abdominal exam early on	Fluid resuscitation, pain control, appendix ultrasound, surgical consultation
Extreme colicky pain with periods of normalcy, currant jelly stools	Intussusception	Typically, 3 months to 6 years old, invagination of bowel at lead point, most common is ileocolic	Intussusception can present with emesis and altered mental status alone	Fluid resuscitation, ileocolic ultrasound, barium or air contrast enema, surgical consultation if unsuccessful, anticipatory guidance as intussusception can recur
Tense inguinal bulge, vomiting	Incarcerated inguinal hernia	Common cause of intestinal obstruction in infants and children, 60% occurring within the 1st year of life	May not have known prior history of hernia; incarceration can progress to strangulation and bowel necrosis within 24 h	May need inguinal ultrasound or abdominal radiograph if unclear diagnosis; otherwise, attempt immediate manual reduction if no sign of bowel necrosis, and early surgical consultation
Infant, projectile nonbilious emesis, hungry after emesis	Pyloric stenosis	Hypertrophied pylorus in infant 2–5 weeks old, occurs in 1 in 250 births, more common in first-born males, can cause hypokalemic, hypochloremic metabolic alkalosis	Does not present with acute abdomen, but due to potential for profound electrolyte disturbance, prompt diagnosis is important; alkalosis can be severe enough to cause apnea	Fluid resuscitation, careful electrolyte management, pyloric ultrasound, surgical consultation
Acute-onset bilious emesis, abdominal pain	Malrotation with midgut volvulus	Most serious cause of intestinal obstruction; congenital malrotation with abnormal fixation of bowel predisposes it to volvulize and obstruct; usually presents neonatally; however, 25% present > 1 year	Variable presentation, from asymptomatic to failure to thrive; complete volvulus for > 1 h causes gut necrosis; a midgut volvulus can cause death of entire small bowel and ascending colon	Resuscitation in case of signs of shock, flat and upright radiographs of abdomen, upper GI series, immediate surgical consultation

(continued)

Table 7.4 (continued)

Features	Disease process	Clinical pearls	Clinical pitfalls	Initial management
Bilious emesis, abdominal distention, significant abdominal surgical history	Surgical adhesions with obstruction	Any child with prior abdominal surgery or peritonitis can develop adhesions; requires low threshold of suspicion	Presentation can occur within days, or months to years after surgery	Fluid resuscitation, abdominal radiographs with more than one view (e.g., flat, upright, and decubitus), gastric decompression, early surgical consultation
Abdominal distention with systemic signs of illness (lethargy, apnea, temperature instability) in neonate	Necrotizing enterocolitis	Typically presents in premature infants or neonates within 10 days of birth, may have history of birth asphyxia or stress	Although most common in premature infants, can occur in term infants	Resuscitation if signs of shock, radiographs of abdomen, prompt surgical and neonatal subspecialty consultation

- Genitourinary
  - Testicular torsion
  - Ovarian torsion
  - Ectopic pregnancy
  - Pelvic inflammatory disease
  - Dysmenorrhea
- Urinary
  - Cystitis, pyelonephritis
  - Nephro-/urolithiasis
- Endocrine/metabolic
  - Diabetic ketoacidosis
- Autoimmune/inflammatory
  - Inflammatory bowel disease
  - Henoch–Schönlein purpura

Summary

- The differential diagnosis of the acute abdomen in pediatric patients is broad, ranging in nature from benign to life-threatening
- A careful history and physical are important to identify surgical emergencies
- Infants present a particularly diagnostic challenge due to difficulty of exam and nonspecific nature of presentation
- Maintain a low threshold for early surgical consultation

ANAPHYLAXIS

Definition

- Serious, systemic, rapid-onset allergic/hypersensitivity reaction
- Variable clinical presentation and severity
- Immunoglobulin E (IgE)-mediated, type I hypersensitivity reaction to antigen

Pathophysiology

- Antigen exposure leads to systemic mast cell and basophil degranulation with large histamine and cytokine release
  - Vasodilation
  - Smooth muscle spasm (can also cause coronary artery vasospasm)
  - Increased vascular permeability
  - End stage: Loss of intravascular volume and hypotension
- Common likely antigens
  - Food allergies (most common in pediatrics)
    - Peanut
    - Tree nuts (cashews, pecans, walnuts, etc.)
    - Milk
    - Wheat
    - Soy
    - Seafood (crustaceans)
    - Fruit

- Other miscellaneous allergens
  - Antibiotics (penicillins, cephalosporins, sulfonamides)
  - Insect stings (*Hymenoptera*, fire ants)

Diagnosis

- Primarily a clinical diagnosis
- If the presentation is unclear, a serum tryptase can help assist future subspecialty management

Clinical criteria

- Anaphylaxis is highly likely with any of the three clinical syndromes, in the context of an exposure to a likely antigen:
  1. Acute onset of illness with skin and/or mucosal symptom, and with one of the following symptoms:
     - Respiratory compromise (such as wheezing or stridor)
     - Hypotension or altered mental status (AMS)/syncope ***or***
  2. Two or more symptoms of the following occurring acutely after exposure to likely antigen:
     - Skin/mucosa (90%) (urticaria, angioedema, flushing)
     - Respiratory (50–70%) (rhinorrhea, oropharyngeal/laryngeal edema, hoarseness, stridor, wheezing, shortness of breath)
     - Gastrointestinal (40%) (nausea, abdominal pain, diarrhea, vomiting)
     - Circulatory (30%) (hypotension, tachycardia)
     - Neurologic (syncope, sense of impending doom, seizures, AMS) ***or***
  3. Age-specific decrease in systolic blood pressure (BP) > 30% from normal or hypotension

Biphasic presentation

- Immediate phase: Occurs within minutes to hours after exposure
- Delayed phase/recurrence: Occurs from hours to days after exposure (up to 72 h)

Differential diagnosis

- Anaphylactoid reaction
  - Non-IgE-mediated anaphylaxis
  - Most commonly caused by aspirin, nonsteroidal anti-inflammatory drugs (NSAIDS), or radiographic contrast
  - Clinically indistinguishable from anaphylaxis
  - Treatment is identical to anaphylaxis

Treatment

- Always attend to the ABCs
- Intramuscular epinephrine is the first-line treatment of choice in anaphylaxis and must be administered immediately
  - Dosage: 0.01 mg/kg intramuscular
    - Maximum of 0.3 mg in prepubertal child
    - Maximum of 0.5 mg in adolescent
  - Timing: Immediate
    - Repeat at 5- to 15-min intervals
    - Up to 19% of patients will need a second intramuscular dose
  - Location: Mid-lateral thigh (vastus lateralis)
  - Autoinjector dosing options
    - Autoinjectors can come in 0.1 mg, 0.15 mg, and 0.3 mg forms:
      - 0.1 mg (7.5–14 kg)
      - 0.15 mg (15–30 kg)
      - 0.3 mg (>30 kg)
  - Pharmacokinetics
    - Alpha- and beta-adrenergic agonist induces bronchodilation, vasoconstriction, and decreased vascular permeability
    - Effectively treating upper airway edema, hypotension, and shock in addition to bronchodilator and positive cardiac inotropic and chronotropic effects

- Intramuscular epinephrine achieves peak concentration faster than subcutaneous injection
  - Intramuscular epinephrine is far safer than intravenous (IV) epinephrine
  - Serious adverse effects of appropriately dosed intramuscular epinephrine are rare
  - Side effects mimic signs of endogenous catecholamine release: Pallor, anxiety, tachycardia, tremor

- Adjuncts therapies: Do not prioritize administration of adjuncts over epinephrine
  - Diphenhydramine (H1 blockers)
  - Ranitidine (H-2 blockers)
  - Albuterol or racemic epinephrine
  - Glucocorticoids

Patient disposition

- Due to potential for rebound anaphylaxis and rapid deterioration, careful consideration of patient risk factors must be made at time of disposition from emergency care
- Risk factors that may necessitate admission to the hospital for observation:
  - First presentation
  - Infants
  - Concomitant asthma
  - Unknown antigen

Home care after anaphylaxis

- Counseling and follow-up are key for safe disposition home after anaphylaxis
  - Avoidance of identified allergen
  - Discussion of signs and symptoms of anaphylaxis
  - Epinephrine auto-injector instruction
    - Because anaphylaxis has highly variable clinical presentation between patients and between episodes, it is not possible to predict disease severity
    - All patients with anaphylaxis should be prescribed an epinephrine auto-injector
    - High-risk patients:
      - Coexisting asthma
      - Reaction to trace amounts of food
      - Idiopathic anaphylaxis
      - Generalized urticaria from insect sting (higher risk of more severe reaction)
      - Patient lives in a remote or rural area
  - Referral to an allergy/immunology specialist

TRAUMA AND BURNS

General principles of pediatric trauma

- Primary survey (identify and treat any life-threatening problems)
  - **A** Airway maintenance
  - **B** Breathing and ventilation
  - **C** Circulation with hemorrhage control
  - **D** Disability; neurologic status (Glasgow Coma Scale [GCS], pupils)
  - **E** Exposure/environment (remove clothing and keep normal body temperature)
- Secondary survey
  - Vital signs
  - Detailed head-to-toe examination
  - History of traumatic event
    - AMPLE history: Allergies, medications, past illnesses, last meal, events or environment related to injury

Pediatric Head Trauma

- Most pediatric head trauma is not serious and requires only observation
- Identification of serious head trauma in pediatric patients requires careful physical examination and understanding of warning signs

Red flags in pediatric head trauma

- Severe mechanisms of injury
  - Motor vehicle collision (MVC) with
    - Patient ejection
    - Fatalities
    - Rollover

  - Motor vehicle vs. pedestrian crash (MPC) or cyclist injury in those without helmets
  - Height of fall
    - Infants under 2 years with falls greater than 3 ft
    - Children 2 years and over with falls greater than 5 ft
  - High-impact or high-speed object striking head
- Patients at higher risk
  - Infants in general, but particularly if < 3 months
  - Known bleeding disorder or anticoagulated
  - Multisystem injuries
  - Suspected nonaccidental trauma
- Patient with findings that are higher risk
  - AMS or acute change in mental status
    - Lethargy
    - Irritability
    - Loss of consciousness
  - Persistent vomiting
  - Severe headache
  - Bulging fontanelle
  - Focal neurologic findings
  - Seizures

Presentation and Localizing Findings of Serious Injury

Basilar Skull Fracture

Clinical presentation

- Raccoon eyes (periorbital ecchymosis, bruising around eyes)
- Battle sign (mastoid ecchymosis, bruising behind the ears)
- Hemotympanum
- Cerebrospinal fluid (CSF) otorrhea or rhinorrhea
- At increased risk for meningitis
- 75% will have a temporal skull fracture

Evaluation and management

- CT scan to evaluate for other fractures and bleeding
- If nondisplaced, then usually heals without intervention
- At higher risk for meningitis, but the utility of prophylactic antibiotics is unclear

Temporal Skull Fracture

Clinical presentation

- Bleeding from ear or hemotympanum
- Facial paralysis
- CSF otorrhea and rhinorrhea
- Vestibular symptoms, vertigo
- Leads to conductive, sensorineural, or both types of hearing loss

Evaluation and management

- CT scan (evaluate for longitudinal or transverse type)
- MR angiography/venography (MRA/MRV), CT angiography/venography (CTA/CTV)
- Usually bedrest and raise head of bed initially
- Surgery if CSF still leaking after 1 week
- Facial paralysis, hearing loss, and vertigo are longer-term issues

Subdural Hematoma

- Bleeding between the inner layer of the dura mater and the arachnoid mater
  - Tearing of the bridging veins across the subdural space
  - Result of acceleration/deceleration mechanism (MVC, shaken babies)
  - Associated with cerebral contusion
  - If acute, the most lethal injury, but can be chronic, developing over days to weeks

Clinical presentation

- Gradually increasing headache and confusion

- Ataxia, slurred speech
- Loss of consciousness or fluctuating level of consciousness

Diagnosis and management

- CT scan (concave, crescent-shaped hematoma)
- Treatment ranges from monitoring to a small burr hole to drain the hematoma to an open craniotomy. Treatment depends on size and speed of growth

Epidural Hematoma

- Bleeding between the dura mater and the skull
  - Often occurs from a break in temporal bone with bleeding from the middle meningeal artery

Clinical presentation

- Classically causes loss of consciousness (LOC) after head injury, followed by brief regaining of consciousness (lucid period), and then LOC again
- Headache, confusion, vomiting
- With progression of bleed, pupils ipsilateral to injury become fixed and dilated (compression of CN III) and gaze is "down and out" (unopposed action of CN IV and VI)
- Seizures and weakness on contralateral side due to compression of crossed pyramidal pathways
- Final stage is tonsillar herniation and death

Diagnosis and management

- CT scan (biconvex, lens-shaped hematoma)
- Emergency burr hole and/or craniotomy

Subarachnoid Bleed

- Bleeding between the arachnoid membrane and pia mater
  - Result of head trauma or can occur spontaneously (e.g., ruptured cerebral aneurysm)

Clinical presentation

- Severe headache of rapid onset
- Vomiting
- Loss of consciousness
- Fever
- Seizures

Diagnosis and management

- CT scan
- Support until neurosurgery

Retinal Hemorrhage

- In an infant, this can indicate possible nonaccidental trauma

Considerations in the Use of Head Computed Tomography (CT)

- Children are at greater risk of developing malignancy as a result of radiation exposure
- Because pediatric head trauma is common and often benign, the use of CT imaging should be applied judiciously and after careful consideration
- The age of the patient, presence of other injuries, suspicion for nonaccidental trauma, and presence or absence of warning signs should all help guide the application of screening head CT (Table 7.5) [1]

Management of Clinically Significant Head Trauma

- Attention to the ABCs, with particularly focus on
  - Protection and establishment of a secure airway
    - Particularly if unresponsive or GCS less than 8
  - Close hemodynamic monitoring and repeat neurologic assessments
  - Avoidance of hypothermia or hyperthermia

Table 7.5 Prediction rules for identification of children at low risk for clinically important traumatic brain injury found on computed tomography (CT) imaging of the head

Age is less than 2 years old	**CT is not recommended if the following criteria are met (risk of clinically important TBI is low)**	**Observation vs. CT recommended if any of the following are present (risk of clinically important TBI is slightly elevated)**	**CT recommended if any of the following are present (risk of clinically important TBI is high)**
	GCS 15 and no altered mental status	Occipital, parietal, or temporal hematoma	GCS less than 15 or altered mental status
	No palpable skull fracture	History of loss of consciousness	Palpable skull fracture
	Frontal hematoma only	Severe mechanism of injury	
	No loss of consciousness	Not acting normally per caretaker	
	Nonsevere mechanism of injury		
	Acting normally per caretaker		
Age is equal to or greater than 2 years old	**CT is not recommended if the following criteria are met (risk of clinically important TBI is low)**	**Observation versus CT recommended if any of the following are present (risk of clinically important TBI is slightly elevated)**	**CT recommended if any of the following are present (risk of clinically important TBI is high)**
	GCS 15 and no altered mental status	History of loss of consciousness	GCS less than 15 or altered mental status
	No signs of basilar skull fracture	History of vomiting	Signs of basilar skull fracture
	No loss of consciousness	Severe mechanism of injury	
	No vomiting	Severe headache	
	Nonsevere mechanism of injury		
	No severe headache		

Pediatric Emergency Care Applied Research Network validated clinical prediction rules; adapted from Kuppermann et al. [1], with permission
TBI traumatic brain injury, *GCS* Glasgow coma scale

  - Monitoring for signs of hypovolemic or neurogenic shock
  - Support to maintain cerebral perfusion pressure (CPP)
    - CPP = mean arterial pressure (MAP) – intracranial pressure (ICP) (or central venous pressure [CVP])
- Watch for clinical signs of increasing ICP
  - Unequal pupil dilation (pressure on cranial nerves)
  - Abnormal posturing
  - Cushing's triad: Irregular, decreased respirations (caused by impaired brainstem function), bradycardia, and systolic hypertension (widening pulse pressure)
- Management of increasing ICP
  - Mild hyperventilation
  - Intravenous mannitol or hypertonic saline (3%) may be needed
  - Immediate consultation to neurosurgery can be life-saving for serious intracranial injuries

Neck/Cervical Spine Trauma

- Penetrating neck injuries generally all need surgical evaluation
- Blunt neck injury may warrant further evaluation for vessel injury and cervical spine injury

Cervical Spine Injury

- Relatively uncommon
- MVC, sports, and falls are most common etiologies
- Children have different injury patterns because of increased physiologic motion due to:

  - Larger size of head compared to trunk, which creates a larger fulcrum
  - Horizontally oriented facet joints
  - Elevated ligamentous laxity
  - Weaker muscles
- Children < 8 years old 87% of spinal injuries are above C3
- Children > 8 years old more commonly have lower cervical injury (adult injury pattern)

Clinical presentation

- Always suspect if head injury or facial fractures
- Full neurologic exam, but note that > 20% with injuries will have normal exam

Diagnosis and management

- Cervical spine neck radiograph
- CT
- MRI
- Proper cervical spine immobilization initially for all
- Some may need longer-term immobilization (halo) or surgery

Abdominal Trauma

- Children are smaller in size; organs are proportionally larger; less fatty tissue around major organs; weaker musculature; more compliant rib cage, all which increase risk of solid organ injury
- Most are MVC, others are MPC, sports, falls, and nonaccidental trauma
- Most common unrecognized fatal injury

Clinical manifestation

- Exams may be difficult due to age, verbal ability, and fear
- Tachycardia can be the only sign (even with 45% circulating blood volume)
- Have high index of suspicion for abdominal injuries
  - Seatbelt sign (ecchymosis of abdominal wall) after MVC has increased risk of intra-abdominal injuries, primarily due to increased risk of gastrointestinal (GI) injuries
  - Handlebar mark (handlebar-shaped ecchymosis over abdominal wall) at increased risk of small bowel hematoma and others

Diagnosis and management

- Complete blood count (CBC), lipase, aspartate transaminase (AST)/alanine transaminase (ALT) coagulation studies, uric acid (elevated AST/ALT combined with positive physical exam has good sensitivity)
- Abdominal radiograph to evaluate for free air
- CT scan (without contrast, with IV contrast, with IV and oral contrast)
- Focused assessment with sonography in trauma (FAST) exam is of unproven utility in pediatrics but combined either with physical exam or serial exams can have good sensitivity and specificity
- Conservative management for most, laparoscopy for some
- Laparotomy for significant injuries

PEDIATRIC WOUNDS AND LACERATIONS

Laceration

- A laceration is a traumatic disruption to the dermis layer of the skin.
- Common locations are the face (~60%) and upper extremities (~25%)
- Most lacerations in pediatric patients are non-life-threatening but require appropriate management
- Severe and life-threatening lacerations must be evaluated and treated promptly
  - Hemostasis: Apply pressure or tourniquet as necessary
  - Injury to deeper structures: Especially at high-risk areas like the neck (carotid) arter-

ies, jugular veins, trachea, and esophagus must be considered

Management and treatment

- Repair may be more difficult in pediatrics due to fear, anxiety, and lack of cooperativity
  - Anesthetic and anxiolytic options
    - Topical anesthetic LET (4% lidocaine, 1:2000 epinephrine, 0.5% tetracaine)
      - Effective 20–30 min after application
      - Blanching of the site after application most often indicates achievement of effective anesthesia
    - Local anesthetic may be used to prepare for placement of sutures
      - Injectable lidocaine alone or with epinephrine
    - Distraction and soothing techniques
      - Child life experts, toys, movies, music, etc.
    - Oral/intranasal pharmacologic options
    - Inhaled and IV pharmacologic options for procedural sedation
- Wound preparation
  - Wound irrigation is the standard of care
  - Evaluation for possible foreign bodies or complications
  - Suspicion for foreign body or associated bony fracture should prompt radiographs of the affected site
  - Tissue adhesive
    - With selection of appropriate type of laceration, tissue adhesive has good cosmetic outcome with benefit of no need for suture or suture removal
- Suture and staples
  - Selection of suture material (absorbable versus nonabsorbable) will depend upon the location and depth of the injury
  - As a general rule, sutures should be evenly spaced
- Wound aftercare
  - All patients should be given follow-up instructions for local wound care, cleansing, timing (if necessary) of removal of suture, monitoring for infection, and use of sunscreen to decrease scar formation
- Consider the need for tetanus prophylaxis
  - Prophylactic antibiotics not recommended with simple, uncontaminated lacerations
    - Lacerations due to bites will typically require antibiotics
    - Lacerations to hands and fingers with associated distal fingertip fracture

Specialized Scenarios

- Lip lacerations
  - An injury crossing the vermilion border will likely require subspecialist repair
  - Failure to align the vermilion border can result in a poor cosmetic outcome and permanent lip deformity
  - Young and uncooperative patients may additionally require varying degrees of anxiolysis and possibly sedation to repair well
- Nail bed lacerations
  - Repair of nail bed lacerations can be particularly painful and anxiety-provoking and may require a higher level of care
  - Approximately half of all nail bed injuries are associated with a fracture of the distal phalanx and will likely require subspecialist repair
    - Low threshold for obtaining plain radiographs of the digit to evaluate for associated fracture
    - Nail bed laceration with associated distal phalanx fracture may be an open fracture
      - Open fractures require antibiotic therapy
- Ear lacerations
  - Significant ear lacerations will likely require subspecialty repair
  - Lacerations of the ear have the potential to involve the avascular cartilaginous support

  - Timely repair of ear laceration and close follow-up are important to avoid complications
    - Auricular hematoma: Disruption to cartilage and underlying perichondrium can lead to a hematoma, which, if untreated, can cause cosmetic deformity of the ear ("cauliflower ear")
- Timing of suture removal
  - Timely removal of sutures reduces likelihood of suture tracks; better cosmetic outcome
  - Face: 3–5 days
  - Scalp: 5–7 days
  - Trunk: 5–7 days
  - Extremities: 7–10 days

Animal and Human Bites

- Dog bites
  - Typically causes a crushing-type wound
  - The extreme pressure of a dog bite may damage deeper structures such as bones, vessels, tendons, muscles, and nerves
  - Dog bites are contaminated and require antibiotic therapy
    - *Staphylococcus* species, *Eikenella* species, *Pasteurella* species
- Cat bites
  - The sharp pointed teeth of cats usually cause puncture wounds and lacerations that may inoculate bacteria deep into the tissues
  - Infections caused by cat bites generally develop faster than those of dogs
  - Cat bites are contaminated and require antibiotic therapy
    - *Pasteurella* species, *Bacteroides* species
- Other animals
  - Foxes, raccoons, skunks, and bats carry a high risk for rabies
  - Bat bites may be asymptomatic
    - If a bat is discovered within a patient's sleeping quarters, rabies treatment should be given regardless of history of a known bite
- Human bites
  - Three general types of injuries can lead to complications:
    - *Closed-fist injury*
    - *Chomping injury to the finger*
    - *Puncture*-type wounds about the head caused by clashing with a tooth
  - Human bites are contaminated and require antibiotic therapy
    - *Eikenella corrodens, Staphylococcus* species, *Streptococcus* species
    - Human bites can also transmit the following organisms: Hepatitis B, hepatitis C, herpes simplex virus (HSV), and syphilis

General evaluation

- Time and location of event
- Type of animal and its status (i.e., health, rabies vaccination history, behavior)
- Circumstances surrounding the bite (i.e., provoked or defensive bite versus unprovoked bite)
- Location of bites

General management

- Local public health authorities should be notified of all animal bites and may help with recommendations for rabies prophylaxis
- Consider tetanus and rabies prophylaxis for all wounds (Tables 7.6 and 7.7) [2, 3]

Table 7.6 Indications for tetanus vaccine and tetanus immune globulin in the United States

Vaccine status	Clean and minor wounds	Contaminated wounds (soil, dirt, feces, saliva)
Unknown or not up to date (< 3 doses)	Tetanus vaccine only	Tetanus vaccine and tetanus immune globulin
Series completed (> 3 doses) less than 5 years ago	No tetanus vaccine or immune globulin	No tetanus vaccine or immune globulin
Series completed but > 5 years since final dose	If > 10 years since final dose, tetanus vaccine only	If > 5 years since final dose, tetanus vaccine only

Centers for Disease Control and Prevention [CDC] Advisory Committee on Immunization Practices on Tetanus 2018. Adapted from Liang et al. [2]

Table 7.7 Indications for rabies prophylaxis and treatment in the United States

Vaccination status	Intervention	Description
No prior vaccine	Human rabies vaccine	Human rabies vaccine (human diploid cell vaccine) should be administered IM on days 0, 3, 7, and 14
	Human rabies immune globulin (HRIG)	Immune globulin (20 IU/kg) should be administered via direct infiltration around the wound, with any remaining volume administered IM at an anatomical site distant from the vaccine site (e.g., the opposite deltoid or lateral thigh)
Previously vaccinated	Human rabies vaccine	Human rabies vaccine (human diploid cell vaccine) should be administered IM on days 0 and 3
	Human rabies immune globulin (HRIG)	Not indicated

Centers for Disease Control and Prevention [CDC] Advisory Committee on Immunization Practices, 2010. Adapted from Rupprecht et al. [3]
IM intramuscular

Laboratory

- Fresh bite wounds without signs of infection do not need to be cultured
- Infected bite wounds should be cultured to guide antibiotic therapy

Imaging studies

- Radiography is indicated if any concerns exist that deep structures are at risk (e.g., hand wounds, deep punctures, crushing bites, especially over joints)

Antibiotic therapy

- All human and animal bites should be treated with antibiotics
- The choice between oral and parenteral antimicrobial agents should be based on the severity of the wound and on the clinical status of the victim
- Oral amoxicillin–clavulanate is the initial drug of choice for empiric oral therapy
- Amoxicillin alone does not provide adequate coverage
- Parenteral ampicillin–sulbactam is the drug of choice in severe cases
- Clindamycin in combination with trimethoprim/sulfamethoxazole can be given if penicillin allergic

Wound care

- Debridement and removal of devitalized tissue
- Irrigation is key to prevention of infection
- 100 ml of irrigation solution per centimeter of wound
- Primary closure may be considered in limited bite wounds that can be cleansed effectively (this excludes puncture wounds, i.e., cat bites)
- Other wounds are best treated by delayed primary closure

Snake Bites

- Most are nonpoisonous and are delivered by nonpoisonous species
- North America is home to 25 species of poisonous snakes
- Characteristics of most poisonous snakes:
  - Triangular head
  - Elliptical eyes
  - Pit between the eyes and nose
  - Examples: Rattlesnakes, cottonmouth and copperheads
  - Few snakes with round head are venomous
    - Example: Coral snakes ("red on yellow—kill a fellow")

Clinical presentation

- Local manifestations
  - Local swelling, pain, and paresthesias may be present
  - Soft pitting edema that generally develops over 6–12 h but may start within 5 min
  - Bullae
  - Streaking

  - Erythema or discoloration
  - Contusions
- Signs of systemic toxicity
  - Hypotension
  - Petechiae, epistaxis, hemoptysis
  - Paresthesias and dysesthesias—Indicate neuromuscular blockade and should be aware of possible respiratory distress (more common with coral snakes)
- The time elapsed since the bite is a necessary component of the history
  - Determine history of prior exposure to antivenin or snakebite (this increases risk and severity of anaphylaxis)
  - Assessment of vital signs, airway, breathing, and circulation

Evaluation and management

- Laboratory
  - CBC with differential and peripheral blood smear
  - Coagulation profile, fibrinogen and split products
  - Blood chemistry, including electrolytes, blood urea nitrogen (BUN), creatinine
  - Urinalysis for myoglobinuria
- Management
  - Support and transfer to definitive care
  - Bitten extremities should be marked proximal and distal to the bite, and the circumference at this location should be monitored every 15 min for progressive edema and compartment syndrome
  - Antivenom
    - Hemodynamic or respiratory instability
    - Abnormal coagulation studies
    - Neurotoxicity, e.g., paralysis of the diaphragm
    - Evidence of local toxicity with progressive soft tissue swelling
    - Antivenom is relatively specific for the snake species against which they are designed to protect
    - There is no benefit to administer antivenom to unrelated species due to risk of anaphylaxis and expense
  - Surgical assessment focuses on the injury site and concern for the development of compartment syndrome
  - Fasciotomy is indicated only for those patients with objective evidence of elevated compartment pressure

Black Widow Spider Bite

- Black spider with bright-red or orange abdomen
- Neurotoxin acts at the presynaptic membrane of the neuromuscular junction; decreased reuptake of acetylcholine and severe muscle cramping

Clinical presentation

- Pricking sensation that fades almost immediately
- Uncomfortable sensation in the bitten extremity and regional lymph node tenderness
- A “target” or “halo” lesion may appear at the bite site
- Proximal muscle cramping, including pain in the back, chest, or abdomen, depending on the site of the bite
- Dysautonomia that can include nausea, vomiting, malaise, sweating, hypertension, tachycardia, and a vague feeling of dysphoria

Management

- Analgesics should be administered in doses sufficient to relieve all pain
  - Oral or IV opioid analgesics
  - Benzodiazepines are adjunctive for cramping
- Hydration
- Management of severe hypertension

Brown Recluse Spider Bite

- Dark, violin-shaped mark on the thorax
- Venom causes significant local skin necrosis

Clinical presentation

- Typically, initially painless bite

- Rarely is the spider found or recovered
- Erythema, itching, and swelling begin one to several hours after the bite
- Central ischemic pallor to a blue/gray irregular macule to the development of a vesicle
- The central area may necrose, forming an eschar
- Induration of the surrounding tissue peaks at 48–96 h
- Lymphadenopathy may be present
- The entire lesion resolves slowly, often over weeks to months

Management

- Tetanus status should be assessed and updated
- Signs of cellulitis treated with an antibiotic that is active against skin flora
- Treatment is directed at the symptoms

Scorpion Stings

- The only scorpion species of medical importance in the United States is the Arizona bark scorpion (*Centruroides sculpturatus*)
- Toxins in its venom interfere with activation of sodium channels and enhance firing of axons

Clinical presentation

- Local pain is the most frequent symptom
- Small children may have more severe symptoms
- Peripheral muscle fasciculation, tongue fasciculation, facial twitching, and rapid disconjugate eye movements, which may be misdiagnosed as experiencing seizures
- Agitation
- Extreme tachycardia
- Salivation
- Respiratory distress

Management

- Supportive care
  - Airway support and ventilation in severe cases
  - Analgesia and sedation
- Antivenom therapy also may obviate or reduce the need for airway and ventilatory support

BURNS

Pediatric Burn Classification

- Superficial burn (formerly first degree)
  - Epidermal injury, intact dermis
  - Erythematous, dry, and painful
  - Minor injuries that heal within 1 week without scarring
- Partial thickness burn (formerly second degree)
  - Partial injury of the dermis, often with edema and blistering
  - Commonly are caused by scald injuries and result from brief exposure to the heat source
  - Blanchable pink or mottled red, often with blisters and moist appearance
  - Typically, painful
  - Healed within 1–3 weeks with minimal scarring
- Deep partial thickness burn (formerly second degree)
  - Injury to epidermis and dermis
  - Dry, pale appearance, non-blanchable, may have speckled appearance
  - Less painful than partial thickness, although some sensation preserved
  - Heals after many weeks, often with significant scarring requiring surgical subspecialty care for optimal cosmetic outcomes
- Full thickness burn (formerly third degree)
  - Most serious and deepest type of burn
  - Destruction of epidermis and entire dermis with necrosis
  - Pearly white, charred, hard, or parchment-like appearance
  - Destruction of cutaneous nerves makes the burn typically nonpainful

  - Loss of tissue elasticity makes the skin scar-like and unable to expand
    - Circumferential or near-circumferential burns can cause compartment syndrome, vascular compromise of distal extremity, respiratory distress if present on the chest
  - Burn cannot re-epithelialize and requires surgical subspecialty care and often skin grafting

Inhalation Injury

- A large percentage of burn-related deaths are due to associated smoke and inhalation injuries
- Evaluate all burn victims for potential for inhalational injury:
  - Signs of respiratory compromise: Coughing, stridor, wheezing, hoarseness
  - Signs of neurologic compromise: Irritability or lethargy
  - Facial burns
  - Black (carbonaceous) sputum
  - Burned nasal hair and eyebrows
- Early and aggressive airway management prior to onset of airway obstruction
  - Suspected inhalation injury with signs of airway compromise will need intubation and bronchoscopy

Electrical Burns

- Electrical current can cause significant internal damage via the arc of current through the body
- External visible injury may be minimal
- Depending upon the arc of the electrical current, multiple complications can arise
  - Cardiac arrhythmia (ventricular fibrillation) and myocardial damage
  - Rhabdomyolysis and renal failure
- Neurologic damage can develop in the years following an electrical burn
- Oral electrical burns affecting the commissure of the lips can be very scarring and at risk of bleeding from the labial artery

Description of Burn

- The percent body surface area is an important calculation in the classification of burns
- "Rule of Nines" can aid in calculation of percent body surface area
  - Imagine the body as a flattened shape, with front and back surface areas added separately (Fig. 7.1) [4]
- Palmar method
  - Surface of palm of hand can be used to approximate 1% body surface area in older children
  - Not useful in small children and infants

Management of Burns

Supportive home therapy for minor burns

- Superficial burns (< 10% total body surface area) can typically be treated on an outpatient basis with supportive care, unless abuse is suspected
  - Cotton gauze occlusive dressing to protect the damaged skin from bacterial contamination:
    - Eliminate air movement over the wound (thus reducing pain)
    - Decrease water loss
    - Change dressings daily
  - Topical antimicrobial agent should be applied to the wound prior to the dressing for prophylaxis
    - Silver sulfadiazine or bacitracin
  - Application of various wound membrane dressings can promote healing and lessen pain of dressing changes
  - Pain control

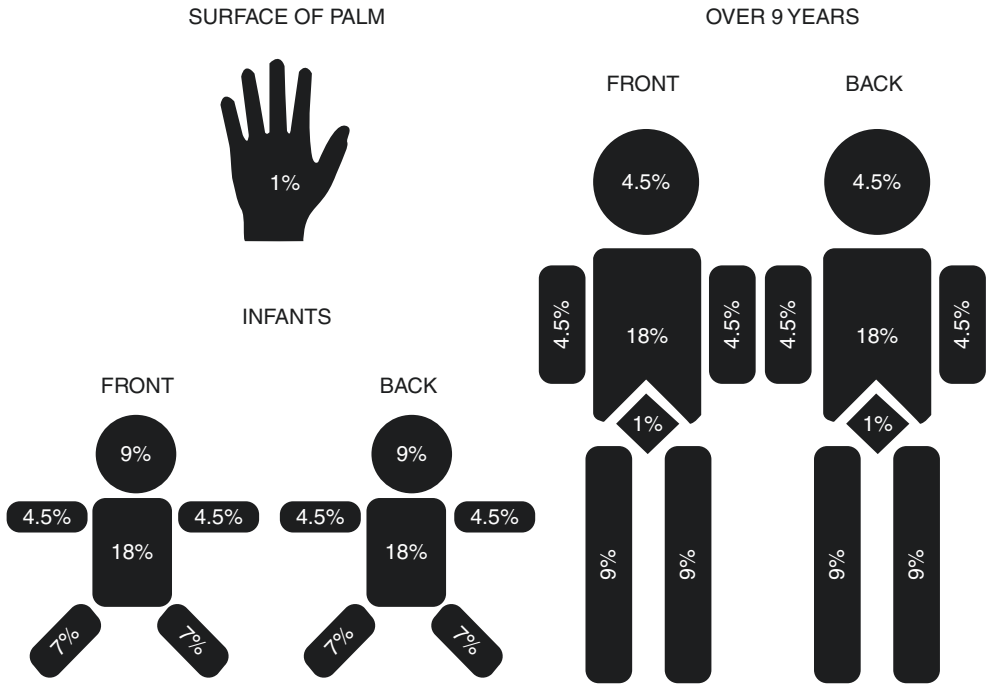

Fig. 7.1 Rule of nines: Infants versus children over 9 years old. (From Suguitan [4]), with permission)

- Alternating over-the-counter medications
- Opioid-containing medications for breakthrough pain
  - Caution in overreliance on opioids due to risk for dependence, withdrawal, and opioid-induced hyperalgesia

Initial treatment of extensive burns

- Extensive burns (> 10% total body surface area) and burns to high-risk areas (face, hands, neck, genitalia) will often require subspecialty care
- Identification of airway involvement due to risk for concomitant inhalation injury
  - Early and aggressive airway management recommended
- Fluid resuscitation to prevent shock
- Early excision and grafting of the burn wound coupled with early nutrition support
- Measures to treat sepsis
- Fluid administration
  - Once the nature and extent of injury are assessed, fluid resuscitation is begun
  - Two large-bore IV catheters
  - Parkland formula for fluid requirements:
    - 4 ml/kg/day for each percent of body surface area (BSA) burned
    - The first half of the fluid load is infused over the first 8 h post-burn
    - The remainder is infused over the ensuing 16 h
    - The infusion rates should be adjusted to maintain a urine flow of 1 ml/kg per hour
    - During the second 24 h, fluid administration is reduced 25–50%

STATUS EPILEPTICUS

Definitions

- A seizure that lasts more than 30 min ***or***
- Multiple seizures that occur without return of the individual to baseline, for a duration of not less than 30 min

Causes and risk factors

- Many conditions can cause status epilepticus
  - Infection (viral, bacterial, fungal, parasitic)
  - Trauma (intracranial hemorrhage, diffuse axonal injury, cerebral contusion)
  - Subtherapeutic anticonvulsant levels (patients with known epilepsy)

- Congenital abnormality
- Metabolic (hypoglycemia, hyponatremia, hypocalcemia, hypomagnesemia, hypercarbia, inborn errors of metabolism, pyridoxine deficiency in neonates)
- Vascular (hypoxic ischemic injury, cerebrovascular accident, hypertensive encephalopathy)
- Toxicologic (tricyclic antidepressants, isoniazid, pesticides (organophosphates), heavy metals (lead), topical anesthetic overdose)
- Endocrine (hyper-/hypothyroidism, Addison's disease)

Management

- Treatment should be based on an institutional protocol

General principles of management

- Attend to the **ABC**s before starting any pharmacologic intervention
  - **Airway**
    - Place patient in the lateral decubitus position to avoid aspiration of emesis and to prevent epiglottis closure over the glottis
    - Make further adjustments of the head and neck if necessary to improve airway patency
    - Suction secretions
    - Immobilize the cervical spine if trauma is suspected
  - **Breathing**
    - Administer 100% oxygen by face mask
    - Assist ventilation
    - Use artificial airways (e.g., endotracheal intubation) as needed
    - Decompress the stomach as needed with a nasogastric tube
  - **Circulation**
    - Carefully monitor vital signs, including BP
    - Carefully monitor the temperature, as hyperthermia may worsen brain damage
    - In the first 5 min of seizure activity, before starting any medications, try to establish IV access and obtain samples for laboratory tests
    - Infuse isotonic IV fluids plus glucose. In children younger than 6 years, use intraosseous (IO) infusion if IV access cannot be established within 5–10 min

Laboratory studies

- Finger-stick blood glucose
  - If serum glucose is low or cannot be measured, give children 2 ml/kg of D25% glucose
- Obtain basic metabolic panel, antiepileptic drug levels, and other labs, depending on the history and physical examination
- If the seizure fails to stop within 4–5 min, prompt administration of anticonvulsants may be indicated

Potential anticonvulsant medication options

- Anticonvulsant medication—selection can be based on seizure duration as follows:
  - Initial seizure activity (5–15 min):
    - Benzodiazepines are first-line GABA receptor blocker
      - Preferred options: Lorazepam IV or diazepam IV/rectal gel
      - Others: Midazolam IM or intranasal
  - Prolonged seizures (> 15 min), if refractory to benzodiazepines:
    - Many options exist with no clear literature to support a particular therapy
    - Selection should be made in consultation with a pediatric neurologist
    - Some options:
      - Phenytoin or fosphenytoin IV
      - Levetiracetam IV
      - Valproic acid IV
  - Continued seizure activity despite second- and third-line agents
    - General anesthesia may be required
    - Options include inhalational agents, pentobarbital anesthesia, continuous benzodiazepines

        - Anesthesia is titrated to achieve electroencephalogram (EE with burst suppression or flat line
    - By this point, advanced airway must be established, if not already
  - Other specific treatments may be indicated if the clinical evaluation identifies precipitants of seizures
    - Pyridoxine—IV/IM for possible dependence/deficiency or isoniazid toxicity
      - Pyridoxine-dependent seizures are a rare but reversible cause of refractory seizures in neonates: Consider administration of pyridoxine for neonatal status epilepticus
    - Naloxone: IV preferably (if needed may administer IM or subcutaneous) for narcotic overdose
    - Antibiotics: If meningitis is strongly suspected, initiate treatment with antibiotics prior to CSF analysis or CNS imaging

Anticipatory guidance

- Patients with history of status epilepticus will need:
  - Rescue abortive benzodiazepine (e.g., rectal diazepam) for prolonged seizures for home administration prior to arrival of emergency medical services
  - Seizure first aid teaching
- Consideration of additional benzodiazepine to raise seizure threshold during times of illness

ALTERED MENTAL STATUS (AMS)

Definitions

- AMS is a state of abnormally activated or abnormally suppressed awareness/consciousness
- It is a symptom and not a diagnosis—caused by an underlying disease or by trauma
- Altered level of consciousness or cognition—varying degrees of alteration of awareness
- Coma—the most severe form of altered level of consciousness in which an individual is not aware of his or her surroundings and cannot be easily aroused. A state lacking consciousness (both wakefulness and awareness) that cannot be overcome by stimulation
- Lethargy—state of altered level of consciousness that resembles deep sleep, from which a person can be aroused but immediately returns to that state. Depressed consciousness that, with adequate stimulation, can be overcome
- Obtunded—state of altered level of consciousness in which a person has greatly decreased responses and/ or is slow to respond
- Delirium (agitation)—abnormally activated consciousness with decreased awareness of environment from fluctuating global cerebral dysfunction, with inability or decreased ability to focus, shift, or sustain attention
- Consciousness
  - State of being awake and aware
  - Result of complex interplay of system controls
    - Ascending reticular activating system (ARAS) in brainstem and pons regulate wakefulness
    - Connections from the ARAS project out to the cortex and regulate awareness
    - Function is dependent on many factors
      - Requires adequate perfusion; adequate perfusion pressure; energy substrate (oxygen, glucose, hydration); electrolyte and acid-base balance (glucose, carbon dioxide, blood pH); removal of toxins (waste products); body temperature; absence of neuronal excitation/irritation (seizures)

Etiology of AMS

- The differential diagnosis of AMS is extremely broad and spans all possible organ systems
- Many possible causes of AMS have the potential to be life-threatening

- Trauma
  - Head trauma (subdural hematoma, epidural hematoma, cerebral edema, severe concussions)
- Infection
  - Meningitis, encephalitis, sepsis, brain abscess, subdural empyema
  - Sepsis with hypotension
- Neoplasm
  - Primary brain neoplasms or secondary brain involvement, blockage of CSF
- Vascular disease
  - Cerebral hemorrhage vs. infarct (arteriovenous malformation, aneurysm, hemangioma, thrombotic stroke), hypertensive emergency
- Obstruction to CSF
  - Malfunctioning ventriculoperitoneal (CSF) shunt
  - Hydrocephalus
- Metabolic anomalies
  - Hypoglycemia, hyponatremia, hypocalcemia, hypo-/hypermagnesemia, hypophosphatemia, metabolic acidosis and metabolic alkalosis, Reye syndrome
- Toxic ingestions
  - Many kinds of ingestions can cause AMS (e.g., severe aspirin ingestion, carbon monoxide poisoning, salicylates, barbiturates, alcohol, antihistamines, narcotics, phenothiazines, GHB [gamma hydroxybutyrate])
- Advanced stages of medical illnesses
  - Liver failure, kidney failure, heart failure, respiratory failure
- Specific disease states
  - Dehydration
  - Hypoxemia or hypercarbia
  - Hypothermia or hyperthermia
  - Hemolytic uremic syndrome: CNS infarction in basal ganglia
  - Intussusception: Infants can present initially with lethargy
  - Seizures: Postictal state, subclinical status epilepticus
  - Psychiatric disorders (pseudoseizure, conversion disorder)

Table 7.8 Glasgow coma scale [5, 6]

Score	Infant	Child	Adult
Eye opening			
4	Spontaneous	Spontaneous	Spontaneous
3	Opens to speech	Opens to speech	Opens to sound
2	Opens to pain	Opens to pressure	Opens to pressure
1	None	None	None
Best verbal response			
5	Coos and babbles	Oriented, appropriate	Oriented, appropriate
4	Irritable or cries	Confused	Confused
3	Cries in response to pain	Words	Words
2	Moans in response to pain	Sounds	Sounds
1	None	None	None
Best motor response			
6	Moves spontaneously and purposefully	Obeys commands	Obeys commands
5	Withdraws to touch	Localizing	Localizing
4	Withdraws to pain	Normal flexion	Normal flexion
3	Abnormal flexion to pain	Abnormal flexion	Abnormal flexion
2	Abnormal extension to pain	Extension	Extension
1	None	None	None

Glasgow Coma Scale

- The Glasgow Coma Scale (GCS) score can be used to help convey the level of AMS (Table 7.8) [5, 6]
  - GCS is 15 for individuals with normal level of mentation
  - GCS < 15 indicates an altered mental state
    - The subcategory including the lowest number should be indicated, e.g., a GCS of 13 (−2 M) indicates −2 from the motor subcategory

Management

- Treatment of AMS requires identification and treatment of the underlying cause
- In all patients, attend to ABCs promptly
  - Vital signs
    - Blood pressure
      - Attend to hypotension or severe hypertension
      - Fluid resuscitation
    - Heart rate
      - Attend to severe bradycardia or tachycardia
    - Hypothermia or hyperthermia
      - Thermoregulate patient
    - Pulse oximetry
      - Administer supplemental oxygen
      - Assess risk for methemoglobinemia or carboxyhemoglobinemia
    - Inadequate respiratory effort
      - Assist ventilation
  - Point-of-care glucose
    - Correction of hypoglycemia
  - Point-of-care blood gas
  - Consider empiric naloxone if clinical concern for opioid exposure

Specific disease categories:

- Trauma
  - This may be related to single system trauma (e.g., involving injury to the head/brain alone) or multisystem trauma
  - Decreased GCS with head trauma is presumed to be increased ICP until proven to be otherwise
  - May not have a history of a traumatic event if nonaccidental trauma is involved or injury was unwitnessed
  - Initial steps
    - Attend to airway, breathing, and circulation
    - Maintain cervical-spine immobilization
    - Obtain emergent noncontrast head CT and prompt neurosurgical consultation
    - Neuroprotective measures: May need 3% saline or mannitol, elevation of head to 30°, midline positioning of head
    - Other ongoing resuscitation measures may also be indicated
- Infection
  - In the presence of fever with meningismus, presume CNS infection
  - Attend to airway, breathing, and circulation promptly
  - Attempt diagnostic lumbar puncture if patient is stable enough to tolerate it
  - Order broad-spectrum IV antibiotics or antifungal medications if suspected fungal process, and do not delay administration
    - Order additional broad-spectrum IV antivirals for febrile neonate with risk factors for HSV encephalitis
  - If focal neurologic deficit or seizures are present, obtain noncontrast head CT prior to lumbar puncture
- Neoplasm
  - Space-occupying mass lesions in the brain can predispose to rapid decompensation
  - Attention to airway, breathing, circulation, and neuroprotective maneuvers and interventions
  - Requires prompt consultation with appropriate subspecialties (neurosurgery, oncology)
- Metabolic abnormalities
  - May be determined from results of testing or if adequate history is obtained, can help aid diagnosis
  - Therapy is directed at the specific abnormal electrolyte abnormality
    - Examples: Administration of glucose in the form of dextrose for hypoglycemia, or calcium for hypocalcemia
- Toxic ingestions
  - Attention to airway, breathing, circulation, and neuroprotective maneuvers and interventions
  - For specific antidotes, see Table 7.10
- Suspected CSF shunt malfunction
  - Attention to airway, breathing, circulation, and neuroprotective maneuvers

- Emergent head CT and radiographs to confirm shunt continuity ("shunt series")
- Timely neurosurgical consultation

POISONING AND TOXIC EXPOSURE

Background

- Children less than 6 years have the greatest risk
- Adolescent exposure either intentional or occupational

Prevention of poisoning

- Child-resistant packaging
- Anticipatory guidance in well childcare
  - Best provided at 6 months well child visit prior to the onset of mobility
  - Poison-proofing child's environment
    - Labeling all hazardous material
    - Locking medicine cabinets and securing cleaning products
    - Securing all medications in purses and handbags

Evaluation of a potentially toxic exposure

- Call the poison control center, describe the toxin, read the label, and follow the instructions
- Determine amount of exposure, number of pills, number of the remaining pills, and/or amount of liquid remaining
- Determine time of exposure
- Evaluate for progression of symptoms (any pattern of toxidromes)
- Consider associated ingestions and underlying medical conditions

General measures for toxic exposures

- For external exposures, remove clothes and wash the skin with soap and water
- For ingestions, can use activated charcoal if within 60 min of ingestion
  - Absorbs substances and decreases bioavailability
  - It is ineffective in the following (mnemonic CHEMICaL)
    - Caustics
    - Hydrocarbons
    - Ethanol (alcohols)
    - Metals
    - Iron
    - Cyanide
    - Lithium
- Ipecac
  - No longer recommended
  - Induction of emesis is particularly contraindicated with ingestions of hydrocarbons and caustics
- Gastric lavage
  - Contraindicated in hydrocarbons, alcohols, and caustics
  - Not recommended in most ingestions
  - May be considered with careful consideration if life-threatening ingestion occurred within 30–60 min of seeking medical attention
- Whole bowel irrigation
  - Reduces drug absorption by decontaminating the entire GI tract via large amounts of an osmotically balanced polyethylene glycol-electrolyte solution (PEG-ES) to induce a liquid stool and empty the bowel
  - Conclusive evidence that it improves outcomes is lacking
  - May cause vomiting and abdominal distention and lead to risk of aspiration
  - Should not be performed routinely, but may be considered for
    - Potentially toxic ingestions of sustained-release or enteric-coated drugs
    - Drugs not adsorbed by activated charcoal (e.g., lithium, potassium, and iron)
    - Removal of illicit drugs in the body (e.g., "packers" or "stuffers")

SPECIFIC INGESTIONS (TABLES 7.9, 7.10, AND 7.11) [7, 8]

Acetaminophen

- Responsible for one-third of all pediatric emergency department visits for ingestions
- The single toxic acute dose is generally considered to be > 200 mg/kg in children, and more than 7.5–10 g in adults and can cause hepatic injury or liver failure
- Any child with history of acute ingestion of > 150 mg/kg of acetaminophen should be referred for assessment and measurement of acetaminophen level

Clinical presentation (4 phases)

- First 24 h
  - Asymptomatic or nonspecific signs
  - Nausea, vomiting, dehydration, diaphoresis, pallor
  - Elevation of liver enzymes

Table 7.9 Toxidromes

Group	Vital signs	Mental status	Pupils	Other
Anticholinergics	Increased P, T	Delirium	Mydriasis	"Dry as a bone, red as a beet" … [a]
Cholinergics	Varies	Normal to depressed	Varies	"Drowning in secretions" DUMBBELLS[a]
Opioids	Decrease BP, P, R, T	Depressed	Miosis	Hyporeflexia
Withdrawal from opioids	Increase BP, P	Normal to anxious	Mydriasis	Vomiting, diarrhea, rhinorrhea
Sympathomimetics	Increase BP, P, R, T	Agitated	Mydriasis	Tremor and seizures
Ethanol or sedatives/hypnotics	Decrease BP, P, R, T	Depressed, agitated	Varies	Hyporeflexia and ataxia
Withdrawal from ethanol or sedatives/hypnotics	Increase BP, P, R, T	Agitated, disoriented	Mydriasis	Tremor and seizures

Adapted from Hoffman et al. [5] with permission McGraw Hill Education
BP blood pressure, *P* Heart rate, *R* Respiratory rate, *T* temperature
[a]See appropriate section

Table 7.10 Common antidotes for poisoning

Poison	Antidote
Acetaminophen	N-acetylcysteine (Mucomyst)
Anticholinergics	Physostigmine
Benzodiazepines	Flumazenil
β-blockers	Glucagon; insulin/glucose
Calcium channel blockers	Insulin and calcium salts
Carbon monoxide	Oxygen
Cyanide	Hydroxocobalamin (B12), Nitrites
Digitalis	Digoxin-specific fragments antigen-binding (Fab) antibodies
Ethylene glycol and methanol	Fomepizole
Iron	Deferoxamine
Isoniazid (INH)	Pyridoxine
Lead and other heavy metals, e.g., mercury and arsenic	British anti-Lewisite (BAL) (dimercaprol)
Methemoglobinemia	Methylene blue
Opioids	Naloxone
Organophosphates	Atropine and pralidoxime
Salicylates	Sodium bicarbonate
Sulfonylureas	Octreotide
Tricyclic antidepressants	Sodium bicarbonate

Table 7.11 Differential of toxic related findings (with mnemonic devices)

Finding	Differential diagnosis	
Anion gap metabolic acidosis	**C**	Carbon monoxide, cyanide
(Anion gap = Na – (HCO3 + Cl)	**A**	Aminoglycosides
	T	Theophylline, toluene (glue sniffing)
	M	Methanol, metformin
	U	Uremia
	D	Diabetic ketoacidosis, starvation
	P	Paraldehyde, paracetamol/acetaminophen, propylene glycol
	I	Iron, isoniazid, ibuprofen, inborn errors of metabolism
	L	Lactic acidosis
	E	Ethylene glycol
	S	Salicylates/aspirin
Widened QRS	Bupivacaine, bupropion, carbamazepine, class I (quinidine, amiodarone, procainamide) antiarrhythmics, class Ic (flecainide, propafenone, moricizine) antiarrhythmics, cocaine, diphenhydramine, lamotrigine, tricyclic antidepressants	
Prolonged QTc	**T**	Thiazides
	O	Ondansetron (antiemetics), opioids (methadone)
	Q	Quinidine (class Ia), quinolones (antibiotics)
	R	Risperidone (antipsychotics)
	S	Sotalol (class III)
	A	Antihistamines
	D	antiDepressants (TCA)
	E	Erythromycin and other macrolides
	S	SSRI (fluoxetine, sertraline)
Hypoglycemia	**H**	Hypoglycemic (sulfonylureas not metformin)
	O	Others (quinine, quinolones, pentamidine)
	BB	β Blockers, Bactrim
	I	Insulin
	E	Ethanol
	S	Salicylates
Bradycardia and hypotension	**C**	Calcium channel blockers (diltiazem, verapamil, amlodipine)
	O	Opiates
	P	Propranolol and other beta-blockers (metoprolol, esmolol)
	A	Anticholinergics (organophosphates, carbamates, neostigmine, sarin, VX)
	C	Clonidine and other central α-2 receptor agonist (guanfacine, dexmedetomidine, oxymetazoline)
	E	Ethanol
	D	Digoxin and cardiac glycosides (oleander, foxglove, lily of the valley)

- 24 to 72 h post-ingestion
  - Tachycardia and hypotension
  - Right upper quadrant pain with or without hepatomegaly
  - Liver enzyme is more elevated
  - Elevated prothrombin time (PT) and bilirubin in severe cases
- 3 to 4 days post-ingestion
  - Liver failure
  - Encephalopathy, with or without renal failure
  - Possible death from multiorgan failure or cerebral edema
- 4 to 14 days post-ingestion
  - Complete recovery or death

Management

- Measure serum acetaminophen level 4 h after the reported time of ingestion
- Acetaminophen level obtained < 4 h after ingestion cannot be used to estimate potential toxicity
- Check acetaminophen level 6–8 h if it is co-ingested with other substance that slows GI motility, e.g., diphenhydramine
- Check liver function AST/ALT/coagulation parameters, renal function
- Start N-acetylcysteine (NAC)
  - If acetaminophen level is above the treatment line on Rumack-Matthew nomogram (4 h and after) (Fig.7.2) [6]
  - If acetaminophen level is low or undetectable with abnormal liver function
  - Patients with a history of potentially toxic ingestion more than 8 h after ingestion (single dose > 200 mg/kg in children and more than 7.5–10 g in adults)
- Liver transplant for liver failure

Ibuprofen

- Inhibit prostaglandin synthesis

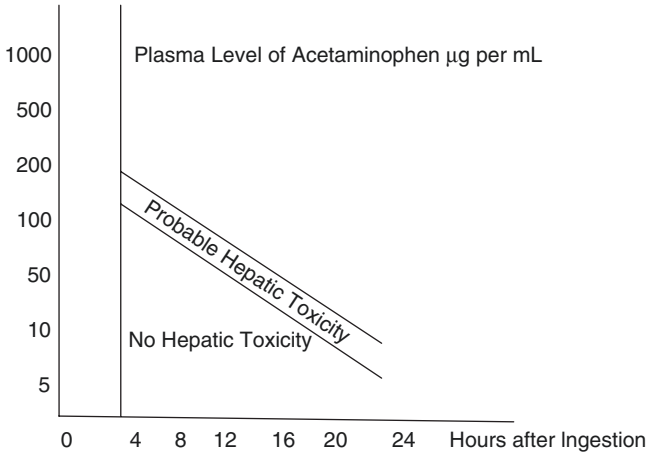

Fig. 7.2 Rumack-Matthew nomogram for acetaminophen poisoning [6]

Clinical presentation

- Nausea, vomiting, and epigastric pain
- Drowsiness, lethargy, and ataxia may occur
- Anion gap metabolic acidosis, renal failure, seizure, and coma may occur in severe cases (usually > 400 mg/kg)
- May cause GI irritation, ulcers, decrease renal blood flow, and platelet dysfunction

Management

- Activated charcoal
- Supportive care

Salicylic Acid

- Aspirin, certain antidiarrheal medications, topical agents, e.g., keratolytics and sports creams
- Refer to an emergency department for ingestions > 150 mg/kg
- Ingestion of > 200 mg/kg is generally considered toxic; > 300 mg/kg is more significant toxicity; > 500 mg/kg is potentially fatal

Clinical presentation

- Acute salicylism
  - Nausea, vomiting, diaphoresis, and tinnitus

- Moderate toxicity
  - Tachypnea, hyperpnea, tachycardia, and AMS
- Severe toxicity
  - Hyperthermia and coma

Management

- Consider activated charcoal
- Check blood gas (respiratory alkalosis, metabolic acidosis, and high anion gap)
- Check serum level every 2 h until it is consistently down trending
- IV fluids
- Sodium bicarbonate therapy in the symptomatic patients
- Goal of therapy includes a urine pH of 7.5–8.0, a serum pH of 7.5–7.55, and decreasing salicylate levels

Anticholinergics

- Diphenhydramine, atropine, scopolamine, hyoscyamine, jimsonweed (*Datura stramonium),* and deadly nightshade (*Atropa belladonna*)

Clinical presentation (anticholinergic symptoms)

- Dry as a bone: Dry mouth, decreased sweating, and urination
- Red as a beet: Flushing
- Blind as a bat: Mydriasis, blurred vision
- Mad as a hatter: Agitation, seizures, hallucinations
- Hot as a hare: Hyperthermia
- Bloated as a toad: Ileus, urinary retention
- Heart runs alone: Tachycardia

Management

- Consider activated charcoal
- Supportive care
- Physostigmine can be considered for severe or persistent symptoms

Beta-Blockers

- Acebutolol, atenolol, bisoprolol, metoprolol, nadolol, sotalol, and propranolol

Clinical presentation

- Hypotension, bradycardia, atrioventricular (AV) block, heart failure
- Bronchospasm
- Hypoglycemia, hyperkalemia
- Stupor, coma, seizures

Management tailored to the symptoms

- Consider dose of activated charcoal
- Hypotension/bradycardia/AV block: Fluid boluses, beta-agonists, vasopressors, atropine, possibly pacing
- Hypoglycemia: Glucose
- Hyperkalemia: Calcium gluconate, dextrose and insulin, sodium bicarbonate, possibly dialysis
- Two special cases
  - Propranolol → causes sodium channel blockade → QRS widening → treat with sodium bicarbonate
  - Sotalol → causes potassium efflux blockade → prolonged QT → monitor for *torsades*

Carbamazepine

Clinical presentation

- Mild ingestion:
  - CNS depression
  - Drowsiness
  - Vomiting
  - Ataxia
  - Slurred speech
  - Nystagmus
- Severe intoxication:
  - Seizures

  - Coma
  - Respiratory depression

Management
- Activated charcoal
- Supportive measures
- Charcoal hemoperfusion for severe intoxication

Cardiac Glycosides (Digitalis)

- Digoxin, foxglove plants, oleander, lily of the valley (*Convallaria*)

Clinical presentation
- Nausea and vomiting
- CNS depression (confusion)
- Blurry vision
- Cardiac conduction abnormalities (irregular pulse, bradycardia or tachycardia)

Management
- Electrocardiogram (ECG) and digoxin levels
- Activated charcoal
- Atropine, cardiac pacing (for severe bradycardia)
- Magnesium sulfate (for premature ventricular contractions [PVCs])
- Management of hyperkalemia, hypokalemia, hypomagnesemia
- If severe, digoxin-specific antibody fragments

Clonidine

- Antihypertensive medication with α-2 adrenergic receptor blocking ability
- Commonly used in children with attention-deficit hyperactivity disorder (ADHD)
- Dose as small as 0.1 mg can cause toxicity in children

Clinical presentation
- Lethargy
- Miosis
- Bradycardia
- Hypotension
  - Can cause transient initial hypertension
- Apnea

Management
- Supportive care, e.g., intubation, atropine, dopamine as needed
- Charcoal usually not recommended due to CNS depression

Opiates

- Morphine, heroin, methadone, propoxyphene, codeine, meperidine
- Most cases are drug abuse

Clinical presentation
- Common triad of opiate poisoning:
  - Pinpoint pupil
  - Mental depression (lethargy to coma)
  - Respiratory depression
- Hypotension with no change in heart rate
- Decreased GI motility, nausea, vomiting, abdominal pain

Management
- Supportive care, e.g., airway, breathing, and circulation; intubation; fluids as necessary
- Naloxone as needed
  - Half-life of naloxone is short
  - Repeat doses and continuous infusions may be necessary
  - Cautious use in known opioid-dependent patients
    - Can induce withdrawal

Phenothiazines

- Promethazine, prochlorperazine, and chlorpromazine

Clinical presentation
- Hypertension

- Cogwheel rigidity
- Dystonic reaction (spasm of the neck, tongue thrusting, oculogyric crisis)
- CNS depression

Management
- Activated charcoal
- Manage high BP
- Diphenhydramine for dystonic reaction

Tricyclic Antidepressants (TCAs)

- TCAs can cause significant toxicity in children even with ingestion of 1–2 pills (10–20 mg/kg)

Clinical presentation
- Anticholinergic toxidrome: Delirium, mydriasis, dry mucous membrane, tachycardia, hyperthermia, hypotension, and urinary retention
- Cardiovascular and CNS symptoms dominate the clinical presentation
- Most common cardiac manifestations: Widening of QRS complex, PVCs, ventricular arrhythmia
- Refractory hypotension is poor prognostic indicator and is the most common cause of death in TCA toxicity

Management
- Supportive measures, ABCs
- ECG:
  - A QRS duration > 100 ms identifies patients at risk for seizures and cardiac arrhythmia
  - An R wave in lead aVR of > 3 mm is an independent predictor of toxicity
- Start sodium bicarbonate therapy: QRS duration > 100 ms, ventricular dysrhythmias, hypotension, and seizures

Carbon Monoxide (CO)

- Wood-burning stove, old furnaces, and automobiles

Prevention of CO poisoning
- CO detectors
- Maintenance of fuel-burning appliances
- Yearly inspection of furnaces, gas pipes, and chimneys
- Car inspection for exhaust system
- No running engine in a closed garage
- Avoid indoor use of charcoal and fire sources

Clinical presentation
- Headache, malaise, nausea, and vomiting are the most common flu- or food poisoning-like early symptoms
- Confusion, ataxia, syncope, tachycardia, and tachypnea at higher exposure
- Coma, seizure, myocardial ischemia, acidosis, cardiovascular collapse, and potentially death in severe cases

Management
- Evaluate for COHb level in symptomatic patients
- Arterial blood gas with CO level
- Check creatine kinase in severe cases
- ECG in any patient with cardiac symptoms
- 100% oxygen to enhance elimination of CO, use until CO < 10% and symptoms resolve
- Severely poisoned patient may benefit from hyperbaric oxygen especially if COHb > 25%, significant CNS symptoms, or cardiac dysfunction

Cyanide

- Seeds (cherries, apricots, peaches, apples, plums); cassava; burning plastics (nitrile-containing products); nitroprusside; some pesticides
- Amygdalin is contained in seeds and produces hydrogen cyanide, which is a potent toxin
- Inhibition of cellular respiration (cytochrome c oxidase) stops ATP production

Clinical presentation
- Decreased level of consciousness

- Low exposures—weakness, headache, dizziness, confusion
- Severe exposures—seizure, apnea, cardiac arrest
- Cherry red skin

Management
- Supportive measures
- Hydroxocobalamin (vitamin B12), nitrites

Ethylene Glycol Ingestion (Antifreeze)

Clinical presentation
- Nausea, vomiting, CNS depression, anion gap metabolic acidosis
- Hypocalcemia, renal failure due to deposition of calcium oxalate crystals in the renal tubules

Management
- Osmolar gap can be used to estimate ethylene glycol level
- IV fluids, glucose, and bicarbonate as needed for electrolyte imbalances and dehydration
- Fomepizole
- Ethanol can be used if fomepizole is unavailable

Methanol

- Toxicity primarily caused by formic acid

Clinical presentation
- Drowsiness, nausea, and vomiting
- Metabolic acidosis
- Visual disturbances: blurred and cloudy vision, feeling of being in a snowstorm, and untreated cases can lead to blindness

Management
- Osmolar gap (may be used as surrogate marker until methanol blood level is available)
- IV fluids, glucose and bicarbonate as needed for electrolyte imbalances and dehydration
- Fomepizole
- Ethanol can be used if fomepizole is unavailable
- Hemodialysis (consider if > 30 ml methanol ingested)

Iron

- Ingestion of > 60 mg/kg/dose is toxic

Clinical presentation
- Gastrointestinal stage (30 min–6 h)
  - Nausea, vomiting, and abdominal pain
  - Hematemesis and bloody diarrhea in severe cases
- Stability stage (6–24 h)
  - No symptoms: Patient must be observed during this stage
- Systemic toxicity within (48 h)
  - Cardiovascular collapse
  - Severe metabolic acidosis
- Hepatotoxicity and liver failure (2–3 days)
- Gastrointestinal and pyloric scarring (2–6 weeks)

Management
- Abdominal radiograph (may show pills and need for GI decontamination)
- Serum iron < 300 mcg/dl is nontoxic
- Chelation with IV deferoxamine if serum iron > 500 mcg/dl

Mushrooms

- Ingestion of mushrooms can have fatal consequences in species that harbor amatoxins (e.g., *Amanita*) and related compounds

Clinical presentation
- Nausea, vomiting, and diarrhea; delayed onset (6 h)
- A second latent period is followed by acute and possibly fulminant hepatitis beginning 48–72 h after ingestion

Management

- Activated charcoal
- Whole bowel irrigation
- Supportive care, including liver transplant, if necessary, is the mainstay of therapy

Caustic Ingestion

- Strong acid and alkalis < 2 or > 12 pH can produce severe injury even in small-volume ingestion
- Patient can have significant esophageal injury without visible oral burns

Clinical presentation

- Pain, drooling, vomiting, and abdominal pain
- Difficulty in swallowing, or refusal to swallow
- Stridor and respiratory distress are common presenting symptoms
- Esophageal stricture caused by circumferential burn; requires repeated dilation or surgical correction

Management

- Supportive measures, ABCs
  - Inducing emesis and lavage are contraindicated
  - Endoscopy should be performed within 12–24 h in symptomatic patients, or on basis of history and characteristics of ingested products

Hydrocarbons

- Products contain hydrocarbon substances, mineral spirits, kerosene, gasoline, turpentine, and others
- Aspiration of even small amount can be serious and potentially life-threatening
- Pneumonitis is the most important manifestation of hydrocarbon toxicity
- Benzene is known to cause cancer
- Inhalants, including toluene, propellants, and volatile nitrite, can cause dysrhythmias and sudden death

Clinical presentation

- Cough, tachypnea, respiratory distress

Management

- Emesis and lavage are contraindicated
- Activated charcoal should be avoided due to risk of inducing vomiting
- Observation and supportive care

Organophosphate and Carbamate Insecticides (Nerve Gas Agents)

- Inhibit anticholinesterase

Clinical presentation

- (DUMBBELLS) "Drowning in your own secretions"
  - Diarrhea
  - Urination
  - Miosis
  - Bradycardia
  - Bronchospasm
  - Emesis
  - Lacrimation
  - Lethargy
  - Salivation and Seizures

Management

- Wash all exposed skin with soap and water and immediately remove all exposed clothing
- Fluid and electrolyte replacement, intubation, and ventilation if necessary
- Atropine and pralidoxime

FOREIGN BODY ASPIRATION AND INGESTION

Foreign Body Aspiration

- Occurs in the context of child's play/exploration of environment
- Foreign bodies may lodge in the upper or lower respiratory tract

Upper airway

- The most commonly implicated foods are
  - Candy, meat, hot dogs, grapes
- Associated symptoms include
  - Choking, coughing, stridor, respiratory distress
  - May result in complete airway obstruction
- In patients with complete airway obstruction, emergency procedures may be life-saving
  - Back blows in infants
  - Heimlich maneuvers in older children
  - If patient becomes unconscious, may need to initiate cardiopulmonary resuscitation (CPR)
    - Convert to rescue breaths and chest compressions
  - If all of the above are unsuccessful, then advanced airway techniques may be necessary

Lower airway

- More common in younger children
- Foreign body may lodge in the right or left lung
- Symptoms may involve coughing, choking, wheezing, or may be asymptomatic
- Diagnosis may be delayed due to lack of symptoms

Management

- If the patient is stable and not in respiratory distress, neck and/or chest radiographs may help with diagnosis
  - Not all inhaled foreign bodies are radiopaque and may not be visualized on radiographs even if present
- Bilateral decubitus views may aid in diagnosis: Bilateral to compare which lung exhibits air trapping, which may be where the foreign body is lodged
- The presence of normal chest radiographs does not exclude this diagnosis in the presence of a compelling history
- For patients in severe respiratory distress, immediate bronchoscopy to remove the foreign body emergently is key to treatment

Foreign Body Ingestion

- Most children will have a history of ingested foreign body, often reported by a caregiver or playmate/sibling
- Most commonly ingested foreign bodies are
  - Food (meat)
  - Followed by coins, pins, toy parts, button batteries, magnets
- Most will pass harmlessly through the GI tract
- Foreign bodies may lodge in areas where there is physiological/anatomic narrowing of the lumen of the GI tract
  - Lodging may occur secondary to pathological narrowing of the lumen of the GI tract (e.g., from previous surgeries such as in tracheoesophageal fistula, esophageal/duodenal webs)
- Symptoms, if present, may include coughing, choking, foreign body sensation, throat pain, drooling, vomiting, refusal or inability to tolerate fluids/food
- Children may also be completely asymptomatic

Esophagus

- The esophagus is the most common site for ingested foreign bodies to become lodged
- Ingested foreign bodies typically become lodged in one of three sites:
  - At the thoracic inlet

  - Mid-esophagus, at the level of the carina and aortic arch
  - Esophago-gastric junction

Management

- If the patient is asymptomatic and there is no airway compromise, may do neck and/or chest radiographs
- If foreign body is not visualized and patient is asymptomatic, then can do an esophagram
- If foreign body is lodged in the esophagus, may need removal by esophagoscopy
- Some authorities report the use of glucagon with varying degrees of success
- Emergent removal is indicated for two or more magnets, button batteries, lodged sharp objects

Stomach and Lower Gastrointestinal Tract

- Most foreign bodies in the stomach will pass harmlessly through the remaining portion of the GI tract
- Single, not sharp foreign bodies may be managed conservatively and observed
- Parents may be advised to watch patient stools, or have repeat abdominal radiographs within 1 week to assess if foreign body has been eliminated
- If two or more button batteries or magnets are located in any portion/part of the GI tract, consultation with a gastroenterologist to facilitate removal is necessary
  - If not removed, the magnets may "adhere" together across tissues/tissue planes and cause necrosis with ensuing perforation
- Sharp objects (particularly if longer than 5 cm) in the stomach and lower GI tract require consultation with a gastroenterologist

DIABETIC KETOACIDOSIS (DKA)

DKA in Pediatric Patients

- DKA is a severe complication of type 1 diabetes
- Occurs in 25–40% of new-onset type 1 diabetes
- Inadequate relative or absolute deficit of insulin leads to starvation of insulin-dependent tissue (muscle, liver, fat) with resultant hyperglycemia
- Starvation state triggers a cascade of hormonal release such as glucagon, catecholamines, cortisol, cytokines
- Results in a catabolic state with lipolysis, proteolysis, adipose tissue metabolism into free fatty acids, hepatic conversion of fatty acid to keto-acids, and anion gap metabolic acidosis
- Hyperglycemia causes osmotic diuresis with hypovolemia and dehydration

Diagnostic laboratory findings

- Acidosis (venous pH < 7.3, serum bicarbonate < 15 mEq/L)
- Serum glucose > 200 mg/dL
- Glucosuria, ketonemia, and ketonuria

Degrees of severity of DKA

- Mild: pH > 7.2 and < 7.3, bicarbonate < 15 mEq/L
- Moderate: pH > 7.1 and < 7.2, bicarbonate < 10 mEq/L
- Severe: pH < 7.1, bicarbonate < 5 mEq/L

Review of pathophysiology

- Inadequate insulin secretion
  - Decrease in cell uptake of glucose, leading to hyperglycemia
  - Hyperglycemia causes osmotic diuresis, leading to dehydration

  - Compounded by stress response with activation of gluconeogenesis, glycogenolysis, and increased insulin resistance
- Protein catabolism
  - Adipose tissue broken down into fatty acids
  - Fatty acids converted to keto-acids in the liver
- Dehydration
  - Osmotic diuresis from hyperglycemia
  - Compounded by acidosis with nausea, vomiting, and oral intolerance
  - Typically occurs in setting of several weeks of polyuria, polydipsia, and weight loss
  - Dehydration can be profound and lead to shock state
- Acidosis
  - Keto-acids from protein catabolism cause anion gap acidosis
  - Poor tissue perfusion in setting of severe dehydration causes lactic acidosis
- Electrolyte abnormalities
  - Potassium
    - Acidosis causes extracellular shift of potassium with hyperkalemia
    - Excess serum potassium is cleared by kidney
    - Hypovolemia stimulates secondary hyperaldosteronism and further urinary potassium excretion
    - Factors leads to total body potassium depletion in all patients with DKA
    - Measured serum potassium levels are highly variable at presentation (hypokalemia, normal, or hyperkalemia) and do not correlate with degree of total body potassium losses
  - Phosphate
    - Acidosis stimulates phosphate depletion due to renal phosphate excretion
  - Sodium
    - Hyperglycemia and osmotic diuresis lead to renal sodium losses and typically hyponatremia

Cerebral Edema

- Most serious immediate risk to child in DKA with mortality rate of 40–90%
- Occurs in 1/100 pediatric cases during the first 24 h of DKA
- Causes 50–60% of diabetes-related pediatric deaths
- Pathophysiology of cerebral edema is controversial
- Risk factors for cerebral edema continue to be studied
  - New-onset diabetes
  - Age < 3 years
  - High BUN at presentation
  - Low PCO2
  - Treatment with bicarbonate
  - Failure of serum sodium to correct with therapy

Signs

- AMS, from agitation to frank coma
- Severe headache
- Heart rate deceleration
- Focal neurologic deficit

Treatment

- Immediate recognition
- Immediate mannitol 1 g/kg over 10–20 min
- Cerebral edema is a reversible condition with prompt treatment

Principles of initial resuscitation of DKA in the emergency department

- Treatment of DKA is often based on institutional protocol
- General concepts are
  - Initial rehydration with isotonic fluid
    - 10–20 ml/kg of 0.9% normal saline over 1–2 h
    - Goals: adequate tissue perfusion, not normovolemia
  - Administration of insulin as continuous IV infusion
    - Regular insulin 0.1 unit/kg/h via IV line

  - Addition of dextrose in fluid after serum glucose begins to fall
    - Varying concentrations of dextrose from 5% up to 12.5% via IV line
    - Plasma glucose target range 200–300 mg/dL
  - Careful correction of electrolyte disturbances
    - Addition of potassium once serum K < 5.0 mEq/L
    - Addition of phosphate as potassium phosphate as serum phosphate allows
  - Prompt subspecialty consultation
  - Frequent laboratory monitoring
  - Frequent neurologic assessments to monitor for cerebral edema

Life-Threatening Complications to Consider in the Emergency Department

- Cerebral edema
- Shock/cardiovascular collapse
  - Dehydration can be profound
  - Requires prompt restoration of intravascular volume
  - Consider infection/sepsis as trigger for stress response and hyperglycemia if clinically warranted
- Hyperkalemia or hypokalemia
  - Obtain ECG in critically ill child
  - May cause life-threatening arrhythmias or cardiovascular collapse
- Profound metabolic acidosis
  - Insulin infusion is necessary to stop primary ketoacidosis
  - Insulin infusion should never be stopped in DKA: If hypoglycemia occurs, adjust dextrose in fluids or rate of fluids to maintain plasma glucose levels
- Hypophosphatemia
  - Caution with replacement of phosphate is advised due to risk of precipitating hypocalcemia, renal failure, and arrhythmias

Ongoing management

- Children with DKA typically require subspecialty monitoring
- Admission frequently required to specialized unit or to the intensive care unit
- Continued monitoring
  - Hourly plasma glucose
  - Hourly neurologic assessments
  - Hourly intake and output
  - Serial measurements of serum potassium, calcium, phosphate, magnesium
  - Serial venous blood gas

Goals of treatment

- These goals are typically achieved in the inpatient setting
  - Resolution of hyperglycemia
  - Correction of dehydration
  - Closure of anion gap acidosis (anion gap normalized between 10 and 12)
  - Oral tolerance to feeds
  - Resolution of other symptoms

CONCUSSION/HEAD INJURY

Definition

- Concussion can be defined as
  - An acute injury to the brain from an external physical force
  - Resultant confusion, disorientation, brief loss of consciousness, self-limited post-traumatic amnesia and/or other transient neurologic abnormalities
  - GCS score of 13–15 after 30 min following the injury
- Also known as a mild traumatic brain injury

Epidemiology and mechanism of pediatric concussions

- Major cause across all age groups for pediatric visits to emergency care
- Arises from either a direct or transmitted blow to the head, face, or neck
- Subsequent injury to the brain is the result of an interplay of pathophysiologic processes, induced by biomechanical forces, without evidence of structural brain injury on standard neuroimaging

Risk factors for a concussion

- An individual's risk for a concussion is multifactorial with no single element being absolutely predictive
  - Boys reportedly affected more than girls due to predilection for more injuries/inclusion in more sports-related activities
  - Girls more likely to report symptoms of concussion
  - History of ADHD or learning problems raises lifetime risk
  - History of prior concussion raises risk for future concussion

Risk factors for prolonged concussion symptoms

- An individual's risk for prolonged symptoms is multifactorial with no single element being absolutely predictive
  - Prior history of concussion-like symptoms
  - Known history of intracranial abnormalities
  - Psychiatric disorders, headache disorders
  - Family and social stressors
  - Female sex predictive of symptoms lasting more than 4 weeks
  - Older age

Signs and symptoms

- The signs and symptoms of pediatric concussions are variable
  - Cognitive
    - Amnesia—may be retrograde or anterograde
    - Confusion
    - Difficulty concentrating
    - Disorientation
    - Persistent crying
  - Neurologic
    - Headache
    - Dizziness
    - Gait abnormalities
    - Sensitivity to light/noise
    - Slurred speech
    - Fatigue
  - Behavioral
    - Increased sleep
    - Emotional lability
  - Gastrointestinal
    - Nausea
    - Vomiting
    - Refusal to feed

Management

- Diagnosis is typically clinical
  - Neuroimaging is not routinely indicated for a diagnosis of a concussion
    - For further considerations of head imaging in association with pediatric head trauma, see Trauma and Burns and Table 7.5 [1]
- Conservative management is the mainstay of treatment for pediatric concussions
  - Antiemetics may be used for nausea and/or vomiting
  - Pain control with nonopioid medications (e.g., ibuprofen and acetaminophen)
  - Stepwise approach to cognitive and physical rest (see Chap. 14 "Sports Medicine")
    - Complete bed rest may be required for a variable length of time (typically no more than 2–3 days)
    - Additional avoidance of activities that have high cognitive load is based upon the patient's individual risk factors and developmental stage
    - Gradual return to daily activities and increasing cognitive and noncontact physical activity in a manner that does not exacerbate symptoms

- Return to full activities only if patient is asymptomatic and has passed prior stages of recovery
- Anticipatory guidance is important to manage expectations and promote recovery
  - Inadequately treated concussions can prolong symptomatology and place patient at risk for reinjury
  - Warnings signs and symptoms to watch for that could indicate more severe injury
  - Recovery time is unique to each patient and for each incidence of head trauma
  - Concussions can cause physical, cognitive, and psychological impairments

DROWNING

Definition

- Drowning is defined as
  - The process of experiencing respiratory impairment from submersion in a liquid
  - Avoid using confusing older terms: Near drowning, secondary drowning, and wet drowning
- Classification of drowning
  - Fatal
  - Nonfatal with no morbidity
  - Nonfatal with morbidity (moderate, severe, vegetative state, brain death)

Epidemiology

- Drowning is a major cause of injuries and death
  - Seasonal variation, with increasing incidence in summer months
- Incidence follows a bimodal distribution
  - First peak occurs in children < 5 years of age
    - Children < 1 year
      - Often drown in bathtubs, buckets, and toilets
      - Over half of infants in bathtubs
    - Children 1–4 years of age
      - Over half of young children drown in swimming pools where they have been unsupervised temporarily (usually for < 5 min)
      - Typical incidents involve a toddler left unattended temporarily or under the supervision of an older sibling
  - Second peak: Occurs at ages 15–24 years
    - Primarily male adolescents and young adults
    - Most incidents occur in natural water

Mechanism of injury

- Initial progression of injury
  - Swallowing of water
    - Laryngospasm
    - Loss of consciousness due to hypoxemia
    - Hypoxia
    - Loss of circulation
    - Tissue ischemia
    - CNS injury (the most common cause of death)
- Secondary progression of injury
  - After nonfatal drowning, further complications can develop:
  - Pulmonary
    - Aspiration pneumonia
    - Acute respiratory distress syndrome (ARDS)
  - Cardiac
    - Myocardial depression, arrhythmias
  - Neurologic
    - Cerebral edema, increased ICP
  - Metabolic
    - Metabolic and respiratory acidosis
  - Hematologic
    - Hemolysis, coagulopathy
  - Renal
    - Acute tubular necrosis

Management

- At the scene
  - Immediate cardiopulmonary resuscitation (CPR) once a submersion has occurred is the most important initial step
- Prompt attention to airway, breathing, and circulation

- Early intubation if
  - Signs of neurologic deterioration
  - Inability to protect airway
  - Inability to maintain adequate oxygenation or ventilation despite supplemental oxygen
    - Remember orogastric tube for gastric decompression if intubated
- Administer 100% oxygen immediately to maintain SpO2 > 94%
  - Prevent further hypoxemia and acidosis
- Support work of breathing
  - If patient does not require intubation but has signs of impaired gas exchange, can elect to use noninvasive positive pressure ventilation such as continuous positive airway pressure (CPAP).
  - If advanced airway is established, mechanical ventilation with positive end-expiratory pressure (PEEP)
- Judicious fluid and shock resuscitation
  - Especially important with high levels of positive airway pressure required for adequate ventilation with poor lung compliance, with resultant increased intrathoracic pressure and decreased venous return to the heart
  - Vasopressors as indicated
  - After hemodynamic stability achieved, severe drowning may require fluid restriction and diuretic therapy due to pulmonary edema
- Cervical spine immobilization if suspected head/neck trauma is present
  - Classic example: Dive into shallow water
- Serial measurements
  - Cardiopulmonary monitoring, temperature, and neurologic assessments
    - Pediatric drowning victims typically are hypothermic even if the water is warm
    - Abrupt change in mental status can reflect worsening lung function and hypoxemia
- Initial laboratory studies
  - Point-of-care glucose, complete blood cell count, electrolytes, urinalysis
- Initial imaging
  - Chest radiograph: indicated for all patients
  - Head CT: may be indicated for patients presenting with AMS

Disposition

- If CPR is required at the scene, recommend admission regardless of clinical status upon presentation
- If patients with mild symptoms on arrival, recommend admission for monitoring
- If patient is asymptomatic with the following criteria, the patient may be monitored for 6–8 h prior to discharge home with close follow-up instructions
  - GCS 15
  - Normal chest radiograph
  - Normal lung exam
  - Normal oxygen saturations
  - Safe home

Prevention and guidance

- Prevention is the key intervention in pediatric drowning
  - Installation of 4-sided fencing
    - Completely prevents direct access to the pool from the house and yard
    - At least 4 ft high (or higher if required by local ordinance) and climb-resistant
    - Distance from bottom to ground less than 4 in.
    - Gate should be self-latching and self-closing, with the latch placed at least 54 in. above the bottom of the gate, open away from the pool, and should be checked often
    - Effective in preventing more than 50% of swimming-pool drownings of young children
- Supervision needs to be close, constant, and capable
  - Never—even for a moment—leave small children alone or in the care of another young child while in bathtubs, pools, spas, or wading pools or near irrigation ditches or other open standing water

  - A supervising adult with swimming skills should be in the water, within an arm's length, providing "touch supervision"
  - With older children and better swimmers, the eyes and attention of the supervising adult should be constantly focused on the child
  - The adult should not be engaged in other distracting activities that can compromise this attention, such as talking on the telephone, socializing, tending chores, or drinking alcohol
  - Children should never swim alone without an adult

Swim lessons

- Swim lessons for children > 4 years: Recommended
- Swim lessons for children 1–4 years: Insufficient data to recommend at this time

Equipment

- Personal flotation devices (PFDs or life jackets) are recommended
- Air-filled swimming aids (inflatable arm bands, floaties) are not recommended
- Do not use air-filled swimming aids in place of PFDs

HYPERTENSIVE CRISIS

With climbing rates of obesity among children, hypertension is now increasingly common among children. This section will discuss an approach to children presenting with hypertension and make distinctions between hypertensive urgencies and hypertensive emergencies (see Chap. 23 "Nephrology" for further details on hypertension in pediatric patients).

Definitions

- Hypertensive urgency
  - Significantly elevated BP with potential for harm but without findings of end-organ damage
  - Develops over days to weeks
- Hypertensive emergency
  - Significantly elevated BP with evidence of secondary organ damage (e.g., encephalopathy or left ventricular failure)
  - Develops over hours
- Essential (primary) hypertension
  - Hypertension in which no underlying disease is discovered
  - Multifactorial causes
  - Increasingly common due to increasing obesity, sedentary lifestyle, and poor diet
  - Diagnosis of exclusion
- Secondary hypertension
  - Result of underlying pathology
  - Many causes: cardiac, endocrine, toxic, renal, CNS

Blood pressure (BP) measurement in pediatrics

- Manual BP with auscultation is ideal
  - If initial BP via automated BP cuff is abnormal, must repeat with manual reading
- Appropriately sized BP cuff
  - Circumference completely encircles arm with overlap
  - Bladder width ~40% of arm circumference
    - Small cuff size can falsely elevate BP readings
- Attempt to obtain when patient is calm and cooperative
  - Crying, fear, pain, anxiety, fever, and hunger can all falsely elevate BP readings
  - If initial BP is elevated and patient shows no sign of end-organ failure, repeat measurement when patient has calmed

Secondary causes of hypertension

- Kidney disease
  - Chronic kidney disease, nephritis, renal artery stenosis, obstructive uropathy, Wilms tumor, etc.
  - More frequent < 6 years of age
- Cardiac disease
  - Coarctation of the aorta (most common)
    - BP in right arm > 20 mmHg above lower extremity BP

  - Others: Abdominal aortic obstruction (abdominal masses), inflammatory arteritis (e.g., Takayasu arteritis, vasculitis)
- Endocrine disease
  - Rare but highly treatable, with many potential causes
  - Examples: Congenital adrenal hyperplasia, familial hyperaldosteronism, hyperthyroidism, hyperparathyroidism, pheochromocytoma, Cushing syndrome, etc.
  - Special note on neurofibromatosis type 1 (NF1)
    - NF1 is particularly known to have multiple potential causes for hypertension
    - Association with renal artery stenosis, coarctation of the aorta, middle aortic syndrome, pheochromocytoma
- Neurologic disease
  - Many causes, including any intracranial process with increased ICP
  - Examples: Familial dysautonomia, Guillain–Barré syndrome, neuroblastoma
- Environmental/drug exposures
  - Many drugs, prescription and otherwise, can elevated BP
  - Common sources: Stimulants (pseudoephedrine, caffeine, cocaine, amphetamines), oral contraceptives, corticosteroids, anabolic steroids
  - Medication withdrawal can also induce hypertension
    - Clonidine withdrawal

Approach to severe hypertension presenting to emergency care

- History and physical to evaluate for secondary causes of hypertension
- Careful attention to signs of end-organ failure
  - Cardiac exam: Signs of congestive heart failure, pulmonary edema, absent or decreased femoral pulses, peripheral edema
  - Abdominal exam: Palpable mass, audible bruit
  - Neurologic exam: Mental status, cerebellar function
  - Ophthalmologic exam: Disc edema, hemorrhage or infarct, visual acuity
- Laboratory investigations
  - Initial investigations should be limited to basic interventions
  - CBC, electrolytes, BUN, serum creatinine, urinalysis and urine culture
  - Four-limb manual BP
  - ECG
  - Further investigations must be targeted to the suspected secondary source of the hypertension
    - Example: Echocardiogram to evaluate for left ventricular hypertrophy and coarctation; Doppler renal ultrasound to evaluate for renal artery stenosis
- Initial interventions
  - Attention to airway, breathing, and circulation
  - Establish IV access
  - Careful selection of a short-acting IV antihypertensive medication
    - The choice of antihypertensive must be tailored to patient need, with consideration of underlying disease process, potential drug–drug interactions, and medication side effects
    - Avoidance of long-acting antihypertensives to avoid overaggressive drop in BP and allow for tighter control of therapy
    - Avoidance of enteral medications due to variable effect and longer half-lives
      - Enteral medications may be considered in some cases of hypertensive urgency
  - Examples of IV pharmacologic options
    - Nicardipine
      - Calcium channel blockade
      - Reduces peripheral vascular resistance

        - Caution with intracranial processes, as can increase ICP
    - Labetalol
        - Alpha-1 and beta-blockade
        - Reduces peripheral vascular resistance
        - Contraindicated in asthma, congestive heart failure, heart block, or in pheochromocytoma or cocaine overdose (unopposed alpha effects)
    - Sodium nitroprusside
        - Potent and direct vasodilator
        - Strong arterial and venous smooth muscle relaxant with instant onset
        - Black box warning: Can cause cyanide toxicity (metabolized to thiocyanate and cyanide), must monitor thiocyanate levels
    - Esmolol
        - Beta-1-blockade
        - Metabolism is independent of liver/kidney
        - Contraindicated in asthma, heart block, congestive heart failure
    - Hydralazine
        - Arterial vasodilator
        - Can cause increased ICP and fluid retention
    - Phentolamine
        - Alpha-adrenergic antagonist
        - Reserved for hypertension from catecholamine excess, e.g., pheochromocytoma or cocaine toxicity
- Goals of therapy
  - Reduction of BP by < 25% in first 6–8 h
    - Cautious approach is warranted due to cerebrovascular autoregulation
    - Overaggressive drop in BP can put patient at risk for brain ischemia
- Further management
  - Patients with hypertensive urgencies typically require admission for further evaluation and management
  - Patients with hypertensive emergencies typically require intensive care monitoring and resuscitation

Table 7.12 Timeline for first hour management of pediatric septic shock

Timeline (min)	Intervention
0–15	ABCs, cardiorespiratory monitoring, supplemental oxygen, frequent blood pressure monitoring, strict intake and output, order intravenous access and labs
5–15	Obtain IV access, escalate to IO if unable to obtain IV
	Labs: Blood culture, venous blood gas, glucose, lactate, electrolytes, complete blood count
0–20	Initial rapid fluid resuscitation of 20 mL/kg normal saline fluid bolus
0–60	Up to 3 fluid boluses with maximum 60 mL/kg, reassessments after each additional bolus for attainment of clinical goals, have vasopressor of choice ready for infusion, monitor for need for intubation and development of pulmonary edema
0–60	Administer intravenous antibiotics within 1 h
60	Initiation of vasopressor of choice if fluid-refractory shock, consideration of need for stress dose steroids, continual reassessments
> 60	Prompt transfer to pediatric ICU within 1 h

SEPSIS AND SHOCK

The first hour management of pediatric sepsis and septic shock. Further details on sepsis and shock can be found in Chap. 8 "Critical Care."

First-hour management of septic shock

- Early recognition and resuscitation is key, although recognition is challenging
- Institutions benefit from implementing a sepsis screening tool and a sepsis intervention protocol (Table 7.12)
- Clinician assessment within 15 min
- Resuscitation begins within 30 min

PEARLS AND PITFALLS

Respiratory distress

- In the search for a localizing problem for respiratory distress, remember to carefully consider disorders external to the lung.

- A significant amount of non-airway disease can cause respiratory distress via derangement of either the respiratory system controls or acid-base status causing a secondary respiratory compensation.
- Be alert for abnormal breathing patterns in the absence of abnormal auscultatory findings.
  - Bradypnea/tachypnea may signify increased ICP or CNS depression, as in hypothermia, narcotic overdose, mass lesion, meningitis, encephalitis, spinal cord injury or neuromuscular disease, or anxiety or pain.
  - Kussmaul respirations: acidosis (diabetic ketoacidosis).
- "Not all that wheezes is asthma."
- For patients with atypical or unexpectedly severe clinical presentations of what appears to be an asthma exacerbation, always consider less common intrathoracic pathology:
  - Intrinsic lung disease (bronchopulmonary dysplasia, cystic fibrosis, pneumonitis, lung malformations), heart failure, mediastinal masses, and GERD.
- Be alert for "red flag" warning signs that can indicate imminent respiratory failure:
  - Upper airway: tripoding, drooling, gurgling, inspiratory and expiratory stridor.
  - Lower airway: grunting, head bobbing, see-saw abdominal retractions.
  - Neurologic: AMS, lethargy, extreme irritability.

The acute abdomen

- See Table 7.4 for Pearls and Pitfalls.

Anaphylaxis

- The absence of skin findings does not rule out anaphylaxis.
  - Many children will have only transient skin changes or rash.
- Anaphylaxis can present with primarily neurologic symptoms such as syncope.
- Prompt administration of intramuscular epinephrine is associated with lower risk of hospitalization and fatality.
- Delayed administration of epinephrine is associated with increased risk of hospitalization and worsened outcomes such as hypoxic-ischemic encephalopathy and death.
- Antihistamines such as diphenhydramine relieve itching through H1-blocker effects but do not help with airway or respiratory symptoms, hypotension, or shock.
- Inhaled bronchodilator therapy with albuterol can reverse bronchospasm but does not help with angioedema, hypotension, or shock.
  - Keep in mind that epinephrine is also a potent bronchodilator and will help relieve acute wheezing from anaphylaxis.

Head trauma

- Infants are at higher risk from mortality and morbidity due to head trauma from nonaccidental trauma and may not have many signs or symptoms due to limited neurologic exams.
- Always maintain a low threshold of suspicion for nonaccidental trauma.

Burns

- Always maintain a low threshold of suspicion for nonaccidental trauma.
- "Red flag" warning signs that should raise suspicion for abuse:
  - "Stocking and glove" distribution of burn: Suggest forceful immersion of extremity in hot liquid.
  - Full thickness burns: Children typically will retract extremity before this degree of injury can occur.

Inhalation injury

- Severe airway injury from smoke inhalation can occur in the absence of external burn findings.

- Risks for smoke inhalation injury include young age and exposure within a closed space.
- Due to risk for CO poisoning, early application of 100% oxygen, blood gas analysis, and carboxyhemoglobin level are important.

Altered mental status

- Broad differential diagnosis can make management of AMS a challenging diagnostic puzzle.
- Careful attention to history and physical clues and low index of suspicion for life-threatening disorders can help guide the differential diagnosis.
- Many of the more common causes of AMS are reversible if discovered promptly
  - Promptly identify and correct hypoglycemia, hypoxia, hypothermia, hypercarbia, and hypotension.

Foreign body ingestion and aspiration

- Unwitnessed foreign bodies within the airway have the potential to masquerade as more common diseases such as croup and bronchiolitis/asthma.
- Esophageal foreign bodies have the potential to cause significant airway compromise.
- Impaction is often to underlying pathology such as eosinophilic esophagitis, GERD, and known prior strictures.

Drowning

- Conduct careful assessment of all end-organ function in drowning victim.
- Underlying disease can be the precipitating cause for or a coexisting risk factor in a drowning event, e.g., toxic ingestion, intentional overdose, recreational drug use, seizure disorder, cardiac arrhythmia, hypoglycemia.
- Prevention of drowning is key.

Hypertensive crisis

- Hypertension in a young child or infant is more likely to represent secondary hypertension with identifiable cause.
- Unless there are signs of acute end-organ dysfunction, treatment of hypertension in pediatric patients is conservative.

Sepsis and shock

- Pediatric sepsis requires prompt recognition and treatment.
- Emphasis on first-hour fluid resuscitation and inotropic therapy with the following clinical goals:
  - Improvement in heart rate.
  - Normalization of BP.
  - Restoration of perfusion and pulses.
  - Reassessments after each bolus for signs of fluid overload.
  - Antibiotic administration within first hour of recognition,
  - Prompt transfer to the intensive care unit for further support.

References

1. Kuppermann N, Holmes JF, Dayan PS, Hoyle JD Jr, Atabaki SM, Holubkov R, Pediatric Emergency Care Applied Research Network (PECARN), et al. Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. Lancet. 2009;374(9696):1160–70.
2. Liang JL, Tiwari T, Moro P, Messonnier NE, Reingold A, Sawyer M, Clark TA. Prevention of pertussis, tetanus, and diphtheria with vaccines in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep. 2018;67(RR-2):1–44.
3. Rupprecht CE, Briggs D, Brown CM, Franka R, Katz SL, Kerr HD, Centers for Disease Control and Prevention (CDC), et al. Use of a reduced (4-dose) vaccine schedule for postexposure prophylaxis to prevent human rabies: recommendations of the advisory committee on immunization practices. MMWR Recomm Rep. 2010;59(RR-2):1–9.

4. Suguitan M. Rule of nines. CodeHealth, 5 Oct 2017. codehealth.io/library/article-85/rule-of-nines/. Accessed 11 Dec 2018.
5. Hoffman RS, Nelson LS, Goldfrank LR, Flomenbaum N, Howland MA. Goldfrank's toxicologic emergencies. 10th ed. New York: McGraw-Hill Education; 2015.
6. Rumack BH, Matthew H. Acetaminophen poisoning and toxicity. Pediatrics. 1975;55(6):871–6.

Suggested Reading

Respiratory Distress

Kämäräinen A. Out-of-hospital cardiac arrests in children. J Emerg Trauma Shock. 2010;3(3):273–5.

Leung AK, Cho H. Diagnosis of stridor in children. Am Fam Physician. 1999;60(8):2289–96. Review.

Weiner DL, Deanehan JK. Respiratory distress. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 451–64.

Young KD, Gausche-Hill M, McClung CD, Lewis RJ. A prospective, population-based study of the epidemiology and outcome of out-of-hospital pediatric cardiopulmonary arrest. Pediatrics. 2004;114(1):157–64.

Acute Abdomen

Bachur RG. Abdominal emergencies. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 1313–33.

Reust CE, Williams A. Acute abdominal pain in children. Am Fam Physician. 2016;03(10):830–6.

Rothrock SG, Pagane J. Acute appendicitis in children: emergency department diagnosis and management. Ann Emerg Med. 2000;36(1):39–51.

Anaphylaxis

Holmes JF, Lillis K, Monroe D, Borgialli D, Kerrey BT, Mahajan P, Pediatric Emergency Care Applied Research Network (PECARN), et al. Identifying children at very low risk of clinically important blunt abdominal injuries. Ann Emerg Med. 2013;62(2):107–16.e2.

Krishnamoorthy V, Ramaiah R, Bhananker SM. Pediatric burn injuries. Int J Crit Illn Inj Sci. 2012;2(3):128–34.

Sicherer SH, Sampson HA. Food allergy: epidemiology, pathogenesis, diagnosis, and treatment. J Allergy Clin Immunol. 2014;133(2):291–307.

Sicherer SH, Simons FER, Section on Allergy and Immunology. Epinephrine for first-aid management of anaphylaxis. Pediatrics. 2017;139(3). pii: e20164006.

Stevenson MD, Ruddy RM. Allergic emergencies. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 616–20.

Trauma and Burns

Holmes JF, Lillis K, Monroe D, Borgialli D, Kerrey BT, Mahajan P, Pediatric Emergency Care Applied Research Network (PECARN), et al. Identifying children at very low risk of clinically important blunt abdominal injuries. Ann Emerg Med. 2013;62(2):107–16.e2.

Krishnamoorthy V, Ramaiah R, Bhananker SM. Pediatric burn injuries. Int J Crit Illn Inj Sci. 2012;2(3):128–34.

Liang JL, Tiwari T, Moro P, Messonnier NE, Reingold A, Sawyer M, et al. Prevention of pertussis, tetanus, and diphtheria with vaccines in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep: Morb Mortal Wkly Rep Recomm Rep. 2018;67(2):1–44.

Menaker J, Blumberg S, Wisner DH, Dayan PS, Tunik M, Garcia M, Intra-abdominal Injury Study Group of the Pediatric Emergency Care Applied

Research Network (PECARN), et al. Use of the focused assessment with sonography for trauma (FAST) examination and its impact on abdominal computed tomography use in hemodynamically stable children with blunt torso trauma. J Trauma Acute Care Surg. 2014;77(3):427–32.

Status Epilepticus

Glauser T, Shinnar S, Gloss D, Alldredge B, Arya R, Bainbridge J, et al. Evidence-based guideline: treatment of convulsive status epilepticus in children and adults: report of the guideline Committee of the American Epilepsy Society. Epilepsy Curr. 2016;16(1):48–61.

Glissmeyer EW, Nelson DS. Coma. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 99–108.

Kimia AA, Chiang VW. Seizures. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 465–71.

Altered Mental Status

Borgialli DA, Mahajan P, Hoyle JD Jr, Powell EC, Nadel FM, Tunik MG, Pediatric Emergency Care Applied Research Network (PECARN), et al. Performance of the pediatric Glasgow coma scale score in the evaluation of children with blunt head trauma. Acad Emerg Med. 2016;23(8):878–84.

Glissmeyer EW, Nelson DS. Coma. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 99–108.

Poisoning and Toxic Exposure

Hoffman RS, Nelson LS, Goldfrank LR, Flomenbaum N, Howland MA. Goldfrank's toxicologic emergencies. 10th ed. New York: McGraw-Hill Education; 2015.

Toce MS, Burns MM. The poisoned pediatric patient. Pediatr Rev. 2017;38(5):207–20.

Foreign Body Aspiration and Ingestion

Kramer RE, Lerner DG, Lin T, Manfredi M, Shah M, Stephen TC, et al. Management of ingested foreign bodies in children: a clinical report of the NASPGHAN Endoscopy Committee. J Pediatr Gastroenterol Nutr. 2015;60(4):562–74.

Diabetic Ketoacidosis

Agus MS, Dorney K. Endocrine emergencies. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 690–717.

Cooke DW, Plotnick L, Cooke DW, Plotnick L. Management of diabetic ketoacidosis in children and adolescents. Pediatr Rev. 2008;29(12):431–6.

Glaser N, Barnett P, McCaslin I, Nelson D, Trainor J, Louie J, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. The Pediatric Medicine Collaborative Research Committee of the American Academy of Pediatrics. New Engl J Med. 2001;(4):264–9.

Rosenbloom AL. The management of diabetic ketoacidosis in children. Diabetes Ther. 2010;1(2):103–20.

Concussion/Head Injury

Iverson GL, Gardner AJ, Terry DP, Ponsford JL, Sills AK, Broshek DK, et al. Predictors of clinical recovery from concussion: a systematic review. Br J Sports Med. 2017;51(12):941–8.

Kuppermann N, Holmes JF, Dayan PS, Hoyle JD Jr, Atabaki SM, Holubkov R, Pediatric Emergency Care Applied Research Network (PECARN), et al. Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. Lancet. 2009;374(9696):1160–70.

Lumba-Brown A, Yeates KO, Sarmiento K, Breiding MJ, Haegerich TM, Gioia GA, et al. Centers for Disease Control and Prevention guideline on the diagnosis and management of mild traumatic brain injury among children. JAMA Pediatr. 2018;172(11):e182853.

Lumba-Brown A, Yeates KO, Sarmiento K, Breiding MJ, Haegerich TM, Gioia GA, et al. Diagnosis and management of mild traumatic brain injury in children: a systematic review. JAMA Pediatr. 2018;172(11):e182847.

O'Brien MJ, Howell DR, Pepin MJ, Meehan WP 3rd. Sport-related concussions: symptom recurrence after return to exercise. Orthop J Sports Med. 2017;5(10):2325967117732516.

Drowning

Brenner RA. Prevention of drowning in infants, children, and adolescents. Pediatrics. 2003;112(2):440–5.

Chandy D, Weinhouse GL. Drowning (submersion injuries). Danzi DF. UpToDate. Waltham: UpToDate. http://www.uptodate.com. Accessed 13 Dec 2018.

Idris AH, Bierens J, Perkins GD, Wenzel V, Nadkarni V, Morley P, et al. 2015 revised Utstein-style recommended guidelines for uniform reporting of data from drowning-related resuscitation: an ILCOR advisory statement. Resuscitation. 2017;118:147–58.

Salomez F, Vincent JL. Drowning: a review of epidemiology, pathophysiology, treatment and prevention. Resuscitation. 2004;63(3):261–8.

Hypertensive Crisis

Constantine E, Merritt C. Hypertension. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 225–32.

Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al; Subcommittee on screening and management of high blood pressure in children. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics. 2017;140(3). pii: e20171904. *Erratum.* Pediatrics. 2018;142(3). pii: e20181739.

Sepsis and Shock

Balamuth F, Potts DA, Funari MK, Lavelle J. Shock. In: Shaw KN, Bachur RG, editors. Fleisher & Ludwig's textbook of pediatric emergency medicine. 7th ed. Philadelphia: Wolters Kluwer; 2016. p. 605–10.

Davis AL, Carcillo JA, Aneja RK, Deymann AJ, Lin JC, Nguyen TC, et al. American College of Critical Care Medicine Clinical Practice Parameters for hemodynamic support of pediatric and neonatal septic shock. Crit Care Med. 2017;45(6):1061–93.

Howell MD, Davis AM. Management of sepsis and septic shock. JAMA. 2017;317(8):847–8.