

THE ACUTELY ILL OR INJURED CHILD

Tara L. Petersen | Abigail M. Schuh

CHAPTER 38

Assessment and Resuscitation

INITIAL ASSESSMENT

Initial assessment of an acutely ill or injured child includes rapid identification of physiologic derangements in **tissue perfusion and oxygenation**. Once identified, immediate resuscitation must be implemented before pursuing the usual information needed to develop a differential diagnosis. Initial resuscitation measures are directed at achieving and maintaining adequate tissue perfusion and oxygenation. **Oxygen delivery** depends on cardiac output, hemoglobin concentration, and hemoglobin-oxygen saturation. The latter depends on air movement, alveolar gas exchange, pulmonary blood flow, and hemoglobin-oxygen binding characteristics.

HISTORY

In the initial assessment of an acutely ill or traumatically injured child, access to historical information may be limited. Characterization of the onset of symptoms, event details, and a brief identification of underlying medical problems should be sought by members of the team not actively involved in the resuscitation. The mnemonic AMPLE can be employed to help direct a focused history during this time: A, Allergies; M, Medications; P, Past medical history; L, Last meal; and E, Events leading up to patient presentation. Attempts at identifying historical issues that affect the physiologic derangements are useful but should not delay intervention if tissue oxygenation and perfusion are markedly impaired.

PHYSICAL EXAMINATION

Initial examination must focus on the responsiveness of a patient followed by assessment of the airway, breathing, and circulation (Table 38.1) to systematically address the issues of oxygen delivery to tissues. In 2010, the American Heart Association (AHA) guidelines recommended a change from the traditional sequence of airway, breathing, circulation (ABCs) to a CAB sequence (circulation, airway, breathing) to decrease the time to the initiation of chest compression and reduce “no blood flow” time for all patients. Since this change, the survival impact of initiating resuscitation with a C-A-B versus an A-B-C sequence specifically in the pediatric population remains unclear. It is important to note that the leading

cause of cardiopulmonary arrest in children is most often of respiratory etiology (compared to a cardiac etiology in adults), and thus rapid initial assessment of the airway and breathing is vital. Regardless of the sequence approach utilized, it is best to use a consistent, algorithmic method to ensure effective and efficient cardiopulmonary assessment.

Assessment of airway patency should include evaluation of the neurologically injured child’s ability to protect the airway. Protection of the cervical spine should be initiated at this step in any child with traumatic injury or who presents with altered mental status of uncertain etiology. Assessment of breathing includes observation of respiratory effort, rate, and chest rise, plus the auscultation of air movement and the application of a pulse oximeter (when available) to identify current oxygenation status. Circulatory status is assessed by palpation for distal and central pulses, focusing on the presence and quality of the pulses. Bounding pulses and a wide pulse pressure are often the first sign of the vasodilatory phase of shock and require immediate resuscitation measures. Weak, thready, or absent pulses are indicators for fluid resuscitation, initiation of chest compressions, or both. When assessment of the ABCs is complete and measures have been taken to achieve an acceptable level of tissue oxygenation, a more complete physical examination is performed. The sequence of this examination depends on whether the situation involves an acute medical illness or trauma. In trauma patients, the examination follows the **ABCDE pathway**. **D** stands for disability and prompts assessment of the neurologic system and evaluation for major traumatic injuries. **E** stands for exposure; the child is disrobed and examined for evidence of any life-threatening or limb-threatening problems. For the acutely ill and the injured child, the physical examination should identify evidence of organ dysfunction, starting with areas suggested in the chief complaint and progressing to a thorough and systematic investigation of the entire patient.

COMMON MANIFESTATIONS

The physiologic responses to acute illness and injury are mechanisms that attempt to correct inadequacies of tissue oxygenation and perfusion. When initial changes, such as increasing heart and respiratory rates, fail to meet the body’s needs, other manifestations of impending cardiopulmonary failure occur (Table 38.2). **Respiratory failure**, the most common cause of acute deterioration in children, may result in inadequate tissue oxygenation and in respiratory acidosis. Signs and symptoms of respiratory failure (tachypnea, tachycardia, increased work of breathing, abnormal mentation) progress as tissue

TABLE 38.1 Rapid Cardiopulmonary Assessment

AIRWAY PATENCY
Able to be maintained independently
Maintainable with positioning, suctioning
Unmaintainable, requires assistance
BREATHING
Rate
Mechanics
• Retractions • Grunting • Use of accessory muscles • Nasal flaring
Air movement
• Chest expansion • Breath sounds • Stridor • Wheezing • Paradoxical chest motion
Color
CIRCULATION
Heart rate
Peripheral and central pulses
• Present/absent • Volume/strength
Skin perfusion
• Capillary refill time • Skin temperature • Color • Mottling
Blood pressure
CENTRAL NERVOUS SYSTEM PERFUSION
Responsiveness (AVPU)
Recognition of parents or caregivers
Pupillary response to light
Posturing

AVPU, Alert, responds to voice, responds to pain, unresponsive.

oxygenation becomes more inadequate. Inadequate perfusion (shock) leads to inadequate oxygen delivery and a resulting metabolic acidosis. **Shock** is characterized by signs of inadequate tissue perfusion (pallor, cool skin, poor pulses, delayed capillary refill, oliguria, and abnormal mentation). The presence of any of these symptoms demands careful assessment and intervention to correct the abnormality and to prevent further deterioration.

TABLE 38.2 Warning Signs and Symptoms Suggesting the Need for Resuscitation

SYSTEM	SIGNS AND SYMPTOMS
Central nervous system	Lethargy, agitation, delirium, obtundation, confusion
Respiratory	Apnea, grunting, nasal flaring, dyspnea, retracting, tachypnea, poor air movement, stridor, wheezing
Cardiovascular	Arrhythmia, bradycardia, tachycardia, weak pulses, poor capillary refill, hypotension
Skin and mucous membranes	Mottling, pallor, cyanosis, diaphoresis, poor membrane turgor, dry mucous membranes

**Action would seldom be taken if only one or two of these signs and symptoms were present, but the occurrence of several in concert foreshadows grave consequences. Intervention should be directed at the primary disorder.*

INITIAL DIAGNOSTIC EVALUATION

Screening Tests

During the initial phase of resuscitation, monitoring vital signs and physiologic status is the key screening activity. Continuous monitoring with attention to changes may indicate response to therapy or further deterioration requiring additional intervention. During the initial rapid assessment, diagnostic evaluation often is limited to pulse oximetry and bedside measurement of glucose levels. The latter is important in any child with altered mental status or at risk for inadequate glycogen stores (infants, malnourished patients). After resuscitation measures, further diagnostic tests and imaging are often necessary.

Diagnostic Tests and Imaging

The choice of appropriate diagnostic tests and imaging is determined by the mechanism of injury/disease and the results of evaluation after initial resuscitation. The initial evaluation of major trauma patients is focused on identifying evidence of hemorrhage and organ and tissue injury. For an acutely ill child with respiratory distress, a chest x-ray is important. Appropriate cultures (such as blood, urine, cerebrospinal fluid, and/or wound) should be obtained when sepsis is suspected. Children with historical or physical evidence of inadequate intravascular volume should have serum electrolyte levels obtained, including bicarbonate, blood urea nitrogen, and creatinine.

RESUSCITATION

Resuscitation is focused on correcting identified abnormalities of oxygenation and perfusion and preventing further deterioration. **Oxygen supplementation** may improve oxygen saturation but may not completely correct tissue oxygenation. When oxygen supplementation is insufficient or air exchange is inadequate, assisted ventilation must be initiated. Inadequate perfusion is usually best managed initially by providing a fluid bolus. **Isotonic crystalloids** (normal saline or a balanced crystalloid solution such as lactated Ringer or Plasma-Lyte) are the initial fluids of choice. A bolus

of 10–20 mL/kg should be delivered in monitored conditions. Improvement, but not correction, after an initial bolus should prompt repeated boluses until circulation has been re-established. Because most children with shock have non-cardiac causes, fluid administration of this magnitude is well tolerated. If hemorrhage is known or highly suspected, administration of packed red blood cells is appropriate. Monitoring for deteriorating physiologic status during fluid resuscitation (increase in heart rate or decrease in blood pressure) identifies children who may have diminished cardiac function. In these cases, as fluid resuscitation increases the preload, impaired cardiac function may worsen, and pulmonary edema can result. If deterioration occurs, fluid administration should be interrupted, and further resuscitation should be aimed at improving cardiac function.

When respiratory support and fluid resuscitation are insufficient, introduction of **vasoactive substances** is the next step. The choice of which agent to use depends on the type of shock present. Hypovolemic shock (when further volume is contraindicated) and distributive shock benefit from drugs that increase systemic vascular resistance (drugs with α -agonist activity, such as epinephrine or norepinephrine). The treatment of cardiogenic shock is more complex. To improve cardiac output by increasing the heart rate, drugs with positive chronotropy are used (epinephrine, norepinephrine, and dopamine). Afterload reduction, using drugs such as dobutamine, nitroprusside, or milrinone, also may be needed. Measuring central mixed venous oxygen saturation, central venous pressure, and regional oxygen saturations can also guide therapy.

CARDIOPULMONARY ARREST

The outcome of cardiopulmonary arrest in children is poor; survival to hospital discharge is ~6% for out-of-hospital arrest and ~27% for in-hospital arrest, with most survivors having some degree of permanent neurologic disability. The ability to anticipate or recognize pre-cardiopulmonary arrest conditions and initiate prompt and appropriate therapy is not only lifesaving but can also preserve functional status (see Table 38.2).

Children who require cardiopulmonary resuscitation (CPR) usually suffered a primary respiratory arrest. Hypoxia can initiate a cascade of further physiologic deterioration with subsequent organ dysfunction or damage (Table 38.3). The approach to cardiopulmonary arrest extends beyond CPR and includes efforts to preserve vital organ function. The goal in resuscitating a pediatric patient following a cardiopulmonary arrest should be to optimize **cardiac output** and tissue oxygen delivery, which may be accomplished by using artificial ventilation, chest compressions, and judicious administration of pharmacologic agents.

Pediatric Advanced Life Support and Cardiopulmonary Resuscitation

The 2010 AHA revised recommendations for resuscitation of adults, children, and infants advocated for initiation of chest compressions immediately, rather than beginning with airway and breathing (i.e., compressions-airway-breathing/ventilation [C-A-B]). The 2015 AHA update reiterated this recommendation.

TABLE 38.3 Target Organs for Hypoxic-Ischemic Damage

ORGAN	EFFECT
Brain	Seizures, cerebral edema, infarction, herniation, anoxic damage, SIADH, diabetes insipidus
Cardiovascular	Heart failure, myocardial infarct
Lung and pulmonary vasculature	Acute respiratory distress syndrome, pulmonary hypertension
Liver	Infarction, necrosis, cholestasis
Kidney	Acute tubular necrosis, acute cortical necrosis
Gastrointestinal tract	Gastric ulceration, mucosal damage
Hematologic	Disseminated intravascular coagulation

SIADH, Syndrome of inappropriate secretion of antidiuretic hormone.

Circulation

Chest compressions should be initiated if a pulse cannot be palpated or if the heart rate is less than 60 beats/min with concurrent signs of poor systemic perfusion. Chest compressions should be performed immediately by one person while a second person prepares to begin ventilation. **Ventilation** is extremely important in pediatric arrests given the high likelihood of a primary respiratory cause; however, ventilation requires equipment and is therefore sometimes delayed. For this reason, the recommendation is to start chest compressions first while preparing for ventilation.

For optimal chest compressions, the child should be supine on a flat, hard surface. Effective CPR requires a compression depth of one third to one half of the anterior-posterior diameter of the chest with complete recoil after each compression. The compression rate should be at least 100/min with breaths delivered 8–10 times/min. If an advanced airway is in place, compressions should not pause for ventilation; both should continue simultaneously.

Airway

Ventilation requires a **patent airway**. In children, airway patency often is compromised by a loss of muscle tone, allowing the mandibular block of tissue, including the tongue, bony mandible, and the soft surrounding tissues, to rest against the posterior pharyngeal wall. The head tilt–chin lift maneuver should be used to open the airway in children with no sign of head or neck trauma. In children with signs of head or neck trauma, the jaw thrust maneuver should be used.

Bag-mask ventilation can be as effective as, and possibly safer than, endotracheal intubation for short periods of time in an out-of-hospital setting. If skilled personnel and proper equipment are available, pediatric patients requiring resuscitation should be endotracheally intubated. Before intubation, the patient should be ventilated with 100% oxygen using a bag and mask. Cricoid pressure can be used to minimize inflation of the stomach. Many conscious patients may benefit from the use of induction medications (sedatives, analgesics, and neuromuscular blockade) to assist intubation, but caution is necessary to prevent further cardiovascular compromise from

TABLE 38.4 Drug Doses for Cardiopulmonary Resuscitation

DRUG	INDICATION	DOSE
Adenosine	Supraventricular tachycardia	IV: 0.1 mg/kg (maximum 6 mg); second dose: 0.2 mg/kg (maximum 12 mg)
Amiodarone	Pulseless VF/VT	IV/IO: 5 mg/kg; may repeat twice up to 15 mg/kg; maximum single dose 300 mg
	Perfusing tachyarrhythmias	Dose as above but administer slowly over 20–60 min. Expert consultation strongly recommended
Atropine	Supraventricular or junctional bradycardia	IV/IO/IM: 0.02 mg/kg; ET: 0.04–0.06 mg/kg; maximum single dose: 0.5 mg; higher doses needed in organophosphate poisoning
Bicarbonate	Hyperkalemia, some toxidromes	IV/IO: 1 mEq/kg bolus; ensure adequate ventilation; monitor ABGs; can repeat every 10 min
Calcium chloride (10%)	Hypocalcemia, calcium channel blocker overdose, hypermagnesemia, hyperkalemia	IV/IO: 20 mg/kg; maximum single dose 2 g; administer slowly
Dextrose	Hypoglycemia	IV/IO: Newborns: 5–10 mL/kg of 10% dextrose; infants and children: 2–4 mL/kg of 25% dextrose; adolescents: 1–2 mL/kg of 50% dextrose
Epinephrine	Hypotension, chronotropy, inotropy	IV/IO: 0.01 mg/kg (0.1 mL/kg 1:10,000); ET: 0.1 mg/kg (0.1 mL/kg 1:1,000); may repeat every 3–5 min; may promote arrhythmias
Fluid	Hypovolemia, sepsis	IV/IO: Administer crystalloid in 20 mL/kg boluses titrated to patient's physiologic needs
Lidocaine	VT	IV/IO: 1 mg/kg/bolus followed by 20–50 µg/kg/min continuous infusion; ET: Loading dose: 2–3 mg/kg/dose; flush with 5 mL of NS and follow with five assisted manual ventilations

ABG, Arterial blood gas; ET, endotracheal tube; IM, intramuscular; IO, intraosseous; IV, intravenous; VF, ventricular fibrillation; VT, ventricular tachycardia. Data from the American Heart Association. Web-based integrated guidelines for cardiopulmonary and emergency cardiovascular care—part 12. Pediatric advanced life support. <https://eccguidelines.heart.org/index.php/circulation/cpr-ecc-guidelines-2/part-12-pediatric-advanced-life-support/>.

vasodilating effects of many of these agents. The correct size of the tube may be estimated according to the size of the child's mid-fifth phalanx or the following formula: 4 + (patient age in years/4).

When the endotracheal tube is in place, the adequacy of ventilation and the position of the tube must be assessed. Use of both clinical assessment and confirmatory devices is recommended. Clinical assessment includes looking for adequate chest wall movement and auscultation of the chest to detect bilateral and symmetric breath sounds. Confirmatory devices, such as end-tidal carbon dioxide (CO₂) monitoring devices, are useful for the validation of endotracheal placement, but low levels of detected CO₂ may be seen secondary to lack of pulmonary circulation. If the patient's condition fails to improve or deteriorates, consider the possibilities of tube Displacement or Obstruction, Pneumothorax, or Equipment failure (mnemonic DOPE).

Breathing

The major role of endotracheal intubation is to protect or maintain the airway and ensure the delivery of adequate oxygen to the patient. Because hypoxemia is the final common pathway in pediatric cardiopulmonary arrests, providing oxygen is more important than correcting the respiratory acidosis. The clinician should deliver 100% oxygen at a rate of 8–10 breaths/min during CPR, or 20–30 breaths/min for a patient who has a perfusing rhythm. Use only the tidal volume necessary to produce visible chest rise. Care should be taken not to hyperventilate the patient.

Medications

When mechanical means fail to re-establish adequate circulation, pharmacologic intervention is essential (Table 38.4). If intravascular access is not present or rapidly established, administration through an intraosseous route is recommended. Some drugs can also be administered effectively through the endotracheal tube or intramuscularly.

Epinephrine, a catecholamine with mixed α -agonist and β -agonist properties, constitutes the mainstay of drug therapy for CPR. The α -adrenergic effects are most important during acute phases of resuscitation, causing an increase in systemic vascular resistance that improves coronary blood flow. Standard dose therapy is recommended for the first and subsequent boluses. There is no benefit offered by high-dose epinephrine. Vasopressin, an endogenous hormone, causes constriction of capillaries and small arterioles and may be useful. Data are insufficient to support its routine use; however, vasopressin may be considered in children failing standard medication administration.

The routine use of **sodium bicarbonate** is currently not recommended. Sodium bicarbonate may be judiciously used to treat toxidromes or hyperkalemic arrest; however, oxygen delivery and elimination of CO₂ must be established first. Side effects include hypernatremia, hyperosmolality, hypokalemia, metabolic alkalosis (shifting the oxyhemoglobin curve to the left and impairing tissue oxygen delivery), reduced ionized calcium level, and impaired cardiac function.

Routine administration of **calcium** is not recommended. It may be useful in cases of documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hypokalemia but is otherwise not beneficial and is potentially harmful.

TABLE 38.5 Recommendations for Defibrillation and Cardioversion in Children**DEFIBRILLATION**

Place self-adhesive defibrillation pads or paddles with electrode gel at the apex of the heart and the upper right side of the chest.

- Use infant paddles or self-adhesive pads for children <10 kg; adult size for children >10 kg.

Notify all participating personnel before discharging paddles so that no one is in contact with patient or bed.

Begin with 2J/kg; resume chest compressions immediately.

If unsuccessful, increase to 4J/kg and repeat.

Higher energy levels may be considered, not to exceed 10J/kg or the adult maximum dose.

CARDIOVERSION

Consider sedation if possible.

For symptomatic supraventricular tachycardia* or ventricular tachycardia with a pulse, synchronize signal with ECG.

Choose paddles, position pads, and notify personnel as above.

Begin with 0.5–1 J/kg.

If unsuccessful, use 2J/kg.

*Consider adenosine first (see Table 38.4).
ECG, Electrocardiogram.

Hypoglycemia is common in infants and children who sustain cardiac arrest. Blood glucose should be checked, and hypoglycemia should be promptly treated with **glucose**.

Prompt electrical **defibrillation** is indicated when ventricular fibrillation or pulseless ventricular tachycardia is noted (Table 38.5). CPR should continue until immediately before defibrillation and resume immediately afterward, minimizing interruptions in compressions. If a second attempt at defibrillation is necessary, it should be followed by a dose of epinephrine. Children failing two episodes of defibrillation may benefit from administration of amiodarone or lidocaine. Defibrillation should be distinguished from **cardioversion** of supraventricular tachycardias (which can also cause cardiovascular compromise). Cardioversion requires a lower starting dose and synchronization of the discharge to the electrocardiogram to prevent discharging during a susceptible period, which may convert supraventricular tachycardia to ventricular tachycardia or fibrillation.

PEARLS FOR PRACTITIONERS

See *The Acutely Ill or Injured Child: Pearls for Practitioners* at the end of this section.

($\text{PaCO}_2 > 50$ mm Hg in previously healthy children), hypoxemic ($\text{PaO}_2 < 60$ mm Hg in previously healthy children without an intracardiac shunt), or both. **Hypoxemic respiratory failure** is frequently caused by ventilation-perfusion mismatch (perfusion of lung that is not adequately ventilated) and shunting (deoxygenated blood bypasses ventilated alveoli). **Hypercarbic respiratory failure** results from inadequate alveolar ventilation secondary to decreased minute ventilation (tidal volume $\times$ respiratory rate) or an increase in dead space ventilation (ventilation of areas receiving no perfusion).

Respiratory failure may occur with **acute respiratory distress syndrome (ARDS)**. The pediatric-specific definition for acute respiratory distress syndrome (PARDS) builds on the adult-based Berlin definition but has been modified to account for differences between adults and children with ARDS. Pediatric ARDS, and its severity, is defined by (1) timing: onset within 7 days of known clinical insult; (2) respiratory failure not fully explained by cardiac failure or fluid overload; (3) chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease; and (4) impairment in oxygenation (as described in Table 39.1). Pediatric ARDS can be triggered by a variety of insults including sepsis, pneumonia, shock, burns, or traumatic injury, all resulting in inflammation and increased vascular permeability leading to pulmonary edema. Numerous mediators of inflammation (tumor necrosis factor, interferon- γ , nuclear factor κB , and adhesion molecules) as well as surfactant action may be involved in the development of ARDS.

EPIDEMIOLOGY

Acute respiratory failure is frequently caused by bronchiolitis, often caused by respiratory syncytial virus (RSV), asthma, pneumonia, upper airway obstruction, and systemic inflammation resulting in ARDS. Notably, bronchiolitis and asthma are the most common causes of acute respiratory failure in pediatric patients. RSV infects 90% of children within the first 2 years of life and frequently reinfects older children and adults. The World Health Organization estimates that RSV is responsible for ~33 million lower respiratory tract illnesses, 3 million hospitalizations, and up to 199,000 childhood deaths each year. Asthma is the most common chronic condition among children, currently affecting an estimated 6.1 million children under 18 years in the United States, of which 3.5 million suffered from an asthma attack or episode in 2016. Environmental factors (exposure to cigarette smoke) and prior disease characteristics (severity of asthma, exercise intolerance, delayed start of therapy, and previous intensive care unit admissions) affect the rate of hospitalization and near-fatal episodes.

Chronic respiratory failure (with acute exacerbations) is often due to chronic lung disease (bronchopulmonary dysplasia, cystic fibrosis), neurologic or neuromuscular abnormalities, and congenital anomalies.

CLINICAL MANIFESTATIONS

Early signs of hypoxic respiratory failure include **tachypnea** and **tachycardia** which are physiologic compensatory mechanisms to improve minute ventilation and cardiac output to maintain delivery of oxygenated blood to the tissues. Further progression of disease may result in dyspnea, nasal flaring,

CHAPTER 39

Respiratory Failure

ETIOLOGY

Acute respiratory failure occurs when the pulmonary system is unable to maintain adequate gas exchange to meet metabolic demands. The resulting failure can be classified as hypercarbic

TABLE 39.1 Pediatric Acute Respiratory Distress Syndrome Severity Based on Oxygenation

NONINVASIVE MECHANICAL VENTILATION PARDS (no severity stratification)	INVASIVE MECHANICAL VENTILATION PARDS (severity per OI or OSI)
Full face mask: bilevel positive airway pressure or continuous positive airway pressure ≥ 5 H ₂ O: PF ratio ≤ 300 SF ratio ≤ 264	Mild: $4 \leq \text{OI} < 8$; $5 \leq \text{OSI} < 7.5$ Moderate: $8 \leq \text{OI} < 16$; $7.5 \leq \text{OSI} < 12.3$ Severe: $\text{OI} > 16$; $\text{OSI} \geq 12.3$

PF ratio = $\text{PaO}_2/\text{FiO}_2$; if PaO_2 is not available, wean FiO_2 to maintain $\text{SpO}_2 \leq 97\%$ to calculate the SF ratio = $\text{SpO}_2/\text{FiO}_2$.

OI = oxygenation index = $[\text{FiO}_2 \times \text{mean airway pressure} \times 100]/\text{PaO}_2$; if PaO_2 is not available, wean FiO_2 to maintain $\text{SpO}_2 \leq 97\%$ to calculate the oxygen saturation index (OSI); $[\text{FiO}_2 \times \text{mean airway pressure} \times 100]/\text{SpO}_2$.

PARDS, Pediatric acute respiratory distress syndrome.

Modified from Khemani RG, Smith LS, Zimmerman JJ, et al. Pediatric acute respiratory distress syndrome: definition, incidence and epidemiology: proceeding from the Pediatric Acute Lung Injury Consensus Conference. *Pediatr Crit Care Med*. 2015;16(5 suppl 1):S23–S40 [fig. 2].

grunting, use of accessory muscles of respiration, and diaphoresis. Late signs of inadequate oxygen delivery include **cyanosis** and **altered mental status** (initially confusion and agitation). Signs and symptoms of hypercarbic respiratory failure include signs of increased minute ventilation (tachypnea and increased depth of breathing) and altered mental status (somnolence).

LABORATORY AND IMAGING STUDIES

A chest radiograph may show evidence of the etiology of respiratory failure. The detection of atelectasis, hyperinflation, infiltrates, or pneumothoraces assists with ongoing management. Diffuse infiltrates or pulmonary edema may suggest ARDS. The chest radiograph may be normal when upper airway obstruction or impaired respiratory controls are the etiology. In patients presenting with stridor or other evidence of upper airway obstruction, a lateral neck film or CT may delineate anatomic defects. Direct visualization through flexible bronchoscopy allows the identification of dynamic abnormalities of the anatomic airway. Helical CT helps diagnose a pulmonary embolus.

Pulse oximetry allows noninvasive, continuous assessment of oxygenation but is unable to provide information about ventilation abnormalities. Determination of CO₂ levels requires a blood gas measurement (venous, capillary, or arterial). An **arterial blood gas** is the gold standard for measurement of serum CO₂ levels and allows for analysis of the severity of oxygenation defect through calculation of an alveolar-arterial oxygen difference. A normal PaCO₂ in a patient who is hyperventilating should heighten concern about the risk of further deterioration.

DIFFERENTIAL DIAGNOSIS

Hypoxic respiratory failure resulting from impairment of alveolar-capillary function is seen in ARDS; cardiogenic pulmonary edema; interstitial lung disease; aspiration pneumonia; bronchiolitis; bacterial, fungal, or viral pneumonia; and sepsis. It also can be due to intracardiac or intrapulmonary shunting seen with atelectasis and pulmonary embolism.

Hypercarbic respiratory failure can occur when the respiratory center fails as a result of drug use (opioids, barbiturates, anesthetic agents), neurologic or neuromuscular junction abnormalities (cervical spine trauma, demyelinating diseases, anterior horn cell disease, botulism), chest wall injuries, or

diseases that cause increased resistance to airflow (croup, vocal cord paralysis, post-extubation edema). Maintenance of ventilation requires adequate function of the chest wall and diaphragm. Disorders of the neuromuscular pathways, such as muscular dystrophy, myasthenia gravis, and botulism, result in inadequate chest wall movement, development of atelectasis, and respiratory failure. Scoliosis, although rarely, can result in significant chest deformity that leads to restrictive pulmonary function. Similar impairments of air exchange may result from distention of the abdomen (postoperatively or due to ascites, obstruction, or a mass) and thoracic trauma (flail chest).

Mixed forms of respiratory failure are common and occur when disease processes result in more than one pathophysiologic change. Increased secretions seen in asthma often lead to atelectasis and hypoxia, whereas restrictions of expiratory airflow may lead to hypercarbia. Progression to respiratory failure results from peripheral airway obstruction, extensive atelectasis, and resultant hypoxemia and retention of CO₂.

TREATMENT

Initial treatment of patients in respiratory distress includes addressing the ABCs (see Chapter 38). **Bag-mask ventilation** must be initiated for patients with apnea. In other patients, oxygen therapy is administered using appropriate methods (e.g., nasal cannula). In patients with mild distress, oxygen therapy administered by nasal cannula or simple mask may be appropriate. However, administration of oxygen by these methods allows the patient to entrain a mixture of room air and supplemental oxygen, making it an insufficient delivery method for most children in respiratory failure. Delivery methods, including intubation and mechanical ventilation, should be employed if there is an inability to increase oxygen saturation or ventilate appropriately.

Patients presenting with hypercarbic respiratory failure are often hypoxic as well. When oxygenation is established, measures should be taken to address the underlying cause of hypercarbia. Patients who are hypercarbic without signs of respiratory fatigue or somnolence may not require intubation based on the PCO₂ alone; however, patients with marked increase in the work of breathing or inadequate respiratory effort may require assistance with ventilation.

After identification of the etiology of respiratory failure, specific interventions and treatments are tailored to the needs

of the patient. External support of oxygenation and ventilation may be provided by **noninvasive ventilation** methods (heated humidified high-flow nasal cannula, continuous positive airway pressure, biphasic positive airway pressure, or negative pressure ventilation) or through invasive methods (traditional **mechanical ventilation**, high-frequency oscillatory ventilation, or extracorporeal membrane oxygenation). Elimination of CO₂ is achieved through manipulation of minute ventilation (tidal volume and respiratory rate). Oxygenation is improved by altering variables that affect oxygen delivery (fraction of inspired oxygen) or mean airway pressure (positive end-expiratory pressure [PEEP], peak inspiratory pressure, inspiratory time, gas flow).

COMPLICATIONS

The major complication of hypoxic respiratory failure is the development of organ dysfunction. **Multiple organ dysfunction** includes the development of two or more of the following: respiratory failure, cardiac failure, renal insufficiency/failure, gastrointestinal or hepatic insufficiency, disseminated intravascular coagulation, and hypoxic-ischemic brain injury. Mortality rates increase with increasing numbers of involved organs (see [Table 38.3](#)).

Complications associated with mechanical ventilation include pressure-related and volume-related lung injury. Both overdistention and insufficient lung distention (loss of functional residual capacity) are associated with lung injury. Pneumomediastinum and pneumothorax are potential complications of both the disease process and iatrogenic overdistention. Inflammatory mediators may play a role in the development of chronic fibrotic lung diseases in ventilated patients.

PROGNOSIS

Prognosis varies with the etiology of respiratory failure. Less than 1% of previously healthy children with bronchiolitis die. Asthma mortality rates, although still low, have increased. Population-based studies report a variable mortality rate for pediatric ARDS from 18–35% depending on the study population.

PREVENTION

Prevention strategies are explicit to the etiology of respiratory failure. Some infectious causes can be prevented through active immunization against organisms causing primary respiratory disease (pertussis, pneumococcus, *Haemophilus influenzae* type b, seasonal influenza) and sepsis (pneumococcus, *H. influenzae* type b). Passive immunization with respiratory syncytial virus immunoglobulins prevents severe illness in highly susceptible patients (prematurity, bronchopulmonary dysplasia). Primary prevention of traumatic injuries may decrease the incidence of pediatric ARDS. Compliance with appropriate therapies for asthma may decrease the number of episodes of respiratory failure (see [Chapter 78](#)).

PEARLS FOR PRACTITIONERS

See *The Acutely Ill or Injured Child: Pearls for Practitioners* at the end of this section.

CHAPTER 40

Shock

ETIOLOGY AND EPIDEMIOLOGY

Shock is the inability to provide sufficient perfusion of oxygenated blood and substrate to tissues to meet metabolic demands. **Oxygen delivery** is directly related to the arterial oxygen content (oxygen saturation and hemoglobin concentration) and to cardiac output (stroke volume and heart rate). Changes in metabolic needs are met primarily by adjustments in cardiac output. Stroke volume is related to myocardial end-diastolic fiber length (preload), myocardial contractility (inotropy), and resistance of blood ejection from the ventricle (afterload; see [Chapter 145](#)). In a young infant whose myocardium possesses relatively less contractile tissue, increased demand for cardiac output is met primarily by a neurally mediated increase in heart rate. In older children and adolescents, cardiac output is most efficiently augmented by increasing stroke volume through neurohormonally mediated changes in vascular tone, resulting in increased venous return to the heart (increased preload), decreased arterial resistance (decreased afterload), and increased myocardial contractility.

Once the initial assessment of an acutely ill child is completed, the constellation of clinical characteristics can suggest one of the five broad classifications of shock: hypovolemic, distributive, cardiogenic, obstructive, and dissociative.

HYPOVOLEMIC SHOCK

Acute hypovolemia is the most common cause of shock in children. It results from loss of fluid from the intravascular space secondary to inadequate intake or excessive losses (vomiting and diarrhea, blood loss, capillary leak syndromes, or pathologic renal fluid losses) ([Table 40.1](#)). Reduced blood volume decreases preload, stroke volume, and cardiac output. Hypovolemic shock results in increased sympathoadrenal activity, producing an increased heart rate and enhanced myocardial contractility. Neurohormonally mediated constriction of the arterioles and capacitance vessels maintains blood pressure, augments venous return to the heart to improve preload, and redistributes blood flow from nonvital to vital organs. If hypovolemic shock remains untreated, the increased heart rate may impair coronary blood flow and ventricular filling, while elevated systemic vascular resistance increases myocardial oxygen consumption, resulting in worsening myocardial function. Ultimately, intense systemic vasoconstriction and hypovolemia produce tissue ischemia, impairing cell metabolism and releasing potent vasoactive mediators from injured cells. Cytokines and other vasoactive peptides can change myocardial contractility and vascular tone and promote release of other inflammatory mediators that increase capillary permeability and impair organ function further.

DISTRIBUTIVE SHOCK

Distributive shock results from a relative inadequacy of intravascular volume caused by venous or arterial vasodilation. This maldistribution of blood flow can result in profound inadequacies in tissue perfusion, even in the presence of a normal

TABLE 40.1 Classification of Shock and Common Underlying Causes

TYPE	PRIMARY CIRCULATORY DERANGEMENT	COMMON CAUSES
Hypovolemic	Decreased circulating blood volume	Hemorrhage Diarrhea Diabetes insipidus, diabetes mellitus Burns Adrenogenital syndrome Capillary leak
Distributive	Vasodilation → venous pooling → decreased preload Maldistribution of regional blood flow	Sepsis Anaphylaxis CNS/spinal injury Drug intoxication
Cardiogenic	Decreased myocardial contractility	Congenital heart disease Arrhythmia Hypoxic/ischemic injuries Cardiomyopathy Metabolic derangements Myocarditis Drug intoxication Kawasaki disease
Obstructive	Mechanical obstruction to ventricular filling or outflow	Cardiac tamponade Massive pulmonary embolus Tension pneumothorax Cardiac tumor
Dissociative	Oxygen not appropriately bound or released from hemoglobin	Carbon monoxide poisoning Methemoglobinemia

CNS, Central nervous system.

or high cardiac output. Septic shock is the most common type of distributive shock in children. Other causes include anaphylaxis, neurologic injury, and ingestion of certain drugs or poisons (see [Table 40.1](#)).

Distributive shock may present with the **systemic inflammatory response syndrome** (SIRS), defined as two or more of the following: temperature greater than 38°C or less than 36°C; heart rate greater than 90 beats/min or more than two standard deviations above normal for age; tachypnea; or white blood cell count that is greater than 12,000 cells/mm³, less than 4,000 cells/mm³, or has greater than 10% immature forms. Although SIRS is part of the criteria for pediatric sepsis (SIRS + proven or suspected infection = sepsis), it can also be seen in non-infectious states such as trauma or postoperatively.

CARDIOGENIC SHOCK

Cardiogenic shock is caused by an abnormality in myocardial function and is expressed as depressed myocardial contractility and cardiac output with poor tissue perfusion. Compensatory mechanisms may contribute to the progression of shock by depressing cardiac function further. Neurohormonal vasoconstrictor responses increase afterload and add to the work of the failing ventricle. Tachycardia may impair coronary blood flow, which decreases myocardial oxygen delivery. Increased central blood volume is caused by sodium and water retention and by incomplete emptying of the ventricles during systole. This results in elevated left ventricular volume and pressure, which then impairs subendocardial blood flow. As compensatory mechanisms are overcome, the failing left ventricle produces increased ventricular end-diastolic volume and pressure, which leads to increased left atrial pressure, resulting in pulmonary edema. This sequence also contributes to right ventricular failure because of increased pulmonary artery pressure and increased right ventricular afterload.

Primary cardiogenic shock may occur in children who have congenital heart disease. Cardiogenic shock also may occur in previously healthy children secondary to viral myocarditis, dysrhythmias, toxic or metabolic abnormalities, or after hypoxic-ischemic injury (see [Chapters 142, 145, and 147](#); see [Table 40.1](#)).

OBSTRUCTIVE SHOCK

Obstructive shock results from mechanical obstruction of the great vessels or of the heart itself. The pathophysiology of obstructive shock can be classified according to the location of the obstruction within the vascular system in relation to the heart. These extravascular, intravascular, or luminal obstructions result in a shock state due to reduced cardiac outflow and the associated critical drop in global oxygen supply. Etiologies of obstructive shock include both congenital lesions (such as coarctation of the aorta, interrupted aortic arch, and severe aortic valvular stenosis) and acquired diseases (such as pulmonary embolus and cardiac tamponade) (see [Table 40.1](#)).

DISSOCIATIVE SHOCK

Dissociative shock refers to conditions in which tissue perfusion is normal, but cells are unable to utilize oxygen because the hemoglobin has an abnormal affinity for oxygen, preventing its release to the tissues (see [Table 40.1](#)).

CLINICAL MANIFESTATIONS

All forms of shock produce evidence that tissue perfusion and/or oxygenation are insufficient (increased heart rate, abnormal blood pressure, alterations of peripheral pulses). The etiology of shock may alter the initial presentation of these signs and symptoms.

Hypovolemic Shock

Hypovolemic shock is distinguished from other causes of shock by history and the absence of signs of heart failure or sepsis. In addition to the signs of sympathoadrenal activity (tachycardia, vasoconstriction), clinical manifestations include signs of dehydration (dry mucous membranes, decreased

urine output) or blood loss (pallor). Recovery depends on the degree of hypovolemia, the patient's pre-existing status, and rapid diagnosis and treatment. The prognosis is good, with a low mortality in uncomplicated cases.

Distributive Shock

Patients with distributive shock usually have tachycardia and alterations of peripheral perfusion. In early stages, when cytokine release results in vasodilation, pulses may be bounding and vital organ function may be maintained (an alert patient, with rapid capillary refill and some urine output in *warm shock*). As the disease progresses untreated, extremities become cool and mottled with a delayed capillary refill time. At this stage, the patient has hypotension and vasoconstriction.

Cardiogenic Shock

Cardiogenic shock results when the myocardium is unable to supply the cardiac output required to support tissue perfusion and organ function. Because of this self-perpetuating cycle, heart failure progressing to death may be rapid. Patients with cardiogenic shock have tachycardia and tachypnea. The liver is usually enlarged, a gallop is often present, and jugular venous distention may be noted. Because renal blood flow is poor, sodium and water are retained, resulting in anasarca.

Obstructive Shock

Restriction of cardiac output results in an increase in heart rate and an alteration of stroke volume. The pulse pressure is narrow (making pulses harder to feel), and capillary refill is delayed. The liver is often enlarged, and jugular venous distention may be evident.

Dissociative Shock

The principal abnormality in dissociative shock is the inability to deliver oxygen to tissues. Symptoms include tachycardia, tachypnea, alterations in mental status, and ultimately cardiovascular collapse.

LABORATORY AND IMAGING STUDIES

Shock requires immediate resuscitation before obtaining comprehensive laboratory or diagnostic studies. Following initial stabilization (including glucose administration if hypoglycemia is present), the type of shock dictates the necessary laboratory studies. Patients with shock may benefit from the determination of a baseline pH via arterial blood gas and blood lactate level to assess the degree of tissue oxygenation impairment. Measurement of a central **mixed venous oxygen saturation** aids in the assessment of the adequacy of oxygen delivery. Notably, in contrast to other forms of shock, patients with vasodilated distributive shock (such as in gram-negative rod sepsis) often have high mixed venous saturation values due to impairment of mitochondrial function and inability of tissues to extract oxygen. A complete blood count (CBC) can reveal altered bone marrow function or provide evidence of disseminated intravascular coagulation (DIC) and may also aid in assessing the severity of blood loss following equilibration in hemorrhagic shock (of note, a CBC may not reveal the true

degree of blood loss in the early acute phase of hemorrhagic shock). Electrolyte measurements in patients with hypovolemic shock may identify abnormalities from losses. Patients presenting in septic shock require appropriate bacterial and viral cultures to aid in identification of a causative pathogen. If cardiogenic or obstructive shock is suspected, an echocardiogram assists with the diagnosis and, in the case of tamponade, assists with placement of a pericardial drain to relieve the fluid. Patients with dissociative shock require detection of the causative agent (carbon monoxide, methemoglobin). The management of shock also requires ongoing monitoring of mixed venous oxygen saturations and arterial blood gases for assessment of oxygenation, ventilation (CO_2), and degree of acidosis. Frequent re-assessment of serum electrolytes and biochemical markers of end-organ impairment, such as liver and renal function panels as well as coagulation factors and CBC, may aid in subsequent therapeutic interventions.

DIFFERENTIAL DIAGNOSIS

See Table 40.1.

TREATMENT

General Principles

The key to therapy is the recognition of shock in its early, partially compensated state, when many of the hemodynamic and metabolic alterations may be reversible. Initial therapy for shock follows the ABCs of resuscitation (see Chapter 38). Later, therapy can then be directed at the underlying cause. Therapy should minimize cardiopulmonary work, while ensuring cardiac output, adequate perfusion, and gas exchange. Intubation, combined with mechanical ventilation with oxygen supplementation, improves oxygenation and decreases or eliminates the work of breathing but may impede venous return if distending airway pressures (positive end-expiratory pressure [PEEP] or peak inspiratory pressure) are excessive. Blood pressure support is crucial in promoting adequate end-organ perfusion.

Monitoring a child in shock requires maintaining access to the arterial and central venous circulation to record pressure measurements, perform blood sampling, and measure systemic blood pressure continuously. These measurements facilitate the estimation of preload and afterload. Regional monitoring with near infrared spectroscopy (NIRS) may allow early, noninvasive detection of alterations in perfusion.

Organ-Directed Therapeutics

Fluid Resuscitation

Alterations in preload have a dramatic effect on cardiac output. In hypovolemic and distributive shock, decreased preload significantly impairs cardiac output. In these cases, early and aggressive fluid resuscitation is important and greatly affects outcome. In cardiogenic shock, an elevated preload contributes to pulmonary edema.

Selection of fluids for resuscitation and ongoing use is dictated by clinical circumstances. Crystalloid volume expanders are generally recommended as initial choices because they are effective, inexpensive, and often readily available. Most acutely ill children with signs of shock may safely receive, and usually benefit greatly from, rapid administration of a 20 mL/kg bolus

TABLE 40.2 Medications Used to Improve Cardiac Output

	POSITIVE INOTROPE	POSITIVE CHRONOTROPE	DIRECT PRESSOR	VASOCONSTRICTOR	VASODILATOR
Dopamine	++	+	±	++ (high dose)	+ (low dose)
Dobutamine	++	±	–	–	+
Epinephrine	+++	+++	+++	++ (high dose)	+ (low dose)
Norepinephrine	+++	+++	+++	+++	–
Milrinone	+	–	–	–	+

of an isotonic crystalloid. This dose may be repeated until a response is noted. Colloids contain larger molecules that may stay in the intravascular space longer than crystalloid solutions and exert oncotic pressure, drawing fluid out of the tissues into the vascular compartment. However, the long-term risks of colloids may exceed the benefits. Care must be exercised in treating cardiogenic shock with volume expansion as the ventricular filling pressures may rise without improvement or with deterioration of cardiac performance. Careful monitoring of cardiac output via vital signs, clinical exam (repeatedly evaluate for development of rales and/or hepatomegaly), and central venous pressure (if available) guides safe volume replacement.

Cardiovascular Support

In an effort to improve cardiac output after volume resuscitation, or when further volume replacement may be dangerous, a variety of inotropic and vasodilator drugs may be useful (Table 40.2). Therapy is directed first at increasing myocardial contractility, then at decreasing left ventricular afterload. The hemodynamic status of the patient dictates the choice of the agent.

Therapy may be initiated with dopamine at 5–20 µg/kg per minute; however, epinephrine or norepinephrine may be preferable in patients with decompensated shock. In addition to improving contractility, certain catecholamines cause an increase in systemic vascular resistance. The addition of a vasodilator drug may improve cardiac performance by decreasing the resistance against which the heart must pump (afterload). Afterload reduction may be achieved with dobutamine, milrinone, amrinone, nitroprusside, nitroglycerin, and angiotensin-converting enzyme inhibitors. The use of these drugs may be particularly important in the later stages of shock, when vasoconstriction is prominent.

Respiratory Support

The lung is a target organ for inflammatory mediators in SIRS and shock. Respiratory failure may develop rapidly and become progressive. Intervention requires endotracheal intubation and mechanical ventilation accompanied by the use of supplemental oxygen and PEEP. Care must be taken with the process of intubation as a child with compensated shock may suddenly decompensate upon administration of sedative medications that reduce systemic vascular resistance or with converting to positive pressure ventilation. Severe cardiopulmonary failure may be managed with inhaled nitric oxide and, if necessary, extracorporeal membrane oxygenation.

Renal Salvage

Poor cardiac output accompanied by decreased renal blood flow may cause prerenal azotemia and oliguria/anuria. Severe hypotension may produce **acute tubular necrosis** and **acute renal failure**. Prerenal azotemia is corrected when blood volume deficits are replaced or myocardial contractility is improved, but acute tubular necrosis does not improve immediately when shock is corrected. Prerenal azotemia is associated with a serum blood urea nitrogen (BUN)–to-creatinine ratio of greater than 10:1 and a urine sodium level less than 20 mEq/L; acute tubular necrosis has a BUN-to-creatinine ratio of 10:1 or less and a urine sodium level between 40 and 60 mEq/L (see Chapter 165). Aggressive fluid replacement is often necessary to improve oliguria associated with prerenal azotemia. Because the management of shock requires administering large volumes of fluid, maintaining urine output greatly facilitates patient management.

Prevention of acute tubular necrosis and the subsequent complications associated with acute renal failure (hyperkalemia, acidosis, hypocalcemia, fluid overload) is important. The use of pharmacologic agents to augment urine output is indicated when the intravascular volume has been replaced. The use of loop diuretics, such as furosemide, or combinations of a loop diuretic and a thiazide agent, may enhance urine output. Nevertheless, if hyperkalemia, refractory acidosis, hypervolemia, or altered mental status associated with uremia occurs, dialysis or hemofiltration should be initiated.

COMPLICATIONS

Shock results in impairment of tissue perfusion and oxygenation and activation of inflammation and cytokine pathways. The major complication of shock is multiple organ system failure, defined as the dysfunction of more than one organ, including respiratory failure, renal failure, liver dysfunction, coagulation abnormalities, or cerebral dysfunction. Patients with shock and multiple organ failure have a higher mortality rate and, for survivors, a longer hospital stay.

PROGNOSIS

Early recognition and **goal-directed intervention** in patients with shock improves survival. Delays in treatment of hypotension, however, increase the incidence of multiple organ failure and mortality.

PREVENTION

Prevention strategies for shock are focused, for the most part, on shock associated with causative exposures (infection, trauma, and ingestion). Some forms of septic shock can be prevented through the use of immunizations (such as *Haemophilus influenzae* type b, meningococcal, pneumococcal vaccines). Decreasing the risk of sepsis in a hospitalized patient requires adherence to strict handwashing, isolation practices, and minimizing the duration of indwelling devices. Measures to decrease pediatric trauma do much to minimize hemorrhage-induced hypovolemic shock. Preventative health measures surrounding environmental toxins (carbon monoxide) and medication storage/administration can aid in the prevention of some forms of dissociative, distributive, and cardiogenic shock (see Table 40.1).

PEARLS FOR PRACTITIONERS

See *The Acutely Ill or Injured Child: Pearls for Practitioners* at the end of this section.