Pediatric Patients with cardiopulmonary compromise require immediate and effective therapy to prevent hypoxic-ischemic organ damage. Treatment should be in accordance with the guidelines of the American Academy of Pediatrics and the American Heart Association. One of the most important priorities during cardiopulmonary resuscitation (CPR) is to have an identified “leader” with the most experience with critically ill children. The leader’s role is to direct the resuscitation and assign specific roles to the members of the resuscitation team. The initial approach to pediatric CPR is the “ABCDE” system (Box 38-1). This provides a systematic way to approach the patient. During the resuscitation, it is often helpful to occasionally restart at ‘A’ to assess the effects of prior interventions.

A (Airway) and B (Breathing)

Proper airway positioning is important to maximize oxygenation and ventilation. In suspected cervical spine injury, a chin lift or jaw thrust maneuver (without distracting the neck) may help relieve upper-airway obstruction caused by oropharyngeal tissues. A nasopharyngeal airway may provide a patent airway in conscious and unconscious patients but is relatively contraindicated in facial or head trauma. An oropharyngeal airway is usually tolerated only by unconscious patients. Suction equipment should always be available in order to remove secretions, vomitus, or other foreign debris that contribute to airway obstruction. Tracheal intubation is indicated for respiratory failure and protection against pulmonary aspiration.

Anesthesiologists are frequently required to deliver pediatric critical care in the perioperative setting. Familiarity with life-threatening pediatric medical conditions, invasive access and monitoring techniques, and pharmacology of medications that affect the cardiovascular system is essential. This chapter provides an overview of the most important aspects of pediatric critical care for the general anesthesiologist.
C (Circulation and Cervical Spine)

Cardiac arrest is the absence of pulses and a perfusing rhythm in the area of the large arteries (brachial, carotid, or femoral). Chest compressions should immediately be initiated. In infants under 12 months old, the hands encircle the chest, and the thumbs are placed one finger-width below the intermammary line to compress the chest evenly over the mid-sternum (Fig. 38-1). The sternum should be depressed one-third to one-half the depth of the infant’s chest at a rate of at least 100 times per minute. Chest compressions should not be administered over the lower third of the infant’s sternum to avoid abdominal trauma. For children aged between 1 and 8 years, the heel of the hand is placed two-finger breaths above the lower end of the sternum. The chest compression to ventilation ratio should be 5:1 (a compression rate of 100 times per minute). Maintaining the frequency and depth of the compressions are the most important elements for the provision of life-sustaining circulation.

Vascular access is critical in pediatric CPR. Peripheral venous access should be attempted, but if it unsuccessful after three attempts or 90 seconds, whichever comes first, intraosseous (IO) vascular access is recommended and now includes all children regardless of age (see Chapter 36 for methods of securing IO access). Once vascular access is established, isotonic fluids such as Ringer’s Lactate or normal saline 10–20 mL/kg should be rapidly administered in every pediatric arrest unless caused by myocardial dysfunction (e.g., myocarditis). Children with hemorrhagic shock should immediately receive un-crossmatched type O Rh-negative packed red blood cells or whole blood. Type-specific blood is recommended if the patient’s blood type is known.

Medications that can be administered through the endotracheal tube include lidocaine, atropine, naloxone, and epinephrine (mnemonic = LANE).

Pulseless electrical activity (PEA) is most commonly caused by mechanical obstruction to blood flow; however, the differential diagnosis should be reviewed quickly. Remember the 4H’s and 4T’s:

Hypoxemia
Hypovolemia
Hypothermia
Hyper/Hypokalemia

and

pericardial Tamponade
Tension pneumothorax
Thromboembolism
Toxins.

Specific therapies should be directed at these causes if spontaneous circulation does not return.

In addition to Circulation, the “C” in “ABCDE” reminds us of cervical spine immobilization, especially if there is a suspected traumatic mechanism of injury. Cervical spinal cord trauma may cause neurologic deficits and neurogenic circulatory shock, which is characterized by hypotension without tachycardia.

D (Disability and Drugs)

Disability

This reminds us to quickly assess the child’s neurologic status. This involves calculating a Glasgow Coma Scale score (see Chapter 36), assessing focal neurologic injury, and examining for pupillary responses that suggest impending cerebral herniation (e.g., unilateral dilated pupil). Glucose measurement should always be checked during a pediatric arrest since hypoglycemia can lead to additional neurologic injury. Hypoglycemia is treated using any immediately available intravenous glucose solution.

Drugs

The most important drug in a pediatric arrest is epinephrine. The recommended initial resuscitation dose of epinephrine for pediatric CPR is 0.01 mg/kg via the intravenous (IV) or IO route, or 0.1 mg/kg by the tracheal route. Repeated doses are recommended every 3–5 minutes for ongoing arrest. The same dose of epinephrine is recommended for subsequent doses, but higher doses (0.1–0.2 mg/kg) may be beneficial.

E (Exposure, Environment, Electrical Activity of the Heart, and Extracorporeal Membrane Oxygenation)

Exposure and Environment

The patient should be fully exposed to identify all injuries and to remove potentially contaminated clothing (e.g., toxic chemicals). Temperature should be closely observed (environment). Hyperthermia should be aggressively treated, especially in patients with head injury or reduced cardiac output.

Electrical Activity of the Heart

This should also be assessed during Circulation of the “ABCDE” system. Anesthesiologists should be familiar with, or have immediate access to, the published algorithms for treating pediatric arrhythmias.

Extracorporeal Membrane Oxygenation

In certain tertiary-care pediatric institutions, some patients who sustained a witnessed cardiopulmonary arrest have been successfully resuscitated with extracorporeal membrane oxygenation (ECMO) when traditional treatments have been unsuccessful. However, ECMO has its own potential complications and further evaluation of its utility for pediatric arrest is ongoing.
COMMON PEDIATRIC CRITICAL ILLNESSES

Status Asthmaticus

Status asthmaticus is a state of severe, persistent, life-threatening bronchospasm that is resistant to standard bronchodilator therapies. Clinical features include cyanosis, absent breath sounds, poor respiratory effort, fatigue, agitation, and depressed consciousness. As airway obstruction worsens, work of breathing increases. The combination of small and large airway collapse leads to air trapping and hyperinflation. In turn, hyperinflation leads to decreased lung and chest wall compliance and impaired diaphragmatic contraction since flattening places it at a mechanically disadvantageous fiber length. As the volume of the lungs increases, the vital capacity decreases and the work of breathing increases further. The increased airway resistance causes the respiratory muscles to perform increased pressure/volume work, which leads to increased CO₂ production, further stressing the respiratory system.

Air trapping is accompanied by ventilation/perfusion mismatch that contributes to increased deadspace and a compensatory increase in minute ventilation. Hypocapnia, respiratory alkalosis, and hypoxemia are seen early in uncomplicated acute asthma. A normal or elevated PaCO₄ raises concern of respiratory muscle fatigue and the inability to maintain adequate ventilation. Hypercapnia normally occurs when the forced expiratory volume at 1 second (FEV₁) falls below 20% of the predicted value, signifying severe obstructive disease.

Cardiovascular effects of severe asthma relate to the dramatic swings in intrathoracic pressure during respiration. Pulsus paradoxus, defined as a >10–12 mmHg difference in systolic blood pressure between inspiration and expiration, is a marker for severity of airway obstruction and increased respiratory muscle work. Assuming pleural pressures are transmitted to the pericardial space, large negative intrathoracic pressures during inspiration increase ventricular afterload, and decrease systemic blood pressure. This is exacerbated by intravascular depletion caused by decreased fluid intake, increased insensible respiratory losses, and an increased metabolic rate.

Treatment of status asthmaticus consists of reducing airway obstruction while maximizing oxygenation and ventilation. Beta-adrenergic agonists reverse bronchiolar smooth muscle contraction and remain the primary treatment modality, but they are associated with adverse effects (Box 38-2). Long-acting β-agonists (e.g., salmeterol) are not indicated during status asthmaticus, and orally administered β-agonists are also ineffective.

In the past, initial therapy for severe bronchospasm consisted of subcutaneous injections of epinephrine. This has been replaced with equally effective inhaled bronchodilators (Table 38-1). Albuterol is most commonly used and can be administered continuously to a child in status asthmaticus. Parenteral β-agonists (e.g., terbutaline) are administered when there is no improvement with continuous nebulized therapy. Subcutaneous administration, as a first-line therapy, is reserved for children with minimal air entry or depressed mental status.

Combination therapy using additional bronchodilators is also available (Table 38-2). Anticholinergic agents (e.g., ipratropium bromide) block the vagal tone of airway smooth muscle with an effect predominantly on M3 receptors; this leads to bronchodilation and may decrease mucous secretion. These drugs have a slow onset of action and do not possess anti-inflammatory action. In severe exacerbations of asthma, the combination of an anticholinergic agent with a β-agonist is significantly more effective than either drug alone; the anticholinergic agent will have a longer duration of action.

Corticosteroids should be started early in any asthma exacerbation. They decrease inflammation, decrease microvascular permeability, and potentiate the response of β₂ receptors to β-agonists. Intravenous corticosteroids are more effective than inhaled steroids.

Magnesium sulfate is increasingly used to treat moderate to severe asthma exacerbations. It is thought to relax bronchiolar smooth muscle by antagonizing calcium or interfering with acetylcholine release at the neuromuscular junction. Dose-related side-effects of magnesium therapy include hypotension, flushing, hyporeflexia, and sedation.

Other therapies exist that are used infrequently or remain unproven. Theophylline has become a third-line therapy. Its mechanism of action is uncertain; it is thought to weakly decrease airway smooth muscle tone, increase central respiratory drive, and enhance diaphragmatic contractility, and it may possess anti-inflammatory or immunomodulator properties. Theophylline possesses a narrow therapeutic range and its use is complicated by a variety of drug interactions. Its side-effects include nausea, vomiting, tachycardia, arrhythmias, and seizures.

Tracheal intubation and mechanical ventilation is reserved for the child with status asthmaticus who develops life-threatening respiratory failure. The absolute indications for instituting mechanical ventilation are a respiratory or circulatory arrest. The relative indications include acidemia, hypercapnia, silent chest, or decreased level of consciousness. The goals of mechanical ventilation are to avoid barotrauma, maximize the expiratory time, and limit the static (plateau) airway pressure to <35 cmH₂O. Eucapnia is not an appropriate objective; the present authors’ utilize a strategy of permissive hypercapnia while maintaining a serum pH above 7.25. Tracheal intubation may aggravate bronchospasm,
and positive-pressure ventilation increases the risk of air leak and circulatory collapse. With the institution of mechanical ventilation, the addition of an inhalational anesthetic agent may decrease bronchospasm.

### Status Epilepticus

Status epilepticus is continuous seizure activity lasting for more than approximately 30 minutes. Pediatric status epilepticus is caused by epilepsy, head trauma, hypoxic-ischemic encephalopathy, infection (e.g., meningitis, encephalitis), medication toxicity, genetic or metabolic disease, or electrolyte imbalance (e.g., hypoglycemia, hyponatremia). However, the etiology is unknown in approximately 50% of cases.

Status epilepticus is a medical emergency, because if left untreated it entails a risk of permanent neurologic damage or death. Hypoventilation and pulmonary aspiration may occur during the seizure or post-ictal period, or as a result of the sedating effects of the treatment drugs. Management priorities include preventing secondary organ damage (especially to the brain), terminating seizure activity, and determining the etiology. Immediate treatment consists of ensuring upper-airway patency, administration of supplemental oxygen, and possibly endotracheal intubation. Use of a neuromuscular blocker to facilitate endotracheal intubation will temporarily mask recognition of continued seizure activity, so only short- or intermediate-acting agents should be used. Hypotension from prolonged seizure activity or cardiodepressant effects of anticonvulsant drugs should be aggressively treated with volume expansion and vasopressors, if necessary. Persistent hypoxemia, hypotension, hypoglycemia, and metabolic acidosis all contribute to additional neuronal damage. Dextrose should be administered if the serum glucose is below 60 mg/dL. Fever should be aggressively treated since an elevated temperature will lower the seizure threshold and increase cerebral oxygen consumption.

Pharmacologic management is aimed at stopping the seizure and preventing recurrence (Fig. 38-2). The longer status epilepticus persists, the harder it is to control. The most common anticonvulsants used during initial treatment are benzodiazepines, phenytoin, and phenobarbital.

Intravenous lorazepam, midazolam, and diazepam are the first-line anticonvulsants in status epilepticus because of their efficacy and rapid onset of action. Lorazepam is usually preferred because of its relatively long duration of action. If vascular or intravenous access is not rapidly obtained, intramuscular, sublingual, or rectal administration is also effective. Midazolam is preferred for intramuscular or rectal treatment. Benzodiazepines are not suited for long-term seizure control, but midazolam infusions have been used as a temporizing measure in the pediatric intensive care unit.

Phenytoin (Dilantin) is a second-line anticonvulsant because of its slower onset of action (peak effect 10–30 minutes) owing to its low lipid solubility. Phenytoin is associated with less sedation and respiratory depression than phenobarbital (see below). An intravenous loading dose of 15–20 mg/kg should be administered slowly (0.5–1 mg/kg/min for children and up to 50 mg/min for adults) because of potential adverse cardiovascular effects such as bradycardia, conduction abnormalities, and hypotension. It should be infused in saline since it is incompatible with dextrose-containing fluids. Close monitoring of the electrocardiograph (EKG) and blood pressure should be performed during administration. Because of the cardiovascular effects, phenytoin should be avoided in children with congenital heart disease or arrhythmias. Manifestations of phenytoin toxicity include diplopia, nystagmus, ataxia, and slurred speech.

Phenobarbital is a third-line anticonvulsant because of its delayed onset of action. The intravenous loading dose is 20 mg/kg infused slowly. Side-effects include respiratory depression (especially when used in combination with benzodiazepines), hypotension, and prolonged sedation.

Status epilepticus that remains unresponsive to conventional therapies should be aggressively managed with adjunct therapies. Pentobarbital, a long-acting barbiturate, can be administered using a loading dose of 5–15 mg/kg, followed by an infusion of 1–2 mg/kg/h. The goal is to obtain burst suppression on electroencephalography (EEG). Side-effects include cardiorespiratory depression (that often requires inotropic therapy) and granulocytopenia. As a last resort, inhalational general anesthetics may be required for neuronal suppression.

Following control of seizures, attention is turned toward determining the etiology. This evaluation includes investigation of metabolic, infectious, and toxic causes. Hyponatremia should be ruled out. A lumbar puncture with an opening pressure measurement may be indicated to rule out meningitis or meningoencephalitis. However, a lumbar puncture should be delayed in the presence of focal neurologic signs, increased intracranial pressure, cardiorespiratory depression, or coagulopathy. The delay in performing a lumbar puncture should not delay administration of broad-spectrum antibiotics or antiviral therapy if an infection is suspected. Head CT imaging should be obtained if there is a history of trauma, signs of elevated intracranial pressure, or presence of focal neurologic signs.

### Shock

Shock occurs when there is insufficient delivery of oxygen and nutrients to meet the metabolic demands of the body. Shock can exist even if the blood pressure is normal. It is helpful to consider three progressive stages of shock: compensated, uncompensated, and irreversible.
Early compensated shock is notable for the activation of mechanisms that maintain a normal blood pressure and organ perfusion. This is usually accomplished by increasing the heart rate and the systemic vascular resistance (SVR). Neonates and young infants are unable to significantly increase their stroke volume, so tachycardia is the only way to augment cardiac output (cardiac output = stroke volume × heart rate). Peripheral vasoconstriction increases the diastolic pressure and narrows the pulse pressure (systolic BP − diastolic BP). The physical exam will reveal cool, pale extremities with delayed capillary refill (>4 seconds), and decreased urine output. In the compensated stage, circulatory compensation fails and leads to cellular dysfunction, ischemia, and endothelial injury. Hypoperfusion of the brain leads to an altered level of consciousness and renal hypoperfusion leads to anuria. The child develops tachypnea to compensate for the metabolic acidosis caused by hypoperfusion and decreased oxygen delivery. Bradycardia is a worrisome sign in an infant because it significantly decreases cardiac output. Hypotension is a late finding in pediatric shock and places the patient at risk for multiorgan system failure (Table 38-5). Irreversible shock occurs when there is unrecoverable end-organ damage.

Shock is classified into four major categories: hypovolemic, septic, distributive, and cardiogenic. **Hypovolemic shock** is the most common cause of shock in children. It results from decreased intravascular volume, which leads to decreased venous return and preload. This can be caused by water and electrolyte loss (e.g., vomiting, diarrhea, renal losses, or heat stroke), hemorrhage, or plasma losses (e.g., burns, nephrotic syndrome). Initial management is focused on airway, breathing, and circulation (Fig. 38-3). Fluid resuscitation is guided by heart rate, peripheral perfusion, and urine output. Isotonic fluids such as Lactated Ringers or normal saline are used during the initial volume replacement. With blood loss, the “3 to 1 rule” applies: for every 1 mL of blood loss, 3 mL of isotonic fluids should be administered since only one-third of the volume remains in the intravascular space. Patients with severe liver disease may be unable to metabolize the lactate in Lactated Ringers solution. Development of a hyperchloremic metabolic acidosis is common and can start when a 1.5–2 blood volume loss is replaced with crystalloid and packed red blood cells alone.

**Septic shock** occurs due to overwhelming infection. It is a combination of hypovolemia, altered vascular tone, cardiac pump failure, and cellular metabolic derangements that result in metabolic acidosis. In the initial stages, the extremities can be warm and well perfused or cold with delayed capillary refill. Vasodilation is caused by inflammatory mediators (e.g., endotoxin, tumor necrosis factor, and interleukin-1), and is manifested clinically as diastolic hypotension and a wide pulse pressure. If untreated, persistent shock will lead to multiorgan system failure. These patients may require massive administration of isotonic fluids (>60 mL/kg) and vasopressors. Further management includes continuous assessment and treatment of end-organ perfusion abnormalities: oliguria, mental status changes, metabolic acidosis, decreased cardiac output, coagulopathy, and disseminated intravascular coagulation (DIC).

**Distributive shock** is caused by abnormalities in vasomotor tone. Vasodilation and peripheral intravascular pooling cause relative hypovolemia. Neurogenic shock caused by spinal cord injury, usually at the cervicothoracic area, is a classic example. Loss of sympathetic vascular tone causes bradyarrhythmias, vasodilation, and hypotension. Another example is anaphylaxis. In distributive shock, Trendelenburg positioning (head down with cervical spine immobilized if indicated) and administration of isotonic fluids can be helpful to restore circulatory stability. Vasopressor drugs with direct α1-adrenergic activity may be necessary.

**Cardiogenic shock** can result from congenital heart disease, trauma, prolonged arrhythmias (e.g., supraventricular tachycardia), cardiac tamponade, infection (e.g., myocarditis), acquired cardiomyopathies (Kawasaki’s disease with coronary aneurysms), and drug intoxications. Central venous pressure monitoring and physical examination will guide cautious fluid administration. These patients will usually benefit from inotropic support and afterload reduction.

Special consideration should be given to neonates presenting in shock. They may have undiagnosed congenital heart disease that has ductal-dependent systemic circulation. These babies are asymptomatic at birth because aortic blood flow is provided by the patent ductus arteriosus (PDA). However, when the PDA closes, shock develops from inadequate systemic output. These patients usually have left-sided obstructive lesions, for example hypoplastic left heart syndrome (HLHS), aortic valve stenosis, interrupted aortic arch, or coarctation of the aorta. Prostaglandin E1 (0.05–0.1 µg/kg/min) should be started in an attempt to open the ductus arteriosus until a definitive surgical procedure can be performed.
Inotropic Agents

Dopamine

Dopamine is the metabolic precursor of norepinephrine and epinephrine. It is a central neurotransmitter that is also found peripherally in the sympathetic nervous system and in the adrenal medulla. Depending on the local concentration, dopamine produces vascular dilation or constriction by stimulation of dopaminergic, α, and β receptors in the brain and peripheral vascular beds.

Dopamine is used to treat hypotension and oliguria in children with distributive, septic, or cardiogenic shock when volume resuscitation has been ineffective. Low infusion rates (1–5 µg/kg/min) stimulate dopamine receptors and are associated with increased glomerular filtration and renal blood flow. Intermediate doses (5–10 µg/kg/min) are associated with increased heart rate and improved myocardial contractility via β-receptor stimulation and release of norepinephrine from nerve terminals. Administration of dopamine in the intermediate dosing range usually results in an increase in systolic blood pressure and minimal change in diastolic pressure. SVR is unchanged; however, a higher infusion dose (>10 µg/kg/min) is associated with an increase in SVR secondary to α-receptor stimulation.

Adverse effects of dopamine include tachycardia, hypertension, and dysrhythmias - and thus, increased myocardial oxygen consumption. Administration of dopamine is associated with decreases in PaO₂ by inhibition of hypoxic vasoconstriction, and dopamine depresses the ventilatory response to hypoxemia by as much as 60%.

The clearance of dopamine decreases with age throughout childhood. During the first 20 months of life, dopamine clearance decreases by almost 50%. All studies of dopamine pharmacokinetics in seriously ill children show substantial interindividual variation in pharmacokinetic parameters. This is especially true for clearance, which appears to be not only age- but also concentration-dependent. This causes a large variation in dose requirements required to achieve a desired clinical response.

Dobutamine

Dobutamine resembles dopamine structurally but has greater selectivity for β₁ and β₂ receptors. When administered in a dose range of 5–20 µg/kg/min, it primarily enhances myocardial contractility and increases stroke volume, with less increase in heart rate than dopamine. Its administration is associated with decreases in SVR and PVR, such that hypotension may occur from vasodilation in children who are volume depleted or who have an elevated baseline sympathetic imbalance.

Dobutamine is indicated in the treatment of cardiac decompensation, as may occur after surgery for congenital heart disease, or in children with congestive heart failure or myocarditis. Dobutamine may be used to treat myocardial dysfunction associated with sepsis, but it is rarely the sole inotropic agent. Because of its inotropic properties, dobutamine increases myocardial oxygen demand, and may predispose to arrhythmias.

Ephedrine

Ephedrine possesses α- and β-agonist activity but primarily acts indirectly by enhancing the release of norepinephrine from sympathetic neurons. Heart rate and cardiac output are increased with a variable increase in SVR. Ephedrine is used intraoperatively (0.2–0.3 mg/kg/dose) to treat hypotension related to administration of general or regional anesthesia.

Epinephrine

Epinephrine is the principal hormone involved in the normal stress response and produces widespread metabolic and hemodynamic effects. It activates α, β₁, and β₂ receptors, and is useful in treating shock associated with myocardial dysfunction and hypotension. During epinephrine infusions, hepatic and splanchnic blood flow increases, while renal blood flow may be reduced. In addition, administration of epinephrine results in bronchial and gastrointestinal smooth muscle relaxation.

At relatively low doses (0.05–0.1 µg/kg/min), epinephrine stimulates β₂ receptors. Therefore, one of the earliest effects is an increase in heart rate and inotropy. At these doses, stimulation of β₂ receptors promotes relaxation of resistance arterioles, promoting a decrease in SVR and a decreased diastolic blood pressure. Higher plasma concentrations activate α receptors, with a subsequent increase in SVR. High infusion doses (1–2 µg/kg/min) are associated with significant vasoconstriction, and possible compromise of blood flow to individual organs. Plasma concentrations of epinephrine correlate with the infusion rate, suggesting linear pharmacokinetics. Epinephrine clearance rates in critically ill children appear to be lower than the reported clearance rates in healthy adults.

In the pediatric critical care environment, the most frequent indications for intravenous epinephrine are cardiogenic shock and septic shock with reduced stroke volume. The septic patient who does not improve after aggressive volume repletion and treatment with dopamine and/or dobutamine may benefit from epinephrine. Epinephrine boluses are used to treat asystole and other dysrhythmias that cause hypotension. The recommended dose is 0.01 mg/kg (0.1 mL/kg of the 1:10,000 preparation). Epinephrine is the treatment of choice for signs and symptoms of anaphylaxis and may occasionally be used in the treatment of severe bronchospasm.
chronotropy in the face of decreased SVR results in an increase in inotropy and chronotropy in the face of decreased SVR results in an increase in cardiac output. Pulmonary bronchial and vascular bed \( \beta_2 \)-adrenergic receptor agonism results in bronchodilation and pulmonary vasodilation.

Isoproterenol currently has limited clinical applicability in children. It may be used to treat hemodynamically significant bradycardia, and has been used in infants after cardiac surgery to improve cardiac index. Isoproterenol has also been used as an adjunct to therapy for children with status asthmaticus, but it is being replaced with continuous nebulized albuterol and intravenous terbutaline.

Norepinephrine

Norepinephrine is an endogenous catecholamine with potent \( \alpha \) and \( \beta_1 \) activity and little \( \beta_2 \) activity. Infusions in normal subjects result in elevations of SVR because \( \alpha \) effects are not opposed by \( \beta_2 \) stimulation. Reflex vagal activity reduces the rate of sinus node discharge, blunting the expected chronotropic effect of \( \beta_1 \) stimulation. Stroke volume increases, but there is minimal change in cardiac output. Resistance increases in most vascular beds, including the kidney, liver, and skeletal muscle. Glomerular filtration is maintained unless renal blood flow is decreased substantially. Coronary blood flow increases, due to direct coronary dilation and an increase in blood pressure.

Norepinephrine improves perfusion in children with hypotension and a normal or elevated cardiac index that has not responded favorably to volume resuscitation. Treatment with norepinephrine is beneficial in the setting of tachycardia, because it can increase SVR, arterial blood pressure, and urine flow without an increase in heart rate. The usual starting infusion dose is 0.05–0.1 \( \mu \)g/kg/min, which is then titrated to the desired effect. Norepinephrine administration may improve blood pressure without improving perfusion. This is most commonly seen in children with a low cardiac index and stroke volume.

Phenylephrine

Phenylephrine is predominantly an \( \alpha \)-adrenergic agonist and is used to treat hypotension when SVR is low. By vasoconstricting arteriolar beds, phenylephrine increases blood pressure and causes a vagally mediated sinus bradycardia. There are few data available on its use in pediatric patients and it is rarely used intraoperatively because children do not usually exhibit hypotension related to regional anesthesia.

Milrinone

Milrinone is a phosphodiesterase inhibitor that produces inotropy (without tachycardia) and vasodilatation by increasing concentrations of intracellular cyclic AMP. Milrinone has been used in children for the treatment of low-output states following cardiac surgery and in the management of shock when catecholamine infusions alone are unsuccessful. Pharmacokinetic parameters of milrinone are different in children from adults, reflecting a higher volume of distribution and more rapid clearance rate. An initial intravenous loading dose of 50–75 \( \mu \)g/kg over 15–60 minutes is followed by a continuous infusion of 0.375–0.75 \( \mu \)g/kg/min, which is then titrated to effect. Administration of milrinone may occasionally cause atrial or ventricular arrhythmias.

Intracranial Hypertension

Intracranial hypertension, defined as an intracranial pressure (ICP) above 20 mmHg persisting for more than 5 minutes, is most commonly due to pediatric traumatic brain injury. No intervention can reverse the primary brain injury; supportive measures are aimed at preventing secondary injury or damage to surrounding neurons.

The components of the rigid cranial vault include brain tissue, cerebrospinal fluid (CSF), and blood. For the ICP to remain normal, alterations in the volume of one compartment must be compensated for by opposite changes in another compartment (the Monro–Kellie doctrine). For example, an epidural blood collection can initially increase without affecting ICP because of a compensatory decrease in CSF volume. However, once these compensatory measures have been maximized, the ICP will rise dramatically with a small increase in blood volume (Fig. 38-4). Clinically, this causes a Cushing’s reflex that consists of hypertension, bradycardia, and abnormal respirations.

Autoregulation refers to the brain’s ability to maintain cerebral blood flow (CBF) despite changes in blood pressure. In the uninjured brain, CBF remains relatively constant within mean arterial pressures between 50 and 150 mmHg. In the traumatized brain, this ability may be lost and CBF may increase or decrease as the blood pressure increases or decreases, respectively. CBF remains constant when the \( P_{aO_2} \) is above 60 mmHg, but increases dramatically with hypoxemia. A linear relationship exists
between CBF and \( P_{aCO_2} \) - blood flow increases as carbon dioxide increases (Fig. 38-5). Furthermore, CBF is closely linked to the cerebral metabolic rate of oxygen consumption (CMR-O\(_2\)). It will increase if CMR-O\(_2\) increases due to seizures, pain, or agitation, and decrease as CMR-O\(_2\) decreases due to hypothermia or sedation.

The major goal in the management of intracranial hypertension is to ensure adequate oxygen and substrate delivery to the brain. This is achieved by ensuring adequate oxygenation and ventilation, lowering the intracranial pressure, and maintaining an adequate cerebral perfusion pressure (Fig. 38-6). The cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and ICP (or central venous pressure (CVP), whichever is greater). In adolescents and adults, CPP greater than 70 mmHg suggests adequate CBF; while in infants, an acceptable CPP is one greater than 50 mmHg. Intracranial pressure can be monitored using extradural, intraparenchymal, or intraventricular catheters. Intraventricular catheters have the additional benefit of providing a therapeutic option of CSF drainage to lower ICP.

Tracheal intubation is indicated if airway protective reflexes are absent or if there is a depressed level of consciousness (Glasgow Coma Scale ≤8). Short-term hyperventilation is one of the most potent therapies to lower elevated ICP. However, prolonged hyperventilation is associated with worsening of cerebral ischemia and therefore is not recommended. Recent studies indicate that positive end-expiratory pressure (PEEP) does not increase intracranial pressure. Therefore, PEEP should be used to maintain adequate oxygenation and prevent alveolar collapse. The current cardiovascular management strategy consists of volume resuscitation and transient periods of hypertension to maintain CPP. Hypotonic solutions will exacerbate cerebral edema.

Additional principles of ICP management include avoidance of hyperthermia, prevention of venous outflow obstruction, normoglycemia, and seizure prophylaxis if indicated. Hyperthermia increases CMR-O\(_2\) and may cause a mismatch between demand and supply. The head should be elevated and maintained in the midline position to avoid jugular venous compression. Cervical collars and endotracheal tube ties should not be too tight. Hyperglycemia will exacerbate neuronal injury and therefore should be aggressively treated with insulin. If an intraventricular catheter is in place, CSF drainage can be therapeutic.

Hyperosmolar therapy continues to be an important component of ICP management. Mannitol (0.25–1 g/kg IV infused over 10–30 minutes) creates an osmotic gradient that draws extracellular brain water across the blood-brain barrier into the intravascular space. It is also thought to decrease blood viscosity, thereby enhancing oxygen delivery. The prophylactic use of mannitol is not recommended owing to its volume-depleting diuretic effect. It should be used in patients demonstrating signs of transtentorial herniation. Hypertonic saline (3% NaCl) is being used more frequently to control ICP. It is thought to reduce cerebral swelling by producing an osmolar gradient to decrease cerebral water content. Continuous infusion of 3% NaCl through a central line will prevent swings in osmolality and maintain intravascular volume. It is not unusual to have serum sodium concentrations between 150 and 160 mEq/L. Side-effects of hyperosmolar therapy include renal failure (especially if the serum osmolality is >320 mEq/L), hemolysis, and subarachnoid hemorrhage related to tearing of bridging vessels due to rapid brain shrinkage. Rapid increases in serum sodium are also associated with development of central pontine myelinolysis.

Barbiturate coma (burst suppression on EEG) is indicated when intracranial hypertension is not controlled with conventional therapies. It lowers ICP by lowering CMR-O\(_2\), which then results in cerebral vasoconstriction and a reduction in cerebral blood flow and volume. Barbiturates produce respiratory depression and a dose-dependent decrease in arterial blood pressure and cardiac output that often requires significant volume resuscitation and inotropic therapy. When the above measures fail, a measure of last resort is a decompressive craniectomy.

**Hepatic Failure**

Acute or fulminant hepatic failure is a clinical syndrome that results from massive necrosis of liver cells leading to the development of hepatic encephalopathy and severe impairment of hepatic function. Hepatic failure can occur as a primary process in a healthy person, as an exacerbation of a chronic liver disease, or as part of multiorgan system failure. Several authors have suggested redefining the syndrome into three categories based on the time interval between the onset of jaundice and encephalopathy: hyperacute (0–7 days), acute (1–4 weeks), and subacute (4–12 weeks). Causes of fulminant hepatic failure in children include viral hepatitis, drug-induced liver injury, and inherited metabolic diseases, among others.

The diagnosis of acute hepatic failure is based on clinical findings (jaundice, enlarged liver; and encephalopathy) in conjunction with abnormal biochemical data (low serum albumin, hyperbilirubinemia, and prolongation of the prothrombin time). There is no specific therapy for acute hepatic injury, except early administration of N-acetylcysteine in acetaminophen toxicity.

Fulminant hepatic failure can adversely affect all organ systems. Hepatic encephalopathy may result from diminished hepatic synthesis of an unknown substance(s) needed for normal brain function or diminished hepatic
metabolism of an unknown substance(s) that possesses neurotoxicity or promote neural inhibition. As liver function deteriorates, hepatic encephalopathy worsens (Table 38-5). Serum ammonia levels do not correlate with the severity of encephalopathy. Advanced encephalopathy is associated with severe cerebral edema and life-threatening cerebral herniation.

Acute renal failure develops in 30–50% of patients with acute liver failure (hepatorenal syndrome). Contributing factors include intravascular volume depletion caused by excessive diuresis or untreated gastrointestinal hemorrhage, and nephrotoxic drug administration.

Hemorrhagic complications in liver failure result from inadequate synthesis of clotting factors, thrombocytopenia from DIC, and splenic sequestration of platelets. Blood loss may occur from stress-induced gastritis or from esophageal or gastric varices that develop secondary to portal hypertension. Vitamin K administration augments production of factors II, VII, IX, and X. Fresh frozen plasma and/or cryoprecipitate is administered when patients are bleeding or prior to an invasive procedure.

Hypoxemia can develop in acute liver failure for several reasons. Failure of the damaged liver to clear vasodilating humoral substances can cause intrapulmonary shunting and ventilation/perfusion mismatch. Pulmonary edema results from low oncotic pressure and fluid overload associated with anti-diuretic-like activity. Atelectasis can develop from massive ascites that impedes full respiratory excursion or hypoventilation caused by encephalopathy. Ascites develops as a result of increased hepatic vascular resistance, decreased oncotic pressure, and altered aldosterone secretion.

Patients with chronic liver failure develop a high cardiac output in conjunction with a low SVR. Oxygen delivery is increased, but oxygen consumption is decreased due to microcirculatory disturbances that lead to tissue hypoxia.

Orthotopic liver transplantation is the most important advance in the therapy of acute liver failure. There is no clear set of indications for liver transplantation in pediatric patients, but without hepatic transplantation, acute hepatic failure carries a grim prognosis with an 80–85% mortality rate. Death is usually caused by cerebral herniation from intracranial hypertension.

**Renal Failure**

Acute renal failure is the sudden inability of the kidney to regulate fluid, electrolyte, and solute balance; this can occur with or without a change in urine volume. Causes of acute renal failure can be categorized into three major categories: prerenal, intrinsic renal parenchymal damage, and postrenal (Table 38-6).

Prerenal renal failure is caused by inadequate renal perfusion from systemic hypovolemia, poor cardiac output, or vascular obstruction. Drugs can also have an adverse effect on renal perfusion: nonsteroidal anti-inflammatory drugs (NSAIDs) promote renal vasoconstriction, and angiotensin-converting (ACE) inhibitors reduce glomerular filtration pressure. Intrinsic renal parenchymal damage affects the structures of the nephron. Most intrinsic renal failure is caused by ischemia, nephron damage from toxins, or inflammation. Acute tubular necrosis (ATN) is the most common form of intrinsic renal failure. Postrenal renal failure is due to mechanical obstruction in the urinary collecting system. When the obstruction occurs at the level of the bladder or urethra, renal function is more likely to be severely affected since both kidneys will be injured. Unilateral obstruction does not result in renal failure if the other kidney is healthy.

The diagnosis of acute renal failure requires a stepwise approach. The patient’s history and physical exam can yield important clues in the evaluation. Analysis of urinary sediment can be helpful: white cell casts suggest interstitial nephritis; red cell casts are found in glomerulonephritis; and heme-positive urine without red blood cells suggest myoglobinuria or hemoglobinuria. Analysis of urine osmolality, urine creatinine (Cr) and electrolytes can also aid diagnosis. Calculation of the fractional excretion of sodium ($\text{FE}_{\text{Na}}$) will help distinguish prerenal from renal causes:

$$\text{FE}_{\text{Na}} = \frac{\text{Urine}_{\text{Na}}}{\text{Plasma}_{\text{Na}}} \times \frac{\text{Plasma}_{\text{Cr}}}{\text{Urine}_{\text{Cr}}} \times 100$$

In general, patients with hypovolemia will have concentrated urine: urine osmolality >500 mOsm/kg, urine sodium content <20 mEq/L, and $\text{FE}_{\text{Na}} < 1$. In patients with tubular necrosis, the urine is dilute: urine osmolality <350 mOsm/kg, urine sodium content usually >40 mEq/L, and $\text{FE}_{\text{Na}}$ usually >1.

The management of acute renal failure is aimed at treating the underlying disorder or causative mechanism. Restoring effective circulating volume is one of the most important management issues, especially if volume depletion causes hemodynamic instability. If the patient is euolemic with oliguria, then replacement of fluid losses is indicated. This includes insensible losses calculated at 300 mL/m²/day in addition to measuring and replacing other losses such as urine output, nasogastric tube drainage and diarrhea. If significant volume over-load exists, then fluid restriction to insensible losses or even smaller quantities may be necessary.

Electrolyte and acid-base abnormalities are frequently encountered in renal failure. Hyponatremia is usually caused by water retention. In hospitalized patients with oliguria, the administration of excessive amounts of hypotonic fluids can contribute or worsen hyponatremia. Restriction of free water is indicated if the patient is volume overloaded. When hyponatremia is symptomatic (i.e., seizures and obtundation), it is appropriate to
administer hypertonic saline (approx. 4 mL/kg) to raise the serum sodium to 125 mEq/L. Rapid correction of serum sodium to normal (140 mEq/L) can lead to central pontine myelinolysis and cerebral injury. Hyperphosphatemia develops because of decreased renal phosphate clearance. This is treated with dietary restriction and agents that bind phosphate enterally (e.g., calcium carbonate). A metabolic acidosis develops because of impaired renal excretion of acids and alterations in renal bicarbonate reabsorption and regeneration. In the case of acidosis and severe hypocalcemia, calcium must be replaced first since alkalization will decrease the ionized calcium levels further and may exacerbate symptoms of hypocalcemia such as tetany or cardiovascular electrical abnormalities.

Hyperkalemia (potassium >6.0 mEq/L) is the major life-threatening electrolyte abnormality in acute renal failure and must be aggressively treated. EKG abnormalities include tall, peaked T waves initially, followed by prolongation of the P-R interval and widening of the QRS complex, leading to ventricular fibrillation and cardiac arrest. Therapy involves stabilizing the myocardium, redistributing potassium from the extracellular to intracellular space to decrease serum potassium levels, and enhancing potassium elimination from the body (Table 38-7).

The absolute indications for dialysis in renal failure are: intractable acidosis; hyperkalemia; symptomatic uremia such as pericarditis, bleeding or encephalopathy; volume overload with congestive heart failure, pulmonary edema, or hypertension; and toxins that are dialyzable (e.g., ammonia, salicylates, methanol, ethylene glycol). A relative indication is to provide better nutrition or multiple blood products during periods of oliguria. Peritoneal dialysis, intermittent hemodialysis, or continuous hemofiltration are all viable options, determined in consultation with a pediatric nephrologist.

Chronic renal failure (Box 38-3) is the irreversible deterioration in the glomerular filtration rate (GFR) to a point that renal replacement therapy is necessary to sustain life. This usually occurs with a GFR below 10–20 mL/min/1.73 m².

### Respiratory Failure

Respiratory failure is the inability to maintain normal exchange of oxygen and carbon dioxide between the atmosphere and blood. It occurs more frequently in infants and children because of a developmentally immature respiratory system, greater oxygen consumption and carbon dioxide excretion demands, and a higher frequency of disease processes with primary or secondary respiratory complications.

One schematic used to identify the etiology for respiratory failure divides the respiratory system into three parts: the extrathoracic airway, the lung, and the respiratory pump. Maturational changes in the structure and function of these parts during infancy and early childhood influence the susceptibility to respiratory failure (see Chapter 2). Respiratory failure can be classified into two main patterns based on blood gas abnormalities (Table 38-8). Type I (hypoxemic) respiratory failure results from poor matching of pulmonary ventilation to perfusion, leading to noncardiac mixing of venous blood with arterial blood. It is characterized by arterial hypoxemia with normal or low arterial carbon dioxide. Type II (hypercarbic), respiratory failure results from inadequate alveolar ventilation in relation to physiologic needs and is characterized by hypoxemia and arterial hypercarbia. This occurs when a disease or injury leads to an imbalance between the power available to do the respiratory work and the load on the respiratory system. In general, diseases that affect the anatomic components of the lung will result in regions of low or absent ventilation/perfusion ratios, leading initially to type I respiratory failure. Diseases of the extrathoracic airway and respiratory pump result in a respiratory power/load imbalance and type II respiratory failure.

Arterial hypoxemia is caused by several mechanisms: hypoventilation; ventilation/perfusion (V/Q) mismatch; shunting of systemic venous blood to the systemic arterial circuit; impaired alveolar diffusion of oxygen; inhalation of a hypoxic gas mixture; and abnormal desaturation of systemic venous blood in the presence of other mechanisms for hypoxemia.

Initial treatment of hypoxemia is supplemental oxygen. When lung disease results in significant oxygenation abnormalities (F\textsubscript{1}O\textsubscript{2} ≥ 0.60 required to maintain P\textsubscript{a}O\textsubscript{2} ≥ 60 mmHg), continuous positive airway pressure (CPAP) may be helpful. CPAP, ranging from 3 to 10 cmH\textsubscript{2}O, increases lung volume, and enhances ventilation to areas with low V/Q ratios and improves respiratory mechanics. CPAP is often applied using a tight-fitting mask or nasal cannula. If CPAP ≥10 cmH\textsubscript{2}O does not relieve severe hypoxemia, decrease work of breathing, or fails to resolve hypercarbia with a pH ≤ 7.25, treatment with mechanical ventilation is indicated.

Diseases of the respiratory pump (central nervous system, respiratory muscle, or chest wall) cause type II respiratory failure. Noninvasive and invasive modes of mechanical ventilation can be instituted to decrease the work of breathing and provide adequate gas exchange. Noninvasive mechanical ventilation refers to nasal prongs or facemask. During the last decade, the widespread availability of inspiratory pressure support has made noninvasive mechanical ventilation more successful than in previous eras. Inspiratory pressure support is a ventilator modality where the patient’s effort is boosted by increased circuited pressure during inspiration. This allows the patient to initiate his or her own
breaths and regulate inspiratory time and tidal volume. The pressure support strategy promotes patient synchrony and comfort with mechanical support. Disease severity limits the use of this technique if periodic relief from the interface is unavailable for days. A tracheal tube is necessary and safer under these circumstances.

Conventional modes of mechanical ventilation are designed to assist physiologic respiratory pump function and improve lung volumes. Positive pressure is used to inflate the lungs. The clinician monitors the $P_{aCO_2}$ to evaluate therapeutic adjustments in the minute ventilation. The minute ventilation is adjusted by modifying the frequency or the tidal volume. In turn, tidal volume is adjusted by controlling either the delivered volume (volume-controlled ventilation) or the inspiratory pressure (pressure-controlled ventilation). The duration of inspiration is adjustable but this parameter only indirectly affects minute ventilation through its effect on expiratory time or when high airway resistance is present. Inspiratory time adjustments have a more direct influence on mean airway pressure (MAP), and therefore oxygenation.

**Principles of Pediatric Mechanical Ventilation**

The goals of mechanical ventilation are to improve alveolar ventilation, reduce ventilation/perfusion (V/Q) mismatch, reexpand collapsed lung segments, reduce work of breathing, and eliminate respiratory muscle fatigue. Indications for institution of mechanical ventilation in children include:

1. **Hypoxemic respiratory failure**, defined as inadequate arterial oxygenation with $P_{aO_2} < 50$ mmHg at $F_{O_2} \geq 0.50$. The most common cause includes alveolar diseases such as pneumonia, acute respiratory distress syndrome, or pulmonary edema.

2. **Type II or hypercarbic respiratory failure**, defined as inadequate ventilation with $P_{aCO_2} > 50$ mmHg and an arterial pH $< 7.30$. It can develop with or without hypoxemia. It is caused by respiratory pump failure, which includes abnormalities in the central nervous system, respiratory muscles, or chest wall. Examples include a child with a neuromuscular disease (e.g., spinal muscular atrophy) or hypoventilation (e.g., accidental narcotic overdose).

3. **Circulatory failure** such as shock or congestive heart failure. In this setting, mechanical ventilation can cause a reduction in metabolic expenditure, decrease respiratory muscle dysfunction caused by hypoxia, or decrease respiratory dysfunction associated with shock (i.e., ARDS).


Mechanical ventilation can control arterial $P_{aCO_2}$, which influences cerebral blood volume and intracranial pressure.

5. **Postoperative surgical conditions** that require sedation and/or immobility.

Mechanical ventilation is accomplished using pressure-controlled or volume-controlled ventilation. The fundamental difference between the two modes is the targeted goal; pressure ventilation modes guarantee a certain peak inspired airway pressure at the expense of a variable tidal volume, while volume ventilation guarantees flow and the set volume at the expense of inspiratory pressures.

With pressure-controlled ventilation, the physician determines the targeted inspiratory pressure and the inspiratory time. After the breath is initiated, the targeted pressure is attained early during the breath and is maintained at the airway opening during inspiration. Pressure ventilation has an almost unlimited ability to deliver flow and possesses a decelerating flow profile that tends to improve the distribution of ventilation in a lung with heterogeneous disease. With comparable settings in volume control, pressure-control ventilation will maintain a higher mean airway pressure. A potential disadvantage with this type of ventilation is that tidal volume, and therefore minute ventilation, is variable. The tidal volume generated will depend on the compliance and resistance of the respiratory system. As lung compliance improves, the tidal volume will increase with the same inspiratory pressure. This type of ventilation will compensate for an air leak around an uncuffed endotracheal tube.

With volume-controlled ventilation, a preset tidal volume (the volume of gas to be moved in and out of the lungs) is delivered unless a pressure limit is exceeded. Tidal volume is controlled by a constant inspiratory flow rate and a set inspiratory time. A decrease in lung compliance or increase in airway resistance will be reflected by an increase in peak inspiratory pressures. For example, if an endotracheal tube becomes occluded, the airway resistance will increase, and the same tidal volume will now generate a higher inspiratory pressure. For healthy individuals, a normal tidal volume breath is 6–8 mL/kg. But when initiating volume control ventilation, the prescribed tidal volume is usually 8–10 mL/kg since “extra” volume is needed to compensate for the compressible volume lost in the ventilator circuit. This volume is adjusted based on adequacy of chest rise in each patient.

In children with severe lung disease, such as acute respiratory distress syndrome (ARDS), closing volume is increased above that of the functional residual capacity, causing diffuse atelectasis. By increasing mean airway pressure, lung volumes may be increased above that of the closing volume, leading to alveolar recruitment. This is why some clinicians prefer the use of
pressure-controlled ventilation, which maintains a higher mean airway pressure. Others will use volume-controlled ventilation and increase the mean airway pressure by optimizing positive end-expiratory pressure (PEEP).

PEEP increases the functional residual capacity of the lung (the volume of gas in the lung after normal exhalation) above the closing volume (the volume of gas in the lung after maximal exhalation at which the airways close). PEEP maintains alveolar volume, prevents atelectasis, and improves oxygenation by increasing the mean airway pressure (MAP). PEEP is set in either volume- or pressure-controlled ventilation; it is the lowest expiratory pressure reached during mechanical ventilation. However, excess PEEP may cause lung hyperinflation, air trapping, or air leaks. PEEP has a double effect on the cardiovascular system: it will increase intrathoracic pressure and may decrease systemic venous return to the heart (preload). On the other hand, PEEP improves cardiac output by decreasing afterload of the left ventricle. One of the major goals in mechanical ventilation is to find a balance between the good and bad effects of PEEP in each patient.

Inverse-ratio ventilation and high-frequency ventilation are unconventional modes of mechanical ventilation used in pediatric patients. Inverse-ratio ventilation prolongs the inspiratory phase in excess of the expiratory phase during positive-pressure ventilation; this increases mean airway pressure and oxygenation during severe acute lung disease. It is a nonphysiologic pattern for breathing and therefore these patients are administered heavy sedation and paralysis. Airway pressure-release ventilation (APRV) is a newer form of inverse-ratio ventilation that utilizes a continuous gas flow circuit to allow the patient to breathe spontaneously throughout the ventilatory cycle. This is more comfortable for the patient. Less sedation is needed, so patients can contribute to their ventilation. High-frequency jet ventilation and high-frequency oscillatory ventilation both combine small tidal volumes (smaller than calculated airway dead-space) with increased frequencies (>1 Hz) in order to minimize the effects of elevated peak and mean airway pressures. High-frequency ventilation reduces the occurrence and treatment of air-leak syndromes associated with neonatal and pediatric acute lung injury.

The radial artery is a favored site for arterial cannulation because the vessel is superficial and easily accessible. Other anatomic sites frequently used are the ulnar, dorsalis pedis, posterior tibial, and femoral arteries. In general, the brachial artery should be avoided because of the risk of median nerve damage.

After palpatting and localizing the artery with the nondominant hand, the selected artery can be cannulated either by inserting the catheter directly into the artery using a catheter-over-needle device or by using the Seldinger technique. The Seldinger technique involves entering the vessel with a needle, placing a guidewire through the needle after the vessel is entered, removing the needle, and then placing the catheter over the wire into the vessel. This can be performed for arterial and venous cannulations. When cannulating a peripheral artery, it is helpful to immobilize the extremity with a board. Aseptic technique should always be followed when placing an arterial line.

All arterial lines must be clearly identified to avoid accidental infusion of hypertonic solutions and sclerosing medications that would injure the artery. Arterial catheters are at risk for infection, disconnection causing significant blood loss, and arterial thrombus formation. Also, ischemic distal necrosis can occur from arteriolar spasm or emboli from air or clot.

### Central Venous Catheter Insertion

Central venous catheters are used in pediatric patients for several reasons. They provide cardiac filling pressure measurements. They are also a secure route for:

- Administration of fluids and drugs to the central circulation
- Rapid infusion of large volumes of fluids and blood products
- Administration of high-concentration parenteral alimentation that would be sclerosing to peripheral veins
- Administration of parenteral fluid and drugs when peripheral venous access is poor
- Access for hemodialysis, plasmapheresis, right heart catheterization and placement of a temporary transvenous pacemaker

The common sites for central venous cannulation are the femoral, internal jugular, and subclavian veins. There are no absolute contraindications to place a central venous catheter, but each site has potential risks. All sites share the common complications of infection (site cellulitis, bacteremia), venous thrombosis with potential emboli, air embolism, catheter malfunction (occlusion, dislodgement, fractures), dysrhythmias (when the catheter tip is in the heart), and bleeding. Universal precautions and sterile technique should be used when placing a central venous catheter.

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**INVASIVE LINE PLACEMENT AND MONITORING**

**Arterial Catheter Insertion**

Arterial catheters are indicated when there is a need for precise beat-to-beat blood pressure monitoring, acid–base status, and arterial blood gases. There are no absolute contraindications to placing an arterial catheter, but a risk/benefit analysis should be performed in patients with a hypercoaguable state or bleeding disorder.
1. **Femoral Vein Cannulation**

   Femoral venous catheterization is the central venous access route used most commonly in infants and children in emergency situations because it lies adjacent to the easily palpable femoral artery. Furthermore, femoral anatomy is easily learned, and hemostasis can be obtained quickly in the event of an accidental arterial puncture. Disadvantages of this approach include the possibility of unintentional femoral artery puncture and subsequent limited ability to flex the hip. Numerous studies have shown no higher incidence of complications from femoral cannulation compared to the subclavian or internal jugular routes. If the patient has abdominal wounds or a significant intraabdominal process, central venous access should be obtained above the diaphragm.

   At the level of the inguinal ligament, the femoral vein is the most medial structure in the femoral sheath, which also contains the femoral nerve and artery. To optimize successful femoral cannulation, the patient’s leg should be slightly rotated externally. In infants and small children, a towel under the buttocks flattens the inguinal area and makes the angle less acute to enter the femoral vein. The femoral artery is identified approximately 1 cm below the inguinal crease where the needle is inserted in a medial direction towards the umbilicus, and advanced at a 45-degree angle until free flow of blood is obtained in the syringe. After successful venous puncture, the Seldinger technique is used to complete the cannulation. If the femoral pulse cannot be palpated, it is found by drawing an imaginary line from the anterior superior iliac crest to the pubic tubercle, and then divided into three equal segments. The femoral artery lies at the junction between the middle and most medial segment, and the femoral vein is medial to that point.

2. **Internal Jugular Vein Cannulation**

   The internal jugular vein is located under the sternocleidomastoid muscle in the neck and follows an oblique course as it runs down the neck in the carotid sheath. It drains into the subclavian vein to form the brachiocephalic (innominate) vein behind the head of the clavicle. The right side is preferred because the vessel runs a longer and follows a straighter path, making it easier to cannulate; the location of the catheter tip should be confirmed radiographically. The most ideal position for the tip of the catheter is at the junction of the superior vena cava and the right atrium. Using the middle insertion approach, the proper length can be estimated from the patient’s weight (Table 38-9).

   Once the venipuncture is accomplished, the syringe is removed and the end of the needle occluded to prevent air embolism. The Seldinger technique is used to complete the cannulation; the location of the catheter tip should be confirmed radiographically. The most ideal position for the tip of the catheter is at the junction of the superior vena cava and the right atrium. Using the middle insertion approach, the proper length can be estimated from the patient’s weight (Table 38-9).

   Successful cannulation of the internal jugular vein can be improved by using ultrasound guidance. Additional maneuvers that improve cannulation success are to enlarge the internal jugular vein by a Valsalva maneuver or external compression over the liver.

   Complications of internal jugular vein cannulation include carotid artery puncture, and thoracic duct injury with left-sided attempts. If a previous attempt led to a hematoma, the other side should not be attempted because bilateral hematomas may lead to upper-airway compromise. Also, placing an internal jugular venous catheter is relatively contraindicated in patients at risk for intracranial hypertension because venous occlusion may increase intracranial pressure. Some drawbacks of a catheter in this position are that conscious patients have limited neck mobility, there is a higher risk of thrombotic occlusion from neck flexion, and it is difficult to keep the area clean, especially in patients with a tracheostomy.

3. **Subclavian Vein Cannulation**

   The subclavian vein is a continuation of the axillary vein as it passes over the first rib and lies posterior to the medial third of the clavicle. At the thoracic inlet, it meets the internal jugular vein to form the brachiocephalic (innominate) vein. The left subclavian vein is longer and follows a straighter path, making it easier to
insert a catheter. The technique is used less frequently in children because the vessel is smaller and more cephalad under the clavicle. The advantages of this approach are its fixed landmarks and patient comfort once secured. The risks of pneumothorax and hemothorax appear to be slightly higher with the subclavian approach than with the internal jugular approach. These risks are increased if there is a chest wall deformity.

To catheterize the subclavian vein, the patient is placed in the Trendelenburg position with the head turned away from the insertion site (if there is no cervical spine injury). With the infraclavicular approach, the needle is inserted at a point one-half to two-thirds its length away from the sternoclavicular junction. If the patient is receiving mechanical ventilation, temporary disruption of ventilation during insertion of the needle keeps the apex of the lung away from the needle. While continuously aspirating, the needle is advanced under the clavicle toward the suprasternal notch. Once a free flow of blood is obtained in the syringe, the syringe is disconnected and a finger is placed over the hub of the needle to prevent entrainment of air. During a positive-pressure breath or exhalation, the guidewire is advanced into the right atrium. The needle is removed, the tract is dilated, and the catheter is inserted using the Seldinger technique. Blood should be easily aspirated from all ports of the catheter. A chest radiograph should be obtained to verify the position of the catheter tip and to rule out pneumothorax.

ARTICLES TO KNOW


### Table 38-1 Beta-adrenergic Therapies for Status Asthmaticus

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route</th>
<th>Dose</th>
<th>Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol 0.5% solution</td>
<td>Aerosolized</td>
<td>0.05 mL/kg (= 0.15 mg/kg)</td>
<td>Every 20 minutes × 3, but can be given continuously</td>
</tr>
<tr>
<td>Terbutaline 0.05%</td>
<td>Subcutaneous</td>
<td>0.01 mg/kg/dose</td>
<td>Every 15–20 minutes × 3</td>
</tr>
<tr>
<td>Epinephrine 1:1000</td>
<td>Subcutaneous</td>
<td>0.01 mL/kg/dose (= 10 µg/kg)</td>
<td>Every 15 minutes × 3</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>Intravenous</td>
<td>Maximum 0.5 mL</td>
<td>Continuous infusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2–10 µg/kg loading dose, then 0.08-6 µg/kg/min</td>
<td></td>
</tr>
</tbody>
</table>
Table 38-2  Additional Therapies for Status Asthmaticus

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosing Regimen</th>
<th>Notable Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>0.03–0.05 mg/kg aerosolized every 1 h or more frequently as needed</td>
<td>Anticholinergic: dry mouth, blurred vision, tachycardia</td>
</tr>
<tr>
<td>Ipratropium bromide</td>
<td>Infants: 0.25 mg aerosolized Children &gt;7 years: 0.5 mg aerosolized</td>
<td>Peak effect: 30 minutes Frequency: Every 4–6 h</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>Loading dose: 2 mg/kg IV Maintenance: 0.5–1 mg/kg IV every 6 h</td>
<td>Hyperglycemia, hypertension, acute psychosis</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>2–4 mg/kg IV every 4–6 h</td>
<td></td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>25–75 mg/kg IV Maximum 1–2 gm/dose</td>
<td>Hypotension, flushing</td>
</tr>
</tbody>
</table>

Table 38-3  Hypotension in Infants and Children

<table>
<thead>
<tr>
<th>Age</th>
<th>Minimum Systolic Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1 month</td>
<td>60</td>
</tr>
<tr>
<td>1–12 months</td>
<td>70</td>
</tr>
<tr>
<td>&gt;1 year</td>
<td>70 + (2 times age in years)</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>90</td>
</tr>
</tbody>
</table>

Table 38-4  Classification of Hemorrhagic Shock

<table>
<thead>
<tr>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated blood volume deficit</td>
<td>10–15%</td>
<td>20–25%</td>
<td>30–35%</td>
</tr>
<tr>
<td>Pulse (beats/min)</td>
<td>&gt;100</td>
<td>&gt;150</td>
<td>&gt;150</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Normal</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>&lt;5 s</td>
<td>5–10 s</td>
<td>10–15 s</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Decreased pulse pressure</td>
<td>Decreased</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal</td>
<td>Anxious</td>
<td>Confused</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Urine output</td>
<td>1–3 mL/kg</td>
<td>0.5–1 mL/kg</td>
<td>&lt;0.5 mL/kg</td>
</tr>
</tbody>
</table>

Table 38-5  Clinical Staging of Hepatic Encephalopathy

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No disease</td>
</tr>
<tr>
<td>1</td>
<td>Periods of lethargy, altered personality, altered spatial orientation</td>
</tr>
<tr>
<td>2</td>
<td>Inappropriate behavior or drowsiness, confusion, asterixis, hyperreflexia</td>
</tr>
<tr>
<td>3</td>
<td>Stuporous but arousable, sleeps most of the time, agitated, incoherent speech</td>
</tr>
<tr>
<td>4</td>
<td>Coma: 4a = responds to painful stimuli; 4b = unresponsive to painful stimuli</td>
</tr>
</tbody>
</table>

### Table 38-6 Causes of Acute Renal Failure

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Clinical Scenario</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal</td>
<td></td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Vomiting, diarrhea, hemorrhage</td>
</tr>
<tr>
<td>Hypoperfusion</td>
<td>Myocardial dysfunction, sepsis, hypoxia</td>
</tr>
<tr>
<td>Drugs</td>
<td>NSAIDs, ACE inhibitors</td>
</tr>
<tr>
<td>Renal</td>
<td></td>
</tr>
<tr>
<td>Vascular occlusion</td>
<td>Bilateral renal artery or vein thrombosis</td>
</tr>
<tr>
<td>Glomerular injury</td>
<td>Hemolytic uremic syndrome, glomerulonephritis</td>
</tr>
<tr>
<td>Tubular injury</td>
<td>Acute tubular necrosis, nephrotoxins</td>
</tr>
<tr>
<td>(aminoglycosides)</td>
<td></td>
</tr>
<tr>
<td>Tubular obstruction</td>
<td>Tumor lysis syndrome, rhabdomyolysis</td>
</tr>
<tr>
<td>Postrenal</td>
<td></td>
</tr>
<tr>
<td>Obstruction</td>
<td>Urinary tract anomalies: posterior urethral valves, renal stones, neurogenic bladder</td>
</tr>
</tbody>
</table>

ACE, angiotensin-converting enzyme; NSAID, nonsteroidal anti-inflammatory drug.

### Table 38-9 Recommended Length of Central Venous Catheter Insertion Using the Middle Approach to the IJV

<table>
<thead>
<tr>
<th>Patient Weight (kg)</th>
<th>Length of CVC Insertion (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2–2.9</td>
<td>4</td>
</tr>
<tr>
<td>3–4.9</td>
<td>5</td>
</tr>
<tr>
<td>5–6.9</td>
<td>6</td>
</tr>
<tr>
<td>7–9.9</td>
<td>7</td>
</tr>
<tr>
<td>10–12.9</td>
<td>8</td>
</tr>
<tr>
<td>13–19.9</td>
<td>9</td>
</tr>
<tr>
<td>20–29.9</td>
<td>10</td>
</tr>
<tr>
<td>30–39.9</td>
<td>11</td>
</tr>
<tr>
<td>40–49.9</td>
<td>12</td>
</tr>
<tr>
<td>50–59.9</td>
<td>13</td>
</tr>
<tr>
<td>60–69.9</td>
<td>14</td>
</tr>
<tr>
<td>70–79.9</td>
<td>15</td>
</tr>
<tr>
<td>≥80</td>
<td>16</td>
</tr>
</tbody>
</table>


### Table 38-8 Classification of Respiratory Failure

<table>
<thead>
<tr>
<th>Arterial Pressure</th>
<th>Type I (Hypoxemic)</th>
<th>Type II (Hypercarbic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{aO_2}$</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>$P_{aCO_2}$</td>
<td>Normal or low</td>
<td>High</td>
</tr>
</tbody>
</table>

### Table 38-7 Treatment of Hyperkalemia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Mechanism of Action</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium gluconate (10%)</td>
<td>50–100 mg/kg IV</td>
<td>Stabilizes myocyte membrane potential</td>
<td>Bradyarrhythmias</td>
</tr>
<tr>
<td>Calcium chloride (10%)</td>
<td>10–20 mg/kg IV</td>
<td>Stabilizes myocyte membrane potential</td>
<td>Bradyarrhythmias</td>
</tr>
<tr>
<td>Sodium Bicarbonate</td>
<td>1–2 mEq/kg IV over 10–30 minutes</td>
<td>Shifts extracellular K⁺ into cells</td>
<td>Hypernatremia, alkalosis, tetany</td>
</tr>
<tr>
<td>Insulin</td>
<td>0.1 units/kg IV</td>
<td>Stimulates cellular uptake of K⁺</td>
<td>(decreases ionized calcium)</td>
</tr>
<tr>
<td>Albuterol (0.5%)</td>
<td>0.03 mL/kg nebulized in normal saline</td>
<td>Stimulates uptake of intracellular K⁺</td>
<td>Hypoglycemia; administer IV glucose 0.5 g/kg</td>
</tr>
<tr>
<td>Sodium polystyrene sulfonate (Kayexalate)</td>
<td>1 g/kg by mouth or per rectum</td>
<td>Exchange of Na⁺ for K⁺ across gut mucosa</td>
<td>Tachycardia; arrhythmias</td>
</tr>
</tbody>
</table>

Hypernatremia, diarrhea.
Box 38-1 The ABCDE Approach to Pediatric CPR

<table>
<thead>
<tr>
<th>A</th>
<th>Airway</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Breathing</td>
</tr>
</tbody>
</table>
| C | Circulation  
Cervical spine immobilization if trauma |
| D | Disability  
Drugs such as epinephrine, antibiotics, and dextrose, if indicated |
| E | Exposure  
Environment (such as temperature)  
Electrical activity of the heart  
Extracorporeal membrane oxygenation, if necessary or available as a last resort |

Box 38-2 Effects of Beta-receptor Stimulation for the Treatment of Status Asthmaticus

**Beneficial Actions**
- Increases cyclic AMP concentration, leading to smooth muscle relaxation
- Prevents and reverses the effect of bronchoconstrictor substances on small- and large-airway smooth muscle cells
- Enhances mucociliary clearance
- Inhibits cholinergic transmission
- Inhibits mediator release

**Adverse Actions**
- Tremors secondary to skeletal muscle $\beta_2$ stimulation
- Tachycardia secondary to $\beta_1$ (cardiac) and $\beta_2$ (vasodilation) stimulation
- Dysrhythmias
- Hypertension or hypotension
- Ventilation/perfusion mismatch with reversal of hypoxic pulmonary vasoconstriction
- Metabolic: hyperglycemia, hypokalemia, hypomagnesemia
- Downregulation of $\beta$ receptors
- AMP, adenosine monophosphate

Box 38-3 Common Features in Chronic Renal Failure

**Electrolyte Disorders**
- Hyperkalemia
- Hyperphosphatemia
- Hypocalcemia
- Hypermagnesemia
- Hyponatremia
- Metabolic acidosis

**Gastrointestinal**
- Delayed gastric emptying
- Nausea and vomiting

**Hematologic**
- Anemia
- Platelet dysfunction secondary to uremia

**Cardiovascular**
- Hypertension
- Volume overload

**Endocrine**
- Growth failure
Figure 38-1  In the two-thumb-encircling hand technique for chest compression in infants, the thumbs should be located one finger width below the intermammary line. The sternum should be depressed one-third to one-half the depth of the infant’s chest at a rate of at least 100 times per minute.

Figure 38-2  Algorithm for treatment of status epilepticus. (Redrawn with permission from Nichols et al: *Golden Hour: The Handbook of Advanced Pediatric Life Support*, 2nd edn, Mosby, Philadelphia, 1996.)
**Fluid Volume Resuscitation in Hypovolemic Shock**

- **Documented Hypovolemic Shock**
  - 10–20 ml/kg IV LR or NS
  - Remains Unstable?
  - Repeat up to 60 ml/kg
  - Remains Unstable?
  - Add Inotropic Drugs: Dopamine and/or Epinephrine
  - Remains Unstable?
  - Measure Central Venous Pressure
  - Continue LR or NS until CVP greater than 10–12 mmHg

**Figure 38-3** Algorithm for treatment of hypovolemic shock. (Redrawn with permission from Nichols et al: *Golden Hour: The Handbook of Advanced Pediatric Life Support*, 2nd edn, Mosby, Philadelphia, 1996.)

**Figure 38-4** Intracranial compliance curve. When the intracranial volume increases from points 1 to 2, the ICP slightly increases. However, from points 3 to 4, a small increase in volume will cause a dramatic increase in ICP (Monro–Kellie principle). (Redrawn with permission from Nichols et al: *Golden Hour: The Handbook of Advanced Pediatric Life Support*, 2nd edn, Mosby, Philadelphia, 1996.)

**Figure 38-5** Relationship between cerebral blood flow and mean arterial pressure, arterial oxygen tension, and arterial carbon dioxide tension. (Redrawn with permission from Nichols et al: *Golden Hour: The Handbook of Advanced Pediatric Life Support*, 2nd edn, Mosby, Philadelphia, 1996.)
Airway, Breathing, Circulation

Maintain Adequate CPP:
- <1 yr.: >50 mm Hg
- 1–10 yrs.: >60 mm Hg
- >10 yrs.: >70 mm Hg
- Elevate HOB to 30°
- Sedation & Analgesia
- Neuromuscular Blockade

ICP >20 mm Hg?

CSF Drainage (if available)

IV Mannitol (0.25–1.0 g/kg)
OR
IV 3% NaCl (3–5 ml/kg), especially if the patient is hypovolemic
May repeat dose of either if serum osmolality <320 mOsm and patient is euvoletic

ICP >20 mm Hg?

Consider repeating CT Scan

IV Pentobarbital (5 mg/kg)
Repeat dose until ICP controlled or burst suppression on EEG

ICP >20 mm Hg?

Decompressive craniectomy

Hyperventilation (PaCO2 <35 mm Hg)
CBF and/or SjO2 monitoring recommended

Hypothermia 32–34°C

Figure 38-6  Algorithm for treatment of increased intracranial pressure. (Redrawn with permission from Nichols et al: Golden Hour: The Handbook of Advanced Pediatric Life Support, 2nd edn, Mosby, Philadelphia, 1996.)