and bronchial smooth muscle relaxation.\textsuperscript{19} Two formulations of epinephrine (adrenaline) can be used for nebulization. The \textit{l}-epinephrine (\textit{l}-adrenaline) dose is 0.5 mL/kg of a 1 mg/mL concentration to a maximum of 5 mL. Racemic epinephrine (adrenaline), which contains both stereoisomers of epinephrine (adrenaline), is dosed as 0.05 mL/kg to a maximum of 0.5 mL of a 2.25% solution with a saline diluent. Comparison of these two forms has not demonstrated a significant difference in efficacy.\textsuperscript{20}

Steroids should be administered early in the management of moderate to severe croup. Nebulized budesonide and dexamethasone, given intramuscularly or orally, are the two corticosteroids that have been studied for the treatment of croup. Comparison studies have revealed no significant difference in efficacy.\textsuperscript{21–23} Dexamethasone is a long-acting corticosteroid with a half-life of 36 to 72 hours. Effective dexamethasone dosages of 0.15 to 0.6 mg/kg per dose either orally or intramuscularly have shown similar benefits. The 0.6-mg/kg dose is more widely used in clinical practice (not to exceed 10 mg).\textsuperscript{16,23,24} Benefits of dexamethasone in the hospital setting include faster improvements in symptoms and croup scores, decreased incidence of endotracheal intubation in children admitted for croup, and shorter hospital stays.\textsuperscript{19} Safety and efficacy of dexamethasone for mild to moderate croup in the outpatient setting have been demonstrated by fewer return visits and lower hospitalization rates.\textsuperscript{21,23,25,26} Budesonide at a dose of 2 mg nebulized in saline solution has shown similar efficacy compared with dexamethasone.\textsuperscript{21,22} In a recent study of American freestanding children’s hospitals, corticosteroids were prescribed for 82% of children with croup seen in the ED.\textsuperscript{27}

Several trials of heliox have demonstrated no advantage over conventional modalities; however, other trials have shown it to be equally effective in moderate-to-severe croup when compared with racemic epinephrine (adrenaline). It has also been shown to improve symptoms in very severe croup that failed to improve with racemic epinephrine (adrenaline). Currently, there is a lack of evidence to establish the effect of heliox inhalation in the treatment of croup in children.\textsuperscript{28,29}

General indications for endotracheal intubation in respiratory failure should be used in the treatment of a child with severe croup. Endotracheal intubation or tracheostomy can be necessary in less than 2% of patients. Tracheal tube size should be half to a full size smaller than predicted by size and age to allow for subglottic edema and to reduce the risk of subglottic stenosis.\textsuperscript{30}

In the past, treatment with nebulized racemic epinephrine (adrenaline) was a criterion for admission to the hospital. This was due to the “rebound” effect with return of symptoms up to 2 hours after treatment with racemic epinephrine (adrenaline). This dogma was mostly driven by limited data from earlier studies with small sample sizes. Recent literature has refuted this practice and concluded that use of racemic epinephrine (adrenaline) in the ED for outpatient treatment of croup is safe and effective. These studies recommended an observation period of 3 to 4 hours in the ED before discharge, with documentation of periodic croup scores and reevaluation for return of symptoms. During observation, patients who experienced relapse of symptoms were hospitalized but did not have posttreatment croup scores that were worse than on presentation to the ED. No significant complications were seen in patients discharged home. All of the patients included in the studies

\section*{KEY POINTS}

Management of Croup

\begin{itemize}
\item Begin cool mist as long as it does not increase agitation.
\item Begin treatment with steroids: dexamethasone, 0.15 to 0.6 mg/kg orally or intramuscularly, or budesonide, 2 mg per 2 mL of saline nebulized.
\item Begin epinephrine (adrenaline) for signs of moderate to severe respiratory distress: racemic epinephrine (adrenaline), 0.05 mL/kg, to a maximum of 0.5 mL of 2.25% in 2 mL of saline nebulized, or \textit{l}-epinephrine (\textit{l}-adrenaline; 1 mg/mL solution), 0.5 mL/kg nebulized.
\item Assist ventilation for signs of respiratory failure.
\end{itemize}
In July 2005, the National Institute of Allergy and Infectious Disease and Food Allergy and Anaphylaxis Network convened an international consensus meeting to develop universal agreement on the definition of anaphylaxis, the criteria for diagnosis, and treatment.65

Diagnosing Studies
The diagnosis of anaphylaxis is made clinically. Routine laboratory studies or radiographic studies are not necessary in the diagnosis or management of anaphylaxis.

Management
Epinephrine (adrenaline) is the treatment of choice in anaphylaxis. The action of epinephrine (adrenaline) is at the α- and β-adrenergic receptors, causing vasoconstriction, increasing cardiac inotropy and chronotropy, and bronchodilation. A dosage of 0.01 mL/kg (0.01 mg/kg) of 1 mg/mL solution to a maximum dose of 0.3 mL (0.5 mg), administered intramuscularly every 5 to 15 minutes as necessary, is the recommended dosage for controlling symptoms and maintaining blood pressure. When the intravenous route is not indicated, the intramuscular route is preferable to the subcutaneous route due to more rapid and reliable absorption. The anterolateral thigh is the preferred site in both children and adults. There is evidence for better absorption at this site compared with a deltoid intramuscular injection or subcutaneous injection.81,85

Histamine-1 (H1)-antagonist antihistamines, such as diphenhydramine, should be given intravenously to all patients with anaphylaxis. These medications are effective in treating the cutaneous symptoms of urticaria and itching. H2-antagonist antihistamines are also recommended. Bronchospasm is treated with nebulized β2-agonists, such as albuterol (salbutamol). Parenteral corticosteroids are given routinely to patients with anaphylaxis.50 Vasopressors should be administered in cases of hypotension refractory to intravenous fluid resuscitation. Dopamine and/or norepinephrine (noradrenaline) is titrated to maintain adequate blood pressure.

Endotracheal intubation is seldom required in the management of anaphylaxis.64 However, it is important to remember that respiratory arrest secondary to airway obstruction is the leading cause of death in fatal cases of anaphylaxis.63,66 In addition to the general indications for endotracheal intubation in respiratory failure, patients in respiratory distress with signs of severe upper airway obstruction, lingual edema, hypopharyngeal edema, or laryngeal edema should be intubated.

**YOUR FIRST CLUE**

Signs and Symptoms of Anaphylaxis
Anaphylaxis is highly likely when any one of the following 3 criteria is fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, or swollen lips, tongue, or uvula)
   AND AT LEAST ONE OF THE FOLLOWING:
   - Respiratory compromise (dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow [PEF], or hypoxemia)
   - Reduced BP or associated symptoms of end-organ dysfunction (hypotonia, syncope or incontinence)
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
   - Involvement of the skin-mucosal tissue (generalized hives, itch or flush, or swollen lips, tongue, or uvula)
   - Respiratory compromise (dyspnea, wheeze-bronchospasm, stridor, reduced PEF, or hypoxemia)
   - Reduced BP or associated symptoms (hypotonia, syncope, or incontinence)
   - Persistent gastrointestinal symptoms (crampy abdominal pain or vomiting)
3. Reduced BP after exposure to known allergen for that patient (minutes to several hours):
   - Infants and children: low systolic BP for age or greater than 30% decrease in systolic BP*
   - Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person’s baseline

*Low systolic blood pressure for children is defined as less than 70 mm Hg from 1 month to 1 year, less than 70 mm Hg + (2 × age) from 1 to 10 years, and less than 90 mm Hg from 11 to 17 years.
early to avoid the dreaded scenario of complete airway obstruction with the inability to intubate or ventilate.

Disposition

No studies have specifically addressed disposition of patients with anaphylaxis from the ED. A reasonable length of time to consider observing the postanaphylactic patient is 4 to 6 hours in most patients, with prolonged observation times or hospital admission for patients with severe or refractory symptoms. More caution should be used in patients with reactive airway disease because most fatalities associated with anaphylaxis occur in these patients. Patients with mild symptoms that resolve after medical therapy can be safely discharged home. Patients who present with moderate to severe anaphylaxis should be admitted to the hospital for observation after treatment in the ED. Biphagic reactions account for up to 50% of fatal cases. In a large pediatric population, biphagic reactions were reported in 6% of patients admitted to the hospital, of which half were serious and required repeat administration of epinephrine (adrenaline) and 1% required intubation. The asymptomatic interval varied widely, from 1 to 28 hours.

Preventive measures are vital in the treatment of anaphylaxis. After an anaphylactic reaction, all patients should undergo skin testing to identify potential allergens. Medical bracelets are recommended for anaphylaxis patients to identify their serious allergies. Epinephrine (adrenaline) self-injection kits should be prescribed to all patients with a history of anaphylaxis. Several studies have demonstrated the deficiencies in proper use of epinephrine (adrenaline) injection kits. A review of anaphylaxis deaths reported patients holding an unused kit in hand, use of an expired kit, and one case of a patient who died waiting for the prescription to be filled at a pharmacy.
TABLE 2-11 Dosages of Drugs for Asthma Exacerbations

<table>
<thead>
<tr>
<th>Medication</th>
<th>Child Dose</th>
<th>Adult Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhaled Short-Acting β₂-Agonists (SABA)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albuterol</td>
<td>0.15 mg/kg (minimum dose 2.5 mg every 20 minutes for 3 doses then 0.15–0.3 mg/kg up to 10 mg every 1–4 hours as needed, or 0.5 mg/kg/hour by continuous nebulization.)</td>
<td>2.5–5 mg every 20 minutes for 3 doses, then 2.5–10 mg every 1–4 hours as needed, or 10–15 mg/hour continuously.</td>
<td>Only selective beta₂-agonists are recommended. For optimal delivery, dilute aerosols to minimum of 3 mL at gas flow of 6–8 L/min. Use large volume nebulizers for continuous administration. May mix with ipratropium nebulizer solution.</td>
</tr>
<tr>
<td>Nebulizer solution</td>
<td>4–8 puffs every 20 minutes for 3 doses, then every 1–4 hours inhalation maneuver as needed. Use spacing chamber; add mask in children &lt;4 years.</td>
<td>4–8 puffs every 20 minutes up to 4 hours, then every 1–4 hours as needed.</td>
<td>In mild-to-moderate exacerbations, MDI plus spacing chamber is as effective as nebulized therapy with appropriate administration technique and coaching by trained personnel.</td>
</tr>
<tr>
<td>Bitolterol</td>
<td>See albuterol dose; thought to be half as potent as albuterol on mg basis.</td>
<td>See albuterol dose.</td>
<td>Has not been studied in severe asthma exacerbations. Do not mix with other drugs.</td>
</tr>
<tr>
<td>Nebulizer solution</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levalbuterol (R-albuterol)</td>
<td>See albuterol MDI dose.</td>
<td>See albuterol MDI dose.</td>
<td>Has not been studied in severe asthma exacerbations.</td>
</tr>
<tr>
<td>Nebulizer solution</td>
<td>0.075 mg/kg (minimum dose 1.25 mg every 20 minutes for 3 doses, then 0.075–0.15 mg/kg up to 5 mg every 1–4 hours as needed.)</td>
<td>1.25–2.5 mg every 20 minutes for 3 doses, then 1.25–5 mg every 1–4 hours as needed.</td>
<td>See albuterol MDI dose. Levalbuterol administered in one-half the mg dose of albuterol provides comparable efficacy and safety. Has not been evaluated by continuous nebulization.</td>
</tr>
<tr>
<td>MDI (45 mcg/puff)</td>
<td>See albuterol MDI dose.</td>
<td>See albuterol MDI dose.</td>
<td></td>
</tr>
<tr>
<td>Pirbuterol</td>
<td>See albuterol MDI dose; thought to be half as potent as albuterol on a mg basis.</td>
<td>See albuterol MDI dose.</td>
<td>Has not been studied in severe asthma exacerbations.</td>
</tr>
<tr>
<td>MDI (200 mcg/puff)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic (Injected) β₂-Agonists</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.01 mg/kg up to 0.3–0.5 mg every 20 minutes for 3 doses subcutaneous.</td>
<td>0.3–0.5 mg every 20 minutes for 3 doses subcutaneous.</td>
<td>No proven advantage of systemic therapy over aerosol.</td>
</tr>
<tr>
<td>1 mg/mL concentration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terbutaline</td>
<td>0.01 mg/kg up to 0.3 mg every 20 minutes for 3 doses then every 2–6 hours as needed subcutaneous.</td>
<td>0.25 mg every 20 minutes for 3 doses subcutaneous.</td>
<td>No proven advantage of systemic therapy over aerosol.</td>
</tr>
</tbody>
</table>
TABLE 3-2 Signs, Symptoms, and Management of Hypovolemic, Septic, Distributive, and Cardiogenic Shock

<table>
<thead>
<tr>
<th>Type of Shock</th>
<th>Pathophysiology</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>( \Delta \text{CO}, \Delta \text{SVR} ) intravascular with or without interstitial volume loss</td>
<td>( \Delta \text{HR}, \Delta \text{pulses}, \text{delayed CR, hyperpnea, dry skin, sunken eyes, oliguria, BP normal until late} )</td>
<td>Repeat boluses of 20 mL/kg of crystalloid as indicated. Blood products as indicated for acute blood loss.</td>
</tr>
<tr>
<td>Septic*</td>
<td>( \Delta \text{CO}, \Delta \text{SVR} ) (classic adult, 20% pediatric)</td>
<td>( \Delta \text{HR}, \Delta \text{BP}, \Delta \text{pulses}, \text{delayed CR, hyperpnea, MS changes, third spacing, edema} )</td>
<td>Repeat boluses of 20 mL/kg of crystalloid; might need &gt;60 mL/kg in first hour. Consider colloid if poor response to crystalloid. Pharmacologic support of BP with dopamine or norepinephrine (noradrenaline).</td>
</tr>
<tr>
<td></td>
<td>( \Delta \text{CO}, \Delta \text{SVR} ) (60% pediatric)</td>
<td>( \Delta \text{HR}, \text{normal to } \Delta \text{BP}, \Delta \text{pulses, delayed CR, hyperpnea, MS changes, third spacing, edema} )</td>
<td>Repeat boluses of 20 mL/kg of crystalloid; might need &gt;60 mL/kg in first hour. Consider colloid if poor response to crystalloid. Pharmacologic support of CO with dopamine or epinephrine (adrenaline).</td>
</tr>
<tr>
<td></td>
<td>( \Delta \text{CO}, \Delta \text{SVR} ) (20% pediatric)</td>
<td>( \Delta \text{HR}, \Delta \text{BP}, \Delta \text{pulses, delayed CR, hyperpnea, MS changes, third spacing, edema} )</td>
<td>Repeat boluses of 20 mL/kg of crystalloid; might need &gt;60 mL/kg in first hour. Consider colloid if poor response to crystalloid. Pharmacologic support of CO and BP with dopamine and epinephrine (adrenaline).</td>
</tr>
<tr>
<td>Distributive Anaphylaxis</td>
<td>( \Delta \text{CO}, \Delta \text{SVR} )</td>
<td>Angioedema, rapid third spacing of fluids, ( \Delta \text{HR, respiratory distress} )</td>
<td>Repeat boluses of 20 mL/kg of crystalloid as indicated. Pharmacologic support of SVR with norepinephrine (noradrenaline) or phenylephrine.</td>
</tr>
<tr>
<td>Spinal cord injury: normal CO, ( \Delta \text{SVR} )</td>
<td>( \Delta \text{BP with normal HR, paralysis with loss of vascular tone} )</td>
<td>Pharmacologic support of SVR with norepinephrine (noradrenaline) or phenylephrine. Fluid resuscitation as indicated by clinical status and associated injuries.</td>
<td></td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>( \text{CO, normal to } \Delta \text{SVR} )</td>
<td>Normal to ( \Delta \text{HR, } \Delta \text{pulses, delayed CR, oliguria, JVD, hepatomegaly, BP normal until late in course} )</td>
<td>Pharmacologic support of CO with dobutamine, milrinone, dopamine. Judicious fluid replacement as indicated clinically.</td>
</tr>
</tbody>
</table>

*In regions with limited access to critical care resources, treat all forms of septic shock with an initial fluid bolus of 20 mL/kg of crystalloid. Additional fluid boluses should be done with extreme caution. The patient should be reassessed after every fluid bolus.

Abbreviations: BP, blood pressure; CO, cardiac output; CR, capillary refill; HR, heart rate; JVD, jugular venous distension; MS, mental status; SVR, systemic vascular resistance.


Hypovolemic shock is decreasing steadily but remains an important cause for hospital admission and frequent ED visits.

Hypovolemic shock can be categorized into two major subtypes: hemorrhagic and nonhemorrhagic. The causes of hypovolemic shock are listed in Table 3-3.

**Etiology**

Hemorrhagic-hypovolemic shock can be caused by trauma or it can be atraumatic. An adult can present after trauma in hemorrhagic-hypovolemic shock with major bleeding into the thorax, abdomenopelvic cavity, retroperitoneum, bilateral thigh compartments, and onto “the street.” A child can present with significant hemorrhagic-hypovolemic shock by bleeding into all of these locations and with intracranial bleeding. In addition to trauma, hemorrhagic-hypovolemic shock can present after gastrointestinal bleeding or surgery. Nonhemorrhagic hypovolemic shock most commonly presents after significant vomiting and...
Distributive Shock

Intramuscularly or intravenously, depending on severity of symptoms. If administered intramuscularly, 0.01 mg/kg of the 1 mg/mL solution can be administered to a maximum of 0.3 mg. If administered intravenously, 0.01 mg/kg of the 0.1 mg/mL solution can be administered to a maximum of 0.5 mg with or without an epinephrine (adrenaline) drip (0.1–1 mcg/kg per minute). Epinephrine (adrenaline) counteracts the vasodilatation caused by the inflammatory cytokines and histamine, raises SVR, improves cardiac inotropy, and decreases capillary permeability and angioedema. Intravenous histamine1 antagonists, such as diphenhydramine (1.25 mg/kg per dose: to maximum of 50 mg), should also be given to decrease further mast cell degranulation. Histamine2 antagonists have also been demonstrated to decrease severity and length of anaphylactoid reactions. Multiple histamine2 antagonists exist in intravenous form. Lastly, intravenous steroids have also been shown to decrease recurrence of symptoms and shorten symptom duration and should be given accordingly.

Neurogenic Shock

Traumatic disruption of the cervicothoracic spinal cord can cause a transient state of hypotension and bradycardia termed neurogenic shock. This constellation of symptoms results from decreased sympathetic outflow to the heart and vasculature, causing a decrease in SVR, HR, and SV. The sympathetic nervous system contains central neurons that start in the reticular system of the brain and then synapse in the lateral gray matter of the thoracolumbar spinal cord. An acute disruption proximal to the thoracolumbar spinal cord decreases all efferent signaling to the sympathetic nervous system. Because the parasympathetic nervous system is controlled above the level of the cervical cord, the end result is unopposed parasympathetic stimulation.

Etiology

Blunt or penetrating trauma to the neck can cause cervical disruption. The degree of sympathectomy depends on the completeness of injury.

YOUR FIRST CLUE

Signs and Symptoms of Anaphylactic Shock
- Pruritus
- Urticaria
- Angioedema
- Tachypnea, wheezing, or stridor
- Delayed capillary refill
- Tachycardia
- Hypotension

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Clinical Features
Patients in neurogenic shock present with signs and symptoms of cervicothoracic neurologic disruption. The preganglionic sympathetic neurons are located in the intermediolateral cell column lamina VII, which is in the lateral aspect of the gray matter in the cervicothoracic spinal cord. Spinotalamic and corticospinal tracts are in close proximity to this area, so disruption of these cell bodies might result in paralysis and sensory deficits. In addition, patients present with signs and symptoms of poor perfusion. Hypotension and paradoxical bradycardia are usually present due to the lack of sympathetic tone to the myocardium and vasculature. One should be aware that hypotension and bradycardia in the traumatized patient does not exclude significant hemorrhage as a cause of shock. Patients with significant cord injuries and neurogenic shock can have hemorrhage without significant tachycardia.

Diagnostic Studies
Laboratory
Laboratory tests to exclude and/or manage hypovolemic hemorrhagic shock should be performed. There are no specific tests in the evaluation of neurogenic shock.

Radiology
Cervical computed tomography has largely supplanted plain radiography in the initial evaluation of the neurologically afflicted trauma patient. It provides detailed information regarding cervical spine fractures, ligamentous instability, and subluxations. Magnetic resonance imaging is also indicated in the evaluation of the neurologically afflicted trauma patient for several reasons. First, the incidence of spinal cord injury without radiographic abnormality is 20% to 36% of pediatric patients with traumatic myelopathy. In addition, magnetic resonance imaging can aid in the prognostication of injuries. Cervical cord contusions and ligamentous injuries can have some functional recovery, whereas cervical cord lacerations and transections generally demonstrate poorer outcomes.

Management
Immediate cervical spine immobilization and management of airway and breathing take priority. Circulatory support can be managed with judicious application of intravenous crystalloid fluids in 20-mL/kg boluses initially. However, because the primary problem is a loss of sympathetic tone, use of a vasopressor, such as norepinephrine (noradrenaline) or dopamine, temporarily to increase SVR and improve perfusion to an already injured cord is of greater efficacy. Early use of systemic steroids are controversial, with recent data revealing no significant trend toward benefit.

Septic Shock
Sepsis remains a major cause of morbidity and mortality among children. The sepsis-associated mortality rate decreased from 97% in 1966 to 9% among young infants in the early 1990s. Although there have been significant improvements in the treatment of sepsis and septic shock, it still remains a significant cause of childhood mortality, with more than 4,300 deaths annually. This equates to approximately 7% of all childhood mortality and an estimated annual total cost of $1.97 billion. This discussion does not apply to resource-limited settings.

Definitions
Because children differ in their response to an infectious agent, it is useful to qualify age groups so that standardized vital signs and laboratory criteria can be used. In 2005, an expert consensus panel agreed on age group definitions for pediatric patients based on observed differences in vital sign parameters and patterns of organ dysfunction.

**Table 3-5: Pediatric Age Groups for Severe Sepsis Definitions**

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>0 days to 1 week</td>
</tr>
<tr>
<td>Neonate</td>
<td>1 week to 1 month</td>
</tr>
<tr>
<td>Infant</td>
<td>1 month to 1 year</td>
</tr>
<tr>
<td>Toddler and preschool</td>
<td>2 to 5 years</td>
</tr>
<tr>
<td>School-age child</td>
<td>6 to 12 years</td>
</tr>
<tr>
<td>Adolescent and young adult</td>
<td>13 to &lt;18 years</td>
</tr>
</tbody>
</table>

to be as reliable as those from internal jugular and subclavicular sites. A mixed venous oxygen saturation (SVO₂) should be measured from direct aspiration from a central venous catheter with the tip near the right atrial and superior vena cava/inferior vena cava junction, continual electronic catheter measurement, or noninvasive spectroscopy.

Catecholamine-resistant shock can next be classified as cold shock with normal blood pressure, cold shock with low blood pressure, and warm shock with low blood pressure. See Figure 3.1 for summary and recommendations.

In cold shock with normal blood pressure, the patient has maximum vasoconstriction and an elevated SVR with a significantly depressed CO. Initial recommendations are to maximize oxygen delivery and capacity with existing CO by transfusing to a hemoglobin level greater than 10 g/dL and titrating epinephrine (adrenaline) infusion to an SVO₂ of greater than 70%. If the patient still presents with signs of poor perfusion and a normal blood pressure, decreasing afterload can improve SV by improving the myocardial contractility on the Starling curve. This can be accomplished with a vasodilator, such as sodium nitroprusside, or phosphodiesterase inhibitor, such as milrinone. Both of these types of agents are direct systemic agents, with milrinone having the added benefit of being a direct cardiac inotrope.

In cold shock with low blood pressure, the patient has a poor CO with submaximal vasoconstriction. Initial recommendations are to maximize oxygen delivery and capacity with existing CO by transfusing to a hemoglobin level greater than 10 g/dL and titrating epinephrine (adrenaline) infusion to an SVO₂ of greater than 70%. If the patient still presents with signs of poor perfusion and a low blood pressure, increasing afterload and inotropy can improve perfusion. This can be accomplished by adding norepinephrine (noradrenaline) and titrating to a normal blood pressure. If after normalization of blood pressure, the CO remains depressed, then an additional inotropic agent, such as a phosphodiesterase inhibitor (eg, milrinone) or β-adrenergic agonist (eg, dobutamine), can be used.

In warm shock with low blood pressure, the patient has a normal CO with severely decreased SVR. Initial recommendations are to titrate norepinephrine (noradrenaline) to a normal oxygen delivery or SVO₂ greater than 70%. If the patient is still hypotensive despite norepinephrine (noradrenaline) titration, then addition of a direct arteriolar vasoconstrictor to increase SVR is warranted. Vasopressin and terlipressin are direct arteriolar vasoconstrictors and act independently of catecholamines and can be administered as initial vasoconstrictors. It is important to continuously monitor CO when infusing vasoconstrictors because these agents can increase afterload significantly, thereby decreasing CO and worsening tissue perfusion.

**Refractory Shock**

Patients who display signs of poor perfusion despite achieving goals of adequate fluid resuscitation, maximum inotropic use, vasoconstrictors, transfusion, intubation, and maintenance of euglycemia and serum calcium are considered to have refractory shock. These patients should have investigations into other causes of decreased CO, including pericardial effusion, tension pneumothorax, abdominal compartment syndrome, significant bleeding, and massive pulmonary embolus. Patients without any readily reversible condition that remains in refractory shock should be considered a potential candidate for extracorporeal membrane oxygenation. The risks of extracorporeal membrane oxygenation and its associated mortality in the neonatal (80%) and pediatric (50%) populations should be weighed against its potential benefit in conjunction with your pediatric intensivist.

**Septic Shock in Resource-Limited Settings (no mechanical ventilation, no inotropic support)**

Studies have demonstrated improved survival with the use of maintenance fluids alone compared to those who received 20 to 40 mL/kg in the first hour of therapy. In this setting, an initial fluid bolus of 20 mL/kg of crystalloid is reasonable. Administration of additional fluid boluses should occur with extreme caution. The patient should be reassessed after every fluid bolus.

**Septic Shock: Neonatal Considerations**

Neonatal septic shock usually occurs as a result of perinatally acquired bacteremia. A history of chooroamnionitis, maternal fever, prolonged rupture of the membranes, and active genital herpes simplex virus lesions at time of delivery put neonates at higher risk for developing neonatal sepsis.

The most common causative organisms are *Streptococcus agalactiae* (Group B Streptococci), *Group B* *Streptococcus*, and *Group B* *Staphylococcus*. Some neonates are at greater risk for developing sepsis and may therefore require earlier initiation of therapy.
8. What is the role of endotracheal intubation and mechanical ventilation in the treatment of children with shock?
   A. Up to 40% of cardiac output might be required to support the work of breathing, and this can be unloaded by ventilation, diverting flow to vital organs.
   B. Decreased intrathoracic pressure also reduces left ventricular afterload that might be beneficial in patients with low cardiac output and high systemic vascular resistance.
   C. Aggressive hyperventilation might be required to compensate for metabolic acidosis by altering the respiratory component of acid-base balance.
   D. Temperature control is prevented and oxygen consumption increased.

References

Shock

CHAPTER REVIEW


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epinephrine (adrenaline) at 0.01 mg/kg (0.1 mL/kg of 0.1 mg/mL solution) or tracheal (endotracheal) 0.1 mg/kg (0.1 mL/kg of 1 mg/mL solution). Repeat administration of epinephrine (adrenaline) every 3 to 5 minutes. If bradycardia persists or transiently responds, consider an epinephrine (adrenaline) infusion. Atropine can be used to treat bradydysrhythmias if there is a suspicion of increased vagal tone, primary AV block, or cholinergic drug toxicity. The recommended dose is 0.02 mg/kg, with a minimum dose of 0.1 mg and a maximum single dose of 0.5 mg in a child and 1 mg in an adolescent. This can be repeated in 5 minutes to a maximum total dose of 1 mg in a child and 2 mg in an adolescent. Atropine can be administered tracheally (endotracheally), if IV or IO access cannot be obtained (0.04 to 0.06 mg/kg).24 Transcutaneous pacing can also be considered in those cases of bradycardia from a congenital or acquired heart disease that has caused a complete heart block or sinus node dysfunction24 (Figure 4.13).

<table>
<thead>
<tr>
<th>TABLE 4-3 Reversible Causes of Pediatric Cardiac Arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Six Hs</strong></td>
</tr>
<tr>
<td>Hypovolemia</td>
</tr>
<tr>
<td>Hypoxia</td>
</tr>
<tr>
<td>Hydrogen ion (acidosis)</td>
</tr>
<tr>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Hypo-/ hyperkalemia</td>
</tr>
<tr>
<td>Hypothermia</td>
</tr>
</tbody>
</table>


Tachydysrhythmias

Sinus tachycardia is not a dysrhythmia but rather a response by the body to increased cardiac output, so the underlying cause should be identified and treated. Common causes include hypoxemia, hypovolemia, hyperthermia, fever, toxins, poisons, drugs, pain, and anxiety. Supraventricular tachycardia is the most common tachydysrhythmia in children and can produce cardiovascular compromise. The heart rate is usually greater than 220/min but can reach as high as 300/min. Usually the QRS interval is narrow or normal (≤0.09 seconds). The cause is most commonly a reentry mechanism from an accessory pathway. It usually occurs quickly and without a history of volume loss, pain, or fever to suggest sinus tachycardia. In many instances, it might be difficult to distinguish between sinus tachycardia and SVT (Table 4-4). If the child is stable and has good perfusion, the treatment of choice is mechanical vagal interventions. If this is unsuccessful, then IV adenosine

<table>
<thead>
<tr>
<th>TABLE 4-4 Comparison of Sinus Tachycardia (ST) and Supraventricular Tachycardia (SVT)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ST</strong></td>
</tr>
<tr>
<td>History of underlying problem, such as fever, dehydration, injury, or pain</td>
</tr>
<tr>
<td>P waves present</td>
</tr>
<tr>
<td>Heart rate varies with activity</td>
</tr>
<tr>
<td>Variable R-R interval with respirations</td>
</tr>
<tr>
<td>Abrupt rate changes (with conversion)</td>
</tr>
<tr>
<td>Infants: heart rate &lt;220/min</td>
</tr>
<tr>
<td>Children: heart rate &lt;180/min</td>
</tr>
</tbody>
</table>

Pediatric Bradycardia
With a Pulse and Poor Perfusion

1. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IO/IV access
   - 12-Lead ECG if available; don’t delay therapy

2. Cardiopulmonary compromise continues?
   No
   - CPR if HR<50/min with poor perfusion despite oxygenation and ventilation

3. CPR if HR<50/min with poor perfusion despite oxygenation and ventilation
   Yes
   - Cardiopulmonary Compromise
     - Hypotension
     - Acutely altered mental status
     - Signs of shock

4a. Support ABCs
   - Give Oxygen
   - Observe
   - Consider expert consultation

4. Bradycardia persists?
   No
   - Do nothing
   Yes
   - Epinephrine
     - Atropine for increased vagal tone or primary AV block
     - Consider transthoracic pacing/transvenous pacing
     - Treat underlying causes

5. If pulseless arrest develops, go to Cardiac Arrest Algorithm

Doses/Details
Epinephrine IO/IV Dose:
0.01mb/kg (0.1 mL/kg of 0.1 mg/mL concentration).
Repeat every 3-5 minutes.
If IO/IV access not available but endotrachial (ET) tube in place, may give ET dose:
0.1 mg/kg (0.1 mL/kg of 1 mg/mL).

Atropine IO/IV Dose:
0.02 mg/kg. May repeat once.
Minimum dose 0.1 mg and maximum single dose 0.5 mg.

Figure 4.13  Pediatric advanced life support bradycardia algorithm.


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Pediatric Cardiac Arrest Algorithm—2015 Update

1. Start CPR
   - Give oxygen
   - Attach monitor/defibrillator

2. Yes
   - Rhythm shockable?

3. Shock
   - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

4. No
   - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

5. Shock
   - CPR 2 min
     - Amiodarone or lidocaine
     - Treat reversible causes

6. No
   - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

7. Shock
   - CPR 2 min
     - Amiodarone or lidocaine
     - Treat reversible causes

8. No
   - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

9. Asystole/PEA
   - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

10. No
    - CPR 2 min
     - IO/IV access
     - Epinephrine every 3-5 min
     - Consider advanced airway

11. CPR 2 min
    - IO/IV access
    - Epinephrine every 3-5 min
    - Consider advanced airway

12. Asystole/PEA → 10 or 11
    - Organized rhythm → check pulse
    - Pulse present (ROSC) → post-cardiac arrest care

CPR Quality
- Push hard (≥⅓ of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 15:2 compression-ventilation ratio.

Shock Energy for Defibrillation
First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy
- **Epinephrine IO/IV dose**: 0.01 mg/kg (0.1 mL/kg of 0.1 mg/mL concentration). Repeat every 3-5 minutes.
  - If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1 mg/mL concentration).
- **Amiodarone IO/IV dose**: 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.
- **Lidocaine IO/IV dose**: Initial: 1 mg/kg loading dose. Maintenance: 20-50 mcg/kg per minute infusion (repeat bolus dose if infusion initiated >15 minutes after initial bolus therapy).

Advanced Airway
- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypo-hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

**Figure 4.15** Pediatric advanced life support cardiac arrest algorithm.

and/or the presence of severe acidosis (pH < 6.9). A bedside glucose test (rapid test), arterial blood gas analysis, and measurement of electrolytes are important to guide resuscitation.

Once the resuscitation has begun, consider potentially reversible causes. Again, the mnemonic of six Hs and five Ts includes the following: hypoxemia, hypovolemia, hypothermia, hyper/hypokalemia, hydrogen ion (acidosis), hypoglycemia, tamponade, toxins/poisons/drugs, tension pneumothorax, thrombosis (coronary), and thrombosis (pulmonary).24

**Management**

For cardiopulmonary arrest, the first priority is to shout for help, start chest compressions at a rate of at least 100 per minute, and ventilate with 100% oxygen via bag mask. The rate for children less than 8 years is 30 compressions to two ventilations for a single rescuer and 15:2 for two rescuers. For those over 8 years it is 30 compressions to two ventilations. If the patient has an advanced airway in place, the ventilations should be given at 8 to 10 breaths per minute with continuous chest compressions.24 A monitor/defibrillator or an automated external defibrillator should be attached as soon as possible. The cardiac activity or the effectiveness of chest compressions (measure blood pressure, perfusion, and oxygen saturation) should be monitored and the rhythm assessed. If the rhythm is shockable (ventricular fibrillation/VT), shock or defibrillation should be performed at 2 J/kg and cardiopulmonary resuscitation (CPR) immediately resumed. Either IV or IO access should be obtained during the next five cycles (2 minutes) of CPR, then the rhythm checked. If shockable, CPR should be continued while the automated external defibrillator or monitor/defibrillator is charging, and the patient should be shocked or defibrillated at 4 J/kg. Cardiopulmonary resuscitation should be resumed immediately. During this next cycle of CPR, epinephrine (adrenaline) (0.01 mg/kg IV/IO of 0.1 mg/mL concentration) should be administered and this dose repeated every 3 to 5 minutes. If there is no IV/IO access, but the patient is intubated, 0.01 mg/kg of a 1 mg/mL concentration of epinephrine (adrenaline) should be administered by the endotracheal tube. When five cycles (2 minutes) are complete, the rhythm should be checked and, if shockable, the patient should be shocked or defibrillated with 4 J/kg and CPR resumed. Amiodarone (5 mg/kg IV/IO bolus) or lidocaine (1 mg/kg) therapy should be considered and administered while CPR is provided for five cycles; the rhythm should then be checked. If rhythm is still shockable, the CPR-drug-shock cycle should be repeated with epinephrine (adrenaline) and the patient shocked or administered a 4-J/kg defibrillation energy dose. Amiodarone can be repeated up to two times in this cycle or lidocaine can be maintained at an infusion of 20 to 50 mcg/kg/min (repeat bolus dose if infusion > 15 min after initial bolus therapy).24

If the rhythm shows PEA or asystole (non-shockable rhythm), CPR should be continued for 2 minutes, IV/IO access obtained, and intubation considered. Epinephrine (adrenaline) (0.01 mg/kg IV/IO of 0.1 mg/mL concentration or 0.01 mg/kg endotracheally of 1 mg/mL concentration) should be administered and CPR resumed. Epinephrine (adrenaline) can be repeated every 3 to 5 minutes. High-dose epinephrine (adrenaline) can be considered in β-blocker overdose.24 If at any time there is electrical activity, the pulse should be checked. If a pulse is present, postresuscitation care should begin. If a pulse is absent, CPR should be continued and the asystole/PEA pathway followed.24

Termination of resuscitation is a critical issue. Studies show little to no meaningful

**THE BOTTOM LINE**

- Treatment of patients with dysrhythmias is driven by the presence or absence of poor perfusion.
- Sinus tachycardia is not an arrhythmia, but its cause must be determined.
- Provide oxygenation and ventilation for all patients in cardiopulmonary arrest as the primary cause is respiratory failure.
- Once resuscitation has begun, evaluate and treat reversible cause of cardiopulmonary arrest.
- Family presence during resuscitation should be encouraged by the ED staff.
important to consider. A mother with diabetes is at increased risk for delivering an infant who is large for his or her gestational age who is at risk for hypoglycemia. A mother who is febrile, has a tender uterus and/or foul smelling discharge, and has a fetus who is tachycardic will likely deliver an infant who will need to be evaluated and treated for suspected neonatal sepsis. If narcotics are given to the laboring mother for analgesia during delivery, the infant will be at risk of respiratory depression.

**Preparation of Equipment**
Gathering and preparing the necessary equipment before delivery will facilitate a successful resuscitation (Table 11-2).

**Environment**
The first thing to identify is where the newborn will be placed once delivery has occurred. Ideally, this would be on a radiant warmer that provides the newborn with an exogenous source of heat. More commonly, the newborn can be placed on an examining table or crib sufficient for evaluation and resuscitation, ensuring that the wet newborn is kept warm and does not fall. Temperature maintenance can be facilitated with warm blankets and dry towels and warming the temperature of the room if possible, particularly if the newborn is anticipated to be preterm.

Efforts to evaluate and resuscitate the newborn in close proximity to an oxygen source and suction will enhance the ability to provide optimal care. Additional equipment that might be necessary includes a blended oxygen source with the ability to provide oxygen concentrations from room air to 100% and an oxygen saturation monitor with a probe that should be placed on the newborn's right wrist or hand to measure the oxygen saturation of preductal blood.

**Airway Management**
If there is evidence of airway obstruction or positive pressure ventilation is required, it will be necessary to clear the airway. Equipment that can be used includes a bulb syringe or wall suction device and a number of commercially available suction catheters appropriately sized for the newborn’s mouth and upper airway. If the newborn requires positive pressure ventilation (PPV), an appropriately sized face mask connected to a device capable of delivering PPV will be needed. These devices include an infant-sized self-inflating bag, a flow inflating bag, or mechanical resuscitator or ventilator. If the newborn requires endotracheal intubation, appropriately sized laryngoscope blades and endotracheal tubes will be needed. For the newborn with difficult airway access, a laryngeal mask airway can offer airway support if bag-mask ventilation is ineffective and endotracheal intubation is difficult. The smallest size of laryngeal mask airway devices currently available restricts their use primarily to infants greater than approximately 2.0 kg.

An exhaled carbon dioxide detector is recommended to confirm appropriate placement of an endotracheal tube. The endotracheal tube should be secured in place at the correct depth of insertion. A special consideration is the need for a meconium aspirator for the infant who requires airway clearance beyond the vocal cords.

**TABLE 11-2  Equipment for Neonatal Resuscitation**

<table>
<thead>
<tr>
<th>Environment</th>
<th>• Radiant warmer, crib, examination table</th>
<th>• Heat source, blankets, towels</th>
<th>• Increase room temperature if feasible</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen administration and monitoring</td>
<td>• Source for adjustable blended oxygen (room air to 100%)</td>
<td>• Oxygen saturation monitoring</td>
<td>• Neonatal oxygen saturation probe to place on right wrist or hand</td>
</tr>
<tr>
<td>Airway management</td>
<td>• Suction source (bulb suction, suction catheters)</td>
<td>• Appropriately sized facial masks for bag-mask ventilation</td>
<td>• Source for positive pressure ventilation (self-inflating bag, flow inflating bag, T-piece Resuscitator or ventilator)</td>
</tr>
<tr>
<td></td>
<td>• Appropriately sized laryngoscope blade (00, 0, 1 Miller blade)</td>
<td>• Appropriately sized endotracheal tubes (2.5, 3.0, 3.5, 4.0)</td>
<td>• Laryngeal mask airway</td>
</tr>
<tr>
<td></td>
<td>• Exhaled carbon dioxide detector or monitor</td>
<td>• Tape</td>
<td>• Stethoscope</td>
</tr>
</tbody>
</table>
YOUR FIRST CLUE

Signs and Symptoms of a High-Risk Delivery

**Is this delivery high-risk?**
- Maternal report of multiple gestations (e.g., twins)
- Maternal report of preterm gestation or fetal anomalies
- Maternal illness of possible consequence to fetus (e.g., diabetes mellitus)
- Presence of meconium-stained amniotic fluid
- Fetal heart rate abnormalities (e.g., decelerations, bradycardia, or tachycardia)

**Does this newborn require intervention?**
- Impaired respiratory effort (e.g., gasping, apnea, labored breathing)
- Bradycardia
- Poor muscle tone (e.g., limp, flaccid)

**Is this neonate at risk for hypoglycemia?**
- Small for gestational age
- Premature
- Infant of a diabetic mother
- Infection
- Hypothermia
- Tremulousness
- Seizures

**Is this neonate at risk for infection?**
- Prematurity
- Maternal colonization with group B Streptococcus
- Maternal infection (e.g., urinary tract infection, chorioamnionitis)
- Respiratory distress
- Poor perfusion
- Seizure

---

**Healthy Newborn**

The term gestation newborn who has good muscle tone and activity level and is breathing spontaneously should receive routine care. Universal suctioning of the newborn’s mouth once delivered is no longer considered mandatory. It is recommended that suctioning immediately after birth should be reserved for newborns who have obvious obstruction to spontaneous breathing or require ventilatory support with positive pressure. Newborns are born wet and will experience heat loss rapidly in the absence of a heat source. For the vigorous, well-appearing infant, this could be prevented by allowing the infant, once dried off, to be held by the mother skin to skin. Ongoing evaluation of risk factors (potential infection, stable blood glucose) should be pursued. Stimulating a newborn can include slapping or flicking of the soles of the feet or gentle rubbing of the newborn’s back, trunk, or extremities during the drying process. Delayed cord clamping for longer than 30 seconds is reasonable for those term and preterm infants who do not require resuscitation.

---

**Immediate Care of the Newborn**

**Primary Assessment**

Most newborns transition to extrauterine life without significant distress. Once born, all newborns require an assessment of risk for requiring resuscitation. The primary assessment of a newborn includes an evaluation of the infant's respiratory effort and muscle tone (**Figure 11.1** and **Table 11-3**).

---

**Your First Clue**

Risk Factors for Pathologic Jaundice

- Excessive weight loss or inadequate weight gain since birth
- Parental report of decreased oral intake
- Parental report of decreased urine or stool output
- Signs of dehydration (e.g., tachycardia, dry mucous membranes)
- Signs of infection (e.g., fever, poor perfusion)
- Pallor
- Depressed sensorium

---

**Immediate Care of the Newborn**

**Primary Assessment**

Most newborns transition to extrauterine life without significant distress. Once born, all newborns require an assessment of risk for requiring resuscitation. The primary assessment of a newborn includes an evaluation of the infant's respiratory effort and muscle tone (**Figure 11.1** and **Table 11-3**).
During these interventions, if respiratory support or supplemental oxygen is considered necessary, a team member should place a pulse oximetry probe on the newborn’s right hand or wrist to enhance monitoring oxygen saturation and heart rate. Application of an oxygen saturation probe can take up to 2 minutes and might not function well during poor cardiac and resuscitation. During transfer, it is essential to support the newborn’s head. There must be careful placement and attention to the position of the head and neck. The correct position of the newborn’s head and neck is slight extension in the “sniffing” position. Hyperextension or flexion of the neck should be avoided (Figure 11.2).

The initial evaluation of the newborn should include an assessment of the newborn’s respiratory effort, muscle tone, and heart rate. If there is decreased respiratory effort (gasping, apnea) or if the heart rate is less than 100/min, the newborn should have the airway cleared and positive pressure ventilation initiated. If an infant has labored breathing or persistent cyanosis, the airway should be positioned and cleared, and supplementary oxygen given as needed.

During these interventions, if respiratory support or supplemental oxygen is considered necessary, a team member should place a pulse oximetry probe on the newborn’s right hand or wrist to enhance monitoring oxygen saturation and heart rate. Application of an oxygen saturation probe can take up to 2 minutes and might not function well during poor cardiac...
output. Direct auscultation of the precordium or palpation of the heart rate by gently squeezing the base of the umbilicus can also provide additional heart rate data (Table 11-4).

The Neonatal Resuscitation Algorithm–2015 Update serves as a useful tool for further assessment of the newborn (Figure 11.3).

**TABLE 11-4 The Three Vital Signs to Monitor During Neonatal Resuscitation**

1. Heart rate
2. Respirations
3. Oxygen saturation

**Neonatal Resuscitation Algorithm—2015 Update**

![Neonatal Resuscitation Algorithm—2015 Update](image)

**Figure 11.3** The Neonatal Resuscitation Algorithm—2015 Update.


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Neonatal Emergencies

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**Oxygenation Assessment**

Included in the *Textbook of Neonatal Resuscitation* 6th edition resuscitation algorithm is a table that indicates the target preductal (right hand) oxygen saturation in newborns. The uncompromised term newborn will transition from fetal circulation to extrauterine circulation during the first 10 minutes of life. It is normal for the uncompromised newborn to have an oxygen saturation in the range of 60% to 70% in the first minute of life. During this transition in circulation, using patient color as an assessment of the newborn’s well-being is unreliable.

There is a growing body of literature that supports the harmful effect of either insufficient or excessive oxygen delivery to the newborn. To properly assess the need for supplemental oxygen or whether the resuscitation of the newborn will involve the administration of supplemental oxygen or PPV, an oxygen saturation probe should be placed on the right wrist or surface of the right palm.

It is recommended that the saturation of newborns being resuscitated at birth be targeted at the mean preductal saturations demonstrated by healthy term newborns after vaginal birth at sea level. Achievement of such targets can be best achieved by administering an adjustable blend of oxygen and air titrated to established preductal normal values. If blended oxygen is not available, resuscitation should begin with air (21% oxygen) for term infants. For preterm infants (<35 wk), resuscitation should be initiated with low oxygen concentration (21 to 30% oxygen) titrated to achieve preductal oxygen saturation. If oximetry is not available, the concentration of oxygen should be adjusted to achieve relief of cyanosis and correction of bradycardia. Bradycardia will generally require PPV and will not improve with supplemental oxygen alone (Table 11-5).

**Positive Pressure Ventilation**

If the infant requires resuscitation beyond warming, drying, stimulation, and clearance of the airway, the algorithm suggests application of PPV. Effective PPV requires the selection and placement of the appropriately sized facial mask (Figure 11.4).

The primary indicator of effective PPV resuscitation is improvement in heart rate. If no improvement in heart rate is observed during positive pressure, assessment of chest rise is recommended. The initial peak inspiratory pressure is variable. Some newborns will experience an improvement in heart rate and chest rise with peak inspiratory pressures of 20 cm H₂O, whereas other compromised apneic term newborns can require up to 40 cm H₂O to establish effective ventilation. The initial assisted

---

**TABLE 11-5 Indications for Oxygen Saturation Monitoring**

1. Resuscitation is anticipated based on risk factors (e.g., prematurity).
2. PPV is administered for more than a few breaths.
3. Cyanosis is persistent.
4. Supplemental oxygen is administered.
Endotracheal Tube Placement

Endotracheal intubation might be indicated during neonatal resuscitation. Indications for endotracheal intubation are included in the NRP resuscitation algorithm. In addition, if the newborn requires prolonged mask ventilation or was born nonvigorous through thick meconium, endotracheal intubation is recommended. Meconium should be suctioned from the trachea, using an endotracheal tube and meconium aspirator, if the newborn is meconium stained, is nonvigorous, or has a persistent bradycardia.

Selecting the appropriately sized endotracheal tube is essential for successful and safe insertion. The proper tube size and insertion depth are dependent on the newborn’s birth weight and gestational age (Table 11-7).

TABLE 11-6 Troubleshooting Ineffective Ventilation

| 1. Is the mask appropriately sized and positioned correctly? |
| 2. Is there a tight seal between the mask and face? |
| 3. Is there airway obstruction? |
| 4. Is the head positioned slightly hyperextended? |
| 5. Is adequate pressure being delivered? |
| 6. Does the self-inflating bag have a PEEP valve? |

Laryngeal Mask Airway

A laryngeal mask airway (LMA) has been shown to be effective for assisted ventilation of newborns who weigh more than 2 kg or are delivered at 34 weeks’ gestation or later.5–7 The appropriately sized device in this population is a size 1 neonatal. Laryngeal mask airways have not been studied for the use during the resuscitation of the newborn delivered through meconium-stained amniotic fluid or for the administration of medications into the trachea. However, there are indications for the use of LMA in this population:

- Congenital anomalies involving the mouth, lip, or palate resulting in difficulties in obtaining a proper seal with face mask ventilation (eg, cleft lip or palate).
- Anomalies of the mouth, tongue, pharynx, or neck where there is difficulty visualizing the larynx with a laryngoscope (eg, cystic hygroma of neck or Beckwith-Wiedemann syndrome and macroglossia).
- A very small mandible or relatively large tongue (eg, Robin sequence).
- PPV provided by bag and mask is ineffective and attempts at intubation are not feasible or are unsuccessful.

Similar to assisted PPV with mask and bag, an improvement in heart rate is the best indicator that the endotracheal tube is in the proper position and effective ventilation is occurring. Tracheal intubation is best confirmed with exhaled carbon dioxide detection. Cyclic rise of condensation in the endotracheal tube, chest rise, and auscultation of symmetric breath sounds are also helpful signs.

Intubation of the newborn can be a high-acuity, low-frequency event at certain locations. If the practitioner is unfamiliar with the unique challenges of intubating a newborn, airway backup should be called for, and effort should certainly be focused on providing effective PPV with face mask and device instead of spending

TABLE 11-7 Endotracheal Tube Size for Infants of Various Weights and Gestational Ages

<table>
<thead>
<tr>
<th>Weight (grams)</th>
<th>Gestational Age (weeks)</th>
<th>Tube Size (Inside Diameter mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1,000</td>
<td>&lt;28</td>
<td>2.5</td>
</tr>
<tr>
<td>1,000–2,000</td>
<td>28–34</td>
<td>3.0</td>
</tr>
<tr>
<td>2,000–3,000</td>
<td>34–38</td>
<td>3.5</td>
</tr>
<tr>
<td>&gt;3,000</td>
<td>&gt;38</td>
<td>3.5–4.0</td>
</tr>
</tbody>
</table>

WHAT ELSE?

Causes of Respiratory Distress in Newborns
- Transient tachypnea of the newborn
- Respiratory distress syndrome (hyaline membrane disease)
- Meconium aspiration
- Bacterial infection (eg, sepsis, pneumonia)
- Viral infections (eg, respiratory tract infection, sepsis)
- Pneumothorax
- Congenital diaphragmatic hernia
- Congenital cystic adenomatoid malformation of the lung
- Pulmonary sequestration
- Tracheoesophageal fistula
- Pleural effusion
- Choanal stenosis or atresia
- Macroglossia
- Retroglossalia
- Laryngeal malformations (eg, laryngomalacia, vocal cord paralysis)
- Tracheobronchial malformations (eg, tracheomalacia, bronchial cysts)
- Mediastinal masses (eg, teratoma)
- Congenital heart disease (eg, obstructed pulmonary veins)
- Metabolic disorders (eg, hyperammonemia)
- Central nervous system malformations (eg, hydrocephalus)
- Neonatal abstinence syndrome
- Intentional trauma

Chest Compressions
If the heart rate remains below 60/min despite adequate PPV, chest compressions are recommended. It is important to reinforce that before initiation of chest compressions, practitioners should ensure that assisted ventilation is being delivered optimally. The preferred technique in the newborn for delivery of higher peak systolic pressures and coronary perfusion pressure is the two-thumb technique. This technique involves encircling the newborn’s torso with both hands. The thumbs are placed on the sternum and the fingers are placed under the infant’s back, supporting the spine. The thumbs can be placed side by side or, on a small infant, one over the other (Figure 11.5).

Compressions and ventilations should be coordinated to avoid simultaneous delivery. The chest should be permitted to fully reexpand during relaxation. There should be a 3:1 ratio of compressions to ventilations with 90 compressions and 30 breaths to achieve approximately 120 events per minute to maximize ventilation at an achievable rate. It is recommended that 3:1 be used for neonatal resuscitation, but rescuers should consider using higher ratios (15:2) when the arrest is believed to be of cardiac origin.

Medications
Epinephrine (Adrenaline)
There are indications within the NRP algorithm for the administration of epinephrine (adrenaline). It is important to remember that most cases of bradycardia in the newborn are secondary to inadequate lung inflation or hypoxemia. If these interventions are ineffective at improving the heart rate, administration of epinephrine (adrenaline) might be warranted.

The preferred route of administration of epinephrine (adrenaline) is intravenous. If it appears that the infant might need epinephrine (adrenaline), the practitioner should obtain venous access, usually by cannulating the umbilical vein. If administration of epinephrine (adrenaline) is indicated before obtaining venous access, an endotracheal dose can be administered. The dose of endotracheal epinephrine (adrenaline) of 0.05 to 0.1 mg/kg is recommended. This is a higher dose than had previously been recommended and remains higher than the recommended intravenous dose. Once venous access has been obtained, epinephrine (adrenaline) can be given with a recommended dose of 0.01 to 0.03 mg/kg per dose. The concentration of epinephrine (adrenaline) for either route should be 0.1 mg/mL.
Neonatal Emergencies

Volume Expansion
If the newborn has not responded to other resuscitation efforts or if the patient is hypotensive with poor perfusion, volume expansion with normal saline or blood can be considered. The starting dose is 10 mL/kg and can be repeated.

Special Considerations
Meconium-Stained Amniotic Fluid
Passage of meconium while in utero can contribute to meconium aspiration syndrome with dark, viscous material aspirated into the terminal airspaces. The approach to the newborn delivered through meconium-stained amniotic fluid has evolved over time. The current recommendation is that if a newborn is delivered through meconium-stained amniotic fluid and has poor tone and respiratory effort at birth, the initial steps of resuscitation should occur under the radiant warmer. If the heart rate is less than 100 bpm or the infant is not breathing, positive pressure ventilation should be initiated. Routine intubation for tracheal suctioning is not recommended.4a
Induced Hypothermia
Several trials of hypothermia of newborns of 36 weeks’ gestation and later with moderate to severe hypoxic ischemic encephalopathy revealed that the newborns who were cooled within the first 6 hours of life had significantly less death and neurodevelopmental disability at 18-month follow-up.8–10 If a newborn requires extensive resuscitation and has low Apgar scores during resuscitation, prompt consultation with a local tertiary care neonatal intensive care unit would be appropriate to decide whether hypothermia treatment should be initiated.

Discontinuing Resuscitative Efforts
In a newborn with an Apgar score of 0 after 10 minutes of resuscitation and no detectable heart rate, it may be reasonable to stop assisted ventilation; however, the decision to continue resuscitation efforts beyond 10 minutes should take into consideration factors such as the presumed cause of the arrest, the gestation of the newborn, the presence or absence of complications, the potential role of therapeutic hypothermia, and the parents’ consent and request for continued resuscitation.4a

Preparing the Newborn for Transfer From the Emergency Department
Once the primary assessment and resuscitation of a newborn have been stabilized in the emergency department, steps must be taken to

Figure 11.5 Thumb technique of chest compressions administered from the bottom (A), from the top (B), and for small chests, with thumbs overlapped (C).
maintain the infant’s stability before and during transfer to an appropriate nursery. Whether the newborn is healthy and full term or exhibits symptoms and signs warranting intensive care, one must consider the basic needs of a newborn for an appropriate degree of warmth and an adequate delivery of glucose and fluids. For infants who require resuscitation or who show signs of instability after delivery, infectious disease should be suspected and treated expeditiously. Jaundice can be identified at birth or in a days-old newborn who presents for evaluation in the emergency department, so familiarity with basic principles of management is essential. Umbilical vascular catheter placement and thoracostomy drainage of a pneumothorax are sometimes required in the treatment of the critically ill newborn.

**Temperature**

Although there is no consensus for what temperature should be considered “normal” for a newborn infant, there are published ranges that practitioners can use to guide their postresuscitation care of the newborn in the emergency department. The American Academy of Pediatrics recommends that an axillary temperature of 36.5° to 37.4°C (97.7° to 99.3°F) should be obtained to safely discharge an infant home,11 so this range could be considered a safe and acceptable range to target when preparing a newborn for transfer. Measurement of the temperature in the central axilla with the arm held to the infant’s side allows for rapid and easy measurement of the temperature without the risks associated with rectal measurements.

Maintaining the temperature of a well, term, or near-term newborn consists of replacing the wet linens used during the resuscitation with warmed, dry linens. Swaddling the newborn from the level of the shoulders down protects the newborn from heat loss while keeping the face free of obstructions. An appropriately sized cap can be placed over the head to limit heat loss. For those newborns who require frequent assessment and intervention, continuous visible assessment of the skin must be countered with any of several measures to limit heat loss. Careful attention to drying the exposed newborn will limit evaporative heat loss. Increasing the ambient temperature of the newborn resuscitation room and limiting drafts will minimize convective heat loss. Periodic replacement of cold linens with warmed linens can prevent some degree of conductive heat loss, and a radiant source of heat can mitigate radiant heat loss as well.

**Glucose**

The energy needs of a newborn reflect a balance between energy expenditure and energy stores on which the infant can draw. Factors that increase energy expenditure (eg, thermogenesis in response to cold stress, increased metabolic rate in the setting of sepsis) and certain characteristics of the newborn (eg, small for gestational age, prematurity) can be present in a newborn delivered in the emergency department, so it is essential for emergency personnel to identify and manage these problems appropriately. Providing intravenous dextrose is often sufficient to overcome the deficits present in a newborn, so a basic knowledge of neonatal glucose needs is helpful in preparing for these emergencies.

Glucose is the principal energy source for the fetus and newborn, with most neonates consuming glucose at a rate of 4 to 8 mg/kg per minute.12 In neonates, most of circulating glucose is consumed by the brain, so prompt provision of exogenous glucose is essential to optimize a newborn’s chance for normal neurodevelopment. For any newborn that requires an advanced level of care, placement of a peripheral intravenous catheter should be followed by the administration of dextrose sufficient to meet the typical needs of a newborn. For example, a simple 10% dextrose solution run at 4 mL/kg per hour would provide a dextrose infusion rate of approximately 7 mg/kg per minute. Fluids can be titrated to maintain a blood glucose level in the normal range. If the newborn is noted to be hypoglycemic, a bolus of 2 mL/kg of 10% dextrose is indicated, and an increase in the basal glucose delivery should be considered. A repeat blood glucose measurement should be obtained with accompanying interventions until the newborn’s glucose level is normal.
Factors that increase the likelihood of infection in the newborn (e.g., prematurity, maternal infection), and these can be elucidated from the maternal medical and obstetric history and newborn physical examination. Acute infectious disease in the newborn most often is caused by bacterial pathogens, the identity of which can be suspected based on the timing of presentation. Perinatal viral infections also can present acutely in the newborn, so these less common but aggressive pathogens must be considered.

Early-onset bacterial sepsis is generally considered to occur during the first 3 days of life. Term infants account for most cases, but the likelihood of infection is greater in premature infants. Gram-negative bacteria now account for most early-onset infections, although the gram-positive group B Streptococcus remains another common pathogen. These infections can present in a florid manner with circulatory, respiratory, and neurologic signs, but many newborns present initially with limited, nonspecific symptoms (e.g., lethargy or intermittent apnea). As a consequence, careful review of the maternal history and critical assessment of the neonate in the emergency department is essential to prevent undiagnosed and untreated early-onset sepsis. Any risk factor or sign of infection should prompt the physician to order a complete blood cell count with differential white blood cell count, blood culture, and lumbar puncture with cerebrospinal fluid differential cell count, chemical analyses, and culture. Radiography of the chest should be ordered to assess for pneumonia. Parenteral antibiotic therapy with ampicillin and gentamicin should be instituted without delay and, when indicated, can precede the blood culture if obtaining an adequate blood sample proves difficult.

Late-onset bacterial sepsis occurs beyond the third day of life and most often is caused by gram-negative rods and group B Streptococcus. These infections are more likely to present with variable symptoms, but a fulminant presentation can be seen as well. The diagnostic workup includes the above laboratory and radiographic studies but, in addition, urine samples should be obtained for analysis and culture. In late-onset bacterial disease in particular, the urinary tract can be the site of infection.
primary site of bacterial infection or it can be secondarily infected after hematogenous spread of the pathogen. In either case, the pathogen and its antibiotic sensitivity profile can be identified rapidly from the urine. Collection of the urine sample should occur under sterile conditions via urethral catheterization or suprapubic aspiration and not by bag collection (because of the possibility of contamination). As with early-onset sepsis, ampicillin and gentamicin are appropriate for empiric antibiotic coverage.

Perinatal or congenital viral infections can present with profound circulatory, respiratory, and neurologic compromise in the newborn. Although these are most often acquired in utero or during parturition, affected neonates typically do not present until late in the first week of life. Although several viruses are known pathogens in neonates, herpes simplex virus (HSV) merits special consideration in this chapter. Neonates with systemic HSV are by far most likely to have acquired their infection during delivery from a mother with a primary HSV outbreak. As a consequence, there might be no history of prior maternal HSV infection, or the mother might report that vesicles were identified at the time of delivery. Systemic HSV in the neonate must be considered based on the timing and mode of presentation (common signs include fever, seizures, and cardiorespiratory compromise). The virus can be detected in many bodily fluids, including blood, urine, and cerebrospinal fluid, and culture of conjunctivae, nasopharynx, and rectum can yield virus as well. Prompt provision of parenteral acyclovir is essential in suspected cases and, as is the case with antibacterial agents, initiation of therapy should not be delayed in the event that diagnostic samples have been difficult to obtain. To prevent spread of the virus to health care workers in the emergency department, contact isolation should be instituted in cases of suspected disseminated HSV infection.

**Jaundice**

Approximately half of all term neonates are jaundiced to some degree during the first week of life. Most of these neonates are well, with levels of unconjugated bilirubin that pose no threat to their neurodevelopment. Nevertheless, any neonate presenting to the emergency department for evaluation of jaundice must be carefully assessed by history, physical examination, and laboratory studies to ensure that pathologic jaundice is not present.

Historical findings that would suggest that a neonate is at risk for unsafe levels of unconjugated bilirubin include excessive weight loss or inadequate weight gain since the time birth, poor feeding per parental report, and limited urine or stool output. A physical examination revealing signs of dehydration or infection, pallor (suggesting hemolysis), or depressed sensorium should prompt the clinician to suspect that an infant’s jaundice can be pathologic and not an incidental finding. More than historical or physical examination findings, however, laboratory evaluation is of critical importance in assessing jaundice in the neonate.

Laboratory testing of the jaundiced neonate must include both total and direct serum bilirubin levels. Although it is the unconjugated (indirect) form of bilirubin that is associated with bilirubin encephalopathy or kernicterus, it is important to exclude hepatobiliary causes of jaundice by assessing the conjugated (direct) fraction of serum bilirubin. The American Academy of Pediatrics has established guidelines for managing total serum bilirubin levels in term and near-term neonates, and this tool is valuable when deciding whether or when a neonate must be seen again by a clinician or whether inpatient hospitalization would be required for phototherapy and additional laboratory studies (*Figure 11.6*). Unless this information is already available to the emergency department clinician, the blood type of both the mother and neonate should be determined and a direct antiglobulin (Coombs) test performed on the neonate's blood. This information, coupled with the neonate's hematocrit, can provide evidence that hemolysis is contributing to the jaundice. In such cases, consultation with a local expert in neonatal care is recommended.

**Umbilical Vascular Catheter Placement**

Placement of umbilical vascular catheters can be indicated in the treatment of a newborn who...
Neonatal Emergencies

would pass through the internal iliac artery from which the umbilical artery originates before it ultimately is located in the descending aorta. Either of two locations of the umbilical artery catheter tip would be appropriate: between the sixth and ninth thoracic vertebrae (a “high line”) or between the third and fourth lumbar vertebrae (a “low line”) (Figure 11.7).

Although the above information is helpful in identifying what is ideal for umbilical vessel catheter placement, it is much more likely that an emergency department clinician would need to obtain emergency vascular access for the administration of fluids or medications. In the event that peripheral venous access cannot be obtained, the umbilical vein can be rapidly and safely catheterized by a skilled clinician. After sterilizing the base of the umbilical cord

Figure 11.7 Management of hyperbilirubinemia in the newborn.

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or stump and the surrounding skin, a sterile tie should be firmly tightened around the base of the cord. The cord stump should be cut horizontally with a scalpel, with careful attention to avoid lacerating the skin at the base of the cord, to expose the umbilical vessels. Once the umbilical vein is visualized, a saline-flushed catheter of appropriate size can be inserted just a few centimeters below the cut surface of the cord until blood return is documented on drawing back on the syringe. With the catheter in place, maintenance or bolus fluids and emergency medications can be safely administered. The catheter should be secured to the non-skin portion of the umbilical cord (known as Wharton jelly) with a suture as soon as is permissible.

**Thoracostomy Drainage of a Pneumothorax**

Pneumothorax is a known complication of neonatal resuscitation and can be due to anatomical or physiologic characteristics of the neonate or the manner in which resuscitation is conducted. When small in volume, pneumothoraces might not result in any symptoms or signs of respiratory distress but instead might appear as an incidental finding on a chest radiograph. Larger pneumothoraces will result in tachypnea, retractions and nasal flaring in the newborn, diminished breath sounds on the affected side, and hypoxemia. Tension pneumothorax will present with more severe hypoxia and severe cardiovascular comprise with a shift of the heart sounds and trachea to the contralateral side. Suspected pneumothorax can be confirmed by chest radiography if the patient is stable.

When significant respiratory distress is thought to be due to pneumothorax, immediate evacuation of the air in the pleural space is essential. The affected side can be determined immediately by transillumination and clinical assessment. Needle aspiration of
A pneumothorax can be performed quickly using a small-bore (18- or 20-gauge) percutaneous catheter. The newborn should be turned on the side so that the side of the pneumothorax is up. After the fourth intercostal space has been located at the anterior axillary line, the skin can be sterilized and the needle inserted just superior to the fourth rib. Once the catheter has been inserted into the pleural space the needle can be removed. The catheter should then be connected to a stopcock and 20-mL syringe. Gentle aspiration of air into the syringe should be performed. If the syringe has been pulled back completely and there is evidence of further air in the pleural space, the stopcock can be turned off to the patient and the air can be expelled through the side port of the stopcock. Continue aspiration until there is resistance to aspiration. At this point the needle should be removed from the chest and a follow-up chest radiograph should be obtained (Figure 11.8).

If needle decompression does not completely remove the intrapleural air and symptoms persist or if it is suspected on clinical grounds that the pneumothorax would persist despite needle decompression, a thoracostomy tube can be inserted at the fifth intercostal space at the anterior axillary line (just lateral to the nipple). A thoracostomy tube of appropriate size can be inserted just over the sixth rib, after which the tube can be advanced toward the anterior aspect of the chest to an appropriate depth. After securing the tube in place, it should be connected to a suction device set to maintain negative 10-cm H₂O pressure. As above, a chest radiograph should be obtained to check the position of the tube and the status of the pneumothorax.

Figure 11.9 A. Locations for percutaneous aspiration of intrapleural air from the chest. Note that the needle enters just above the rib, so as to avoid the artery lying just under the rib above. B. Insertion of a percutaneous catheter for drainage of a pneumothorax or pleural fluid (see text). The needle can be placed at either of the “X” marks shown in part A but should always be perpendicular to the chest surface. Note that the needle present in part A has been removed and only the catheter remains in the pleural space.

10. Which of the following statements regarding umbilical catheter placement is true?
   A. The umbilical cord contains two umbilical veins and one umbilical artery.
   B. Catheterization of the umbilical vessels can be performed rapidly and safely in an emergency.
   C. The exact location of an emergently placed umbilical venous catheter must be confirmed by radiography before it can be used for fluid or medication administration.
   D. All are true.

References

### TABLE 5-1  RSI Drugs, Doses (mg/kg), Sizes, Distances

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Abbreviation: ICP, intracranial pressure.

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