Shock
Phoebe Yager and Natan Noviski
*Pediatrics in Review* 2010;31:311
DOI: 10.1542/pir.31-8-311

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://pedsinreview.aappublications.org/content/31/8/311
Objectives  After completing this article, readers should be able to:

1. Describe the basic pathophysiology of shock.
2. Characterize the various causes of shock and recognize their clinical presentations.
3. Discuss the importance of early, goal-directed treatment of shock.
4. Know the guidelines for the type and volume of fluid to be infused initially in hypovolemic or septic shock.
5. Choose the correct drug for the initial management of septic versus cardiogenic shock.
6. Be familiar with some of the recent therapies under investigation for the treatment of shock.

Introduction

A 9-month-old girl presents to the emergency department (ED) with a 4-day history of profuse diarrhea and poor oral intake. On physical examination, she appears irritable. Her respiratory rate (RR) is 70 breaths/min, heart rate (HR) is 180 beats/min, and blood pressure (BP) is 80/50 mm Hg. She has cool, mottled extremities, with sluggish capillary refill and weak peripheral pulses. Is this just a case of dehydration or could this be shock?

A 17-year-old boy presents to the ED with a 1-day history of headache, general malaise, and fevers. On physical examination, he appears confused. He has a temperature of 39.9°C, HR of 120 beats/min, and BP of 85/28 mm Hg. His skin appears plethoric. His extremities are hot, with flash capillary refill and bounding pulses. Is this the same entity that is affecting the previous patient?

A 2-week-old boy presents to the ED with a 1-day history of poor feeding. On physical examination, he is difficult to arouse. His RR is 80 breaths/min, HR is 220 beats/min, and BP is undetectable. He appears cyanotic and has cold extremities and a 5-second capillary refill time. Is this the same entity as seen with the other two patients? How should you proceed?

All three scenarios describe patients in various stages of shock, the first due to hypovolemic shock, the second due to septic shock, and the third due to cardiogenic shock.

The presentation of shock may vary, depending on the cause and stage of illness, although the pathophysiology and general management are similar. Prompt recognition of shock followed by early, goal-directed therapy and frequent reassessment are paramount to a successful outcome.

Definition and Pathophysiology

Shock is a life-threatening state that occurs when oxygen and nutrient delivery are insufficient to meet tissue metabolic demands. The crisis may occur when a disease compromises any of the factors that contribute to oxygen and nutrient delivery. Familiarity with a few simple equations is key not only to understanding the myriad factors that may contribute to shock but to understanding how the body...
attempts to compensate for the condition and how the clinician may intervene to reverse shock.

Oxygen delivery (DO₂) is determined by cardiac output (CO) and the arterial content of oxygen (CaO₂):

\[
DO₂ \ (\text{mL/min}) = CO \ (\text{L/min}) \times CaO₂ \ (\text{mL/L})
\]

Cardiac output is the product of stroke volume (SV) and HR:

\[
CO \ (\text{L/min}) = SV \ (\text{L}) \times HR/\text{min}
\]

Stroke volume is determined by:

- Preload: the amount of filling of the ventricle at end-diastole
- Afterload: the force against which the ventricle must work to eject blood during systole
- Contractility: the force generated by the ventricle during systole
- Lusitropy: the degree of myocardial relaxation during diastole

Heart rate variability relies on an intact autonomic nervous system and a healthy cardiac conduction system.

Arterial oxygen content also dictates oxygen delivery and is determined by hemoglobin (Hgb), oxygen saturation (SaO₂), and the partial pressure of oxygen (PaO₂), as follows:

\[
CaO₂ \ (\text{mL/L}) = \left\{ \left[ \text{Hgb} \ (g/\text{dL}) \times 1.34 \ (\text{mL} \ \text{O}_2/\text{g} \ \text{Hgb}) \times (\text{SaO}_₂/100) \right] + (\text{PaO}_₂ \times 0.003 \ \text{mL} \ \text{O}_2/\text{mm Hg}/\text{dL}) \right\} \times 10 \ \text{dL}/\text{L}
\]

For example, for a patient who has an Hgb value of 15 g/dL, PaO₂ of 100 torr, CO of 5 L/min, and SaO₂ of 98%, the DO₂ can be calculated as follows:

\[
\text{CaO}_₂ = \left\{ \left[ 15 \ g/\text{dL} \times 1.34 \ \text{mL} \ \text{O}_2/\text{g} \ \text{Hgb} \times (98/100) \right] + (100 \times 0.003 \ \text{mL} \ \text{O}_2/\text{mm Hg}/\text{dL}) \right\} \times 10 \ \text{dL}/\text{L}
\]

\[
\text{CaO}_₂ = 200 \ \text{mL}/\text{L}
\]

\[
\text{DO}_₂ = 5 \ \text{L/min} \times 200 \ \text{mL}/\text{L} = 1,000 \ \text{mL/min}
\]

It is important to recognize that oxygen is not distributed uniformly to the body. Modulation of systemic vascular resistance (SVR) in different vascular beds is one of the body’s primary compensatory mechanisms to shunt blood preferentially to vital organs such as the heart and brain. In this way, an increase in SVR may maintain a normal blood pressure even in the face of inadequate oxygen delivery. In other words, hypotension need not be present for a child to be in shock.

Shock refers to a dynamic state ranging from early, compensated shock to irreversible, terminal shock. During the earliest stage of shock, vital organ function is maintained by a number of compensatory mechanisms, and rapid intervention can reverse the process. If unrecognized or undertreated, compensated shock progresses to decompensated shock. This stage is characterized by ongoing tissue ischemia and damage at the cellular and subcellular levels. Inadequate treatment leads to terminal shock, defined as irreversible organ damage despite additional resuscitation.

**Classification and Clinical Presentation**

**Hypovolemic Shock**

Hypovolemic shock is the most common form of shock occurring in children around the world. Diarrheal illnesses are the cause in most of these patients. Some other causes include bleeding, thermal injury, and inappropriate diuretic use.

Signs and symptoms of hypovolemic shock include tachycardia, tachypnea, and signs of poor perfusion, including cool extremities, weak peripheral pulses, sluggish capillary refill, skin tenting, and dry mucous membranes. Orthostatic hypotension may be an early sign. As the body’s ability to compensate reaches its limit, hypotension develops, along with additional signs of hypoperfusion and end-organ damage. At this stage, the clinical findings include weak central pulses, poor urine output, mental status changes, and metabolic acidosis.

**Cardiogenic Shock**

Cardiogenic shock refers to failure of the heart as a pump, resulting in decreased cardiac output. This failure may be due to depressed myocardial contractility, arrhythmias, volume overload, or diastolic dysfunction. Depressed myocardial contractility may be seen with primary neuromuscular disorders or may be acquired in any number of settings, such as infection, following exposure to a toxin, or when a patient suffers a metabolic derangement such as severe hypocalcemia or hyperkalemia. Myocardial ischemia due to inadequate coronary perfusion occurs with a number of congenital cardiac lesions as well as with dysrhythmias that may compromise cardiac output severely.

Hypoplastic left heart syndrome, aortic stenosis, and coarctation of the aorta are three life-threatening congenital lesions that obstruct outflow from the left heart. Systemic outflow and coronary perfusion in these conditions depend on right-to-left flow from the pulmonary artery to the aorta via a patent ductus arteriosus. Tricuspid atresia, pulmonary atresia, and tetralogy of Fallot are three cyanotic congenital lesions that obstruct outflow from the right heart. In these lesions, adequate pulmonary blood flow depends on a left-to-right shunt.
across a patent ductus arteriosus. Infants born with ductal-dependent congenital heart disease often present within the first 2 weeks after birth, by which time the ductus arteriosus has closed.

Infants born with congenital lesions resulting in significant left-to-right shunts (e.g., ventricular septal defects, truncus arteriosus, and anomalous left coronary artery from the pulmonary artery [ALCAPA]) typically present between 6 weeks and 3 months of age as pulmonary vascular resistance (PVR) falls. This change in resistance results in a steal phenomenon whereby blood preferentially flows to the pulmonary bed and away from the systemic bed, leading to inadequate cardiac output. In the case of ALCAPA, as PVR falls, blood flows to the pulmonary bed and away from the myocardium. Coronary ischemia develops, resulting in poor contractility, diminished cardiac output, and life-threatening dysrhythmias.

Other types of dysrhythmias (e.g., supraventricular tachycardia) infections causing myocarditis or pericarditis, and congenital cardiomyopathies can present at any time and should be part of the differential diagnosis for any patient presenting with signs of poor perfusion.

Infants and children who have cardiogenic shock often present with lethargy, poor feeding, tachycardia, and tachypnea. They typically appear pale and have cold extremities and barely palpable pulses. In the case of critical coarctation of the aorta, the infant may have absent femoral pulses and a significantly lower BP in the lower extremities compared with the right upper extremity. Oliguria is present.

Initially, it may be impossible to differentiate cardiogenic shock from septic shock. Findings more specific to cardiogenic shock include a gallop rhythm, rales, jugular venous distension, and hepatomegaly. Chest radiography reveals cardiomegaly and pulmonary venous congestion. Unlike other forms of shock in which central venous pressure (CVP) is low, in cardiogenic shock, CVP is elevated above 10 cm H2O. Although it is imperative to obtain electrocardiography and echocardiography immediately if there is any suspicion of cardiogenic shock, empiric treatment for possible septic or cardiogenic shock should not be delayed.

Two noncardiac conditions that can lead to cardiogenic shock are bilateral pneumothoraces and cardiac tamponade. Both prevent diastolic filling of the heart, which leads to decreased SV and poor CO. These disorders should be suspected in the patient presenting with signs of poor perfusion accompanied by a narrow pulse pressure and, in the case of tamponade, muffled heart tones on auscultation.

### Distributive or Neurogenic Shock

Distributive shock is caused by derangements in vascular tone that lead to end-organ hypoperfusion. This outcome is seen with anaphylaxis, an immunoglobulin E-mediated hypersensitivity reaction in which mast cells and basophils release histamine, a potent vasodilator, and there is massive production of other potent vasodilators, including prostaglandins and leukotrienes. Spinal cord trauma and spinal or epidural anesthesia also can cause widespread vasoplegia due to loss of sympathetic tone. This situation sometimes is referred to as neurogenic shock. Unlike other forms of shock, patients who experience neurogenic shock exhibit hypotension without reflex tachycardia. Finally, septic shock in some children presents with vasoplegia.

### Septic Shock

In 1992, the American College of Chest Physicians and the Society of Critical Care Medicine formally defined sepsis and its related syndromes. They introduced the important concept of a systemic inflammatory response syndrome (SIRS), whereby the body responds to various insults (infection, trauma, thermal injury, acute respiratory distress syndrome) with overwhelming inflammation resulting in hypo- or hyperthermia, tachycardia, tachypnea, and either an elevated or depressed white blood cell count. When SIRS is triggered by an infection, it is defined as sepsis, and when this condition is associated with organ dysfunction, it is referred to as severe sepsis. Septic shock in the pediatric population is characterized by sepsis accompanied by tachycardia and signs of inadequate perfusion. (1)

The host response to infection is one of the primary determinants of whether an individual develops septic shock. Endotoxin released by gram-negative rods and antigens presented by various other pathogens set in motion in the host an inflammatory cascade resulting in the activation of lymphocytes and the release of proinflammatory cytokines such as tumor necrosis factor, interleukin-1 (also known as endogenous pyrogen), and interleukin-6. These cytokines activate other proinflammatory cytokines and mediators of sepsis, including nitric oxide (a potent vasodilator), platelet-activating factor, prostaglandins, thromboxane, and leukotrienes. Overproduction of these mediators disrupts the delicate balance between pro- and anti-inflammatory factors and can lead to unchecked inflammation and septic shock.

“Cold” versus “warm” shock refers to the two primary clinical presentations of septic shock. “Cold” shock describes the pattern of signs and symptoms seen with
Patients suffering shock may develop acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). ALI and ARDS are marked by increasingly poor oxygenation (PaO₂/FiO₂ < 300 in ALI and PaO₂/FiO₂ < 200 in ARDS) and ventilation, despite escalating ventilatory support and worsening bilateral infiltrates on chest radiograph without signs of left-sided heart failure. It is important to recognize ALI or ARDS and respond appropriately with a lung-protective strategy of ventilation.

Access
Obtaining rapid vascular access with at least two wide-bore peripheral intravenous lines is critical to the timely treatment of circulatory shock. Obtaining such access can be challenging because most patients present with cool, poorly perfused extremities. If sufficient percutaneous venous access cannot be obtained quickly, the Neonatal Resuscitation Program and Pediatric Advanced Life Support (PALS) guidelines recommend placement of an umbilical venous catheter (neonates only) or intra-osseous needle (infants and children). (2) Central venous access provides more stable, long-term access and should be obtained in patients who have fluid-refractory shock and who require titration of vasopressors and inotropes. In addition, a central venous catheter enables the clinician to monitor CVP. This measurement can be helpful for diagnosing cardiogenic shock and guiding the clinician during volume administration in all forms of shock.

Sedatives and Analgesics
Many of the sedating medications used for intubation and other invasive procedures in children have properties that may exacerbate shock. Benzodiazepines, opioids, and propofol can decrease the BP and must be used judiciously. Etomidate has been shown to induce adrenal insufficiency. The 2007 updated clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock from the American College of Critical Care Medicine (ACCM) recommend against the use of etomidate and for the use of atropine and ketamine for children (experience with ketamine use in neonates was insufficient to recommend its use in this age group). (3)

Fluid Therapy
Rapid volume resuscitation is the single most important intervention to help restore adequate organ perfusion in patients presenting with various forms of hypovolemic shock. Volume resuscitation should occur early and be goal-directed. The American Heart Association and PALS guidelines recommend an initial rapid bolus of 20 mL/kg of isotonic fluid followed by immediate reassessment and titration of additional fluid administration to goals of normal BP and perfusion (capillary refill < 2 seconds, 1 mL/kg per hour urine output, normal mental status) or until signs of fluid overload occur (rales, increased work of breathing, gallop rhythm, hepatomegaly, CVP increases without additional hemodynamic improvement). Patients may require up to 200 mL/kg of isotonic fluid within the first hour, particularly in cases of vascular paralysis, to restore adequate perfusion.

The 2007 ACCM clinical practice guidelines for treatment of neonatal and pediatric septic shock recommend further that volume resuscitation beyond the first hour be titrated not only to signs of improved perfusion and normal blood pressure, but to an age-appropriate perfusion pressure (approximately 55 to 68 mm Hg), a mixed venous saturation greater than 70%, and a cardiac index greater than 3.3 L/min/m² and less than 6.0 L/min/m². The Figure provides an algorithm for goal-directed management of hemodynamic support in septic shock based on these guidelines.
Fluid resuscitation in infants and children who have cardiogenic shock should be approached carefully because these patients may be hypo-, hyper-, or euvolemic. In the euvolemic or hypervolemic patient, volume loading the failing heart may exacerbate pump failure and contribute to pulmonary congestion. Frequent clinical assessment is required during initial fluid resuscitation for patients who have potential cardiogenic shock. Ideally, a central venous line should be placed to monitor CVP and to assist in adjusting therapy.

Antibiotics

Broad-spectrum antibiotics based on age should be administered within the first hour of presentation when sepsis is suspected. Because it can be difficult to differentiate septic shock from cardiogenic shock in the neonate, this age group always should be treated with antibiotics. Appropriate specimens for blood, urine, and cerebrospinal fluid cultures should be obtained before antibiotic administration, although difficulty obtaining samples should not delay administration.

Crystalloid Versus Colloid

The 2007 ACCM clinical practice guidelines for treatment of neonatal and pediatric septic shock recommend either isotonic crystalloid or 5% albumin for volume resuscitation in the first hour. Beyond the first hour, the guidelines recommend crystalloid for patients who have Hgb values greater than 10 g/dL (100 g/L) and packed red blood cell transfusion for those whose Hgb values are less than 10 g/dL (100 g/L). In addition to restoring circulating volume, packed red blood cells also serve to increase oxygen-carrying capacity. Fresh frozen plasma administered as an infusion is recommended for patients who have a prolonged International Normalized Ratio.

Cardiovascular Support

In cases of fluid-refractory shock and cardiogenic shock, cardiovascular agents are necessary. The choice of agent depends largely on the underlying cause and the clinical presentation of shock. Selection of an appropriate agent is based on its known effects on inotropy, chronotropy, SVR, and PVR. The Table provides a summary of cardiovascular medications used in shock.

INOTROPIC AGENTS. Dopamine, dobutamine, and epinephrine work on beta receptors in the myocardium to increase cytoplasmic calcium concentration and enhance myocardial contractility. Dopamine is considered first-line therapy for patients who have fluid-refractory, hypovolemic, or septic shock. However, infants younger than 12 months of age may not respond effectively to dopamine, in which case epinephrine should be considered. Epinephrine also should be added for children experiencing dopamine-refractory hypovolemic or septic shock, defined as persistent hypotension despite at least 60 mL/kg volume and dopamine infusing at 10 mcg/kg per minute. For patients who have cardiogenic shock, early inotropic support rather than large-volume resuscitation is indicated.
tation is required. Dopamine, dobutamine, and epinephrine are acceptable therapies.

**VASOPRESSORS.** All of the previously discussed inotropic agents also have vasoactive properties that must be considered when selecting an appropriate agent and titrating the dose. At higher doses, for example, dopamine and epinephrine have increasing alpha-adrenergic effects, leading to peripheral vasoconstriction and increased SVR. Dobutamine, on the other hand, causes peripheral and pulmonary vasodilation due to beta2-adrenergic effects. This effect may be deleterious in the patient who has low CO and low SVR but beneficial in the child presenting with low CO and high SVR or the neonate experiencing acute cardiac failure and pulmonary hypertension.

Phenylephrine is a pure alpha-agonist used to achieve systemic vasoconstriction in distributive shock and septic shock presenting with high CO and low SVR. Norepinephrine is a more potent vasoconstrictor used in the same setting. At higher doses, norepinephrine also acts on beta receptors, exerting inotropic and chronotropic effects. Both drugs should be avoided in cardiogenic shock due to their powerful effect of increasing afterload.

Arginine-vasopressin and its synthetic analog, terlipressin, have been investigated for their potential use in vasodilatory shock refractory to first-line catecholamine agents. Given the limited pediatric experience with these agents for the treatment of catecholamine-refractory vasodilatory shock, no recommendation can be made at this time, and their use should be considered on a case-by-case basis. (4)

**VASODILATORS.** Nitroprusside is a pure vasodilator used to decrease afterload and improve coronary perfusion in neonates and children who have cardiogenic shock. Its use is limited by the need for adequate perfusion.

---

**Table. Cardiovascular Medications for the Treatment of Shock**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Site of Action</th>
<th>Clinical Effects</th>
<th>Uses in Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>β &lt; α (at higher doses), D1, D2</td>
<td>↑ inotropy, ↑ HR, ↑ SVR, ↑ PVR</td>
<td>● Hypovolemic shock (temporizing measure only during volume expansion)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>β1 &gt; β2</td>
<td>↑ inotropy, ↑ HR, ↓ SVR, ↓ PVR</td>
<td>● Cardiogenic shock</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>β &gt; α</td>
<td>↑ inotropy, ↑ HR, ↑ SVR</td>
<td>● Cardiogenic shock</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>α &gt;&gt; β</td>
<td>↑ SVR, ↑ inotropy, ↑ HR</td>
<td>● Hypovolemic shock (temporizing measure only during volume expansion)</td>
</tr>
<tr>
<td>Milrinone</td>
<td>PDE III inhibitor</td>
<td>↑ inotropy, ↓ SVR, ↓ PVR, ↑ lusitropy</td>
<td>● Cardiogenic shock with stable BP</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>α1 &gt; α2</td>
<td>↑ SVR</td>
<td>● Septic shock with ↑ CO, ↓ SVR</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>V1, V2</td>
<td>↑ SVR</td>
<td>● Septic shock with ↑ CO, ↓ SVR</td>
</tr>
<tr>
<td>Prostaglandin E1</td>
<td>PGE1</td>
<td>Dilation of ductus arteriosus</td>
<td>● Cardiogenic shock in neonate with suspected ductus-dependent lesion</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>arteries &gt; veins</td>
<td>↓ SVR, ↑ coronary perfusion</td>
<td>● Cardiogenic shock</td>
</tr>
<tr>
<td>Inhaled nitric oxide</td>
<td>pulmonary vessels</td>
<td>↓ PVR</td>
<td>● Cardiogenic shock with PHTN, RV failure</td>
</tr>
<tr>
<td>Levosimendan, enoximone</td>
<td>cardiac troponin C, ATP-dependent potassium channels in cardiac myocytes</td>
<td>↑ inotropy, ?cardioprotection</td>
<td>● Experimental use in cardiogenic shock</td>
</tr>
</tbody>
</table>

ATP = adenosine triphosphate, BP = blood pressure, CO = cardiac output, HR = heart rate, PDE = phosphodiesterase, PGE = prostaglandin E, PHTN = pulmonary hypertension, PVR = pulmonary vascular resistance, RV = right ventricle, SVR = systemic vascular resistance
Children presenting in shock often have a number of metabolic derangements, including hyper- or hypoproteinemia, hypocalemia, and hypoglycemia. These disorders should be suspected and treated promptly. However, persistent hyperglycemia often is seen beyond the initial resuscitation of shock and has been associated with increased severity of illness and increased mortality in hospitalized patients. Tight glycemic control (<150 mg/dL [8.3 mmol/L]) in critically ill adults has been advocated. However, to date no pediatric studies have analyzed the effects of tight glycemic control with insulin. Due to a lack of data and the known risk of hypoglycemia in children dependent on intravenous glucose, the 2008 Surviving Sepsis Campaign guidelines and 2007 ACCM parameters make no recommendations beyond the use of maintenance fluid containing 10% glucose.
Activated Protein C
Severe sepsis and septic shock often are accompanied by a significant disruption of the delicate balance between pro- and anticoagulants, leading to life-threatening disseminated intravascular coagulation. Uncontrolled coagulation ultimately consumes procoagulants, resulting in bleeding. Activated protein C is an anticoagulant that helps regulate coagulation and inflammation, and it has been found to be deficient in patients experiencing septic shock. Adult studies have shown that treatment with recombinant human activated protein C (rhAPC) reduces mortality with only a small increase in risk of bleeding. However, a large randomized, controlled trial of rhAPC in children was halted prematurely after interim analysis of the data from 474 enrolled patients revealed no difference in mortality. (8) Due to the inherent risk of bleeding and the absence of proven efficacy, the Surviving Sepsis Campaign guidelines recommend against the use of rhAPC in children who have septic shock.

Extracorporeal Life Support
Although ECMO has a definitive role in the treatment of cardiogenic shock refractory to maximum pharmacologic support, its role in the treatment of refractory septic shock has been less clear. Over the past decade, more centers have begun to use ECMO as rescue therapy for septic shock with circulatory collapse and multiorgan failure. The current ECMO survival rate for term newborns who have septic shock is 80%; that for older children is 50%. Based on limited retrospective data, the Surviving Sepsis Campaign guidelines and the ACCM parameters for support of pediatric and neonatal septic shock suggest that ECMO be considered only in cases of refractory septic shock or respiratory failure that cannot be managed adequately with conventional support.

Summary
- Based on strong research evidence (2), if sufficient peripheral access cannot be obtained quickly, placement of an intraosseous needle should be considered in the initial management of shock.
- Based on strong research evidence (9), fluid resuscitation for hypovolemic and septic shock should start immediately and the volume be titrated to attain specific goals indicating improved perfusion.
- Based on some research evidence and consensus (3), inotropic support should be administered through a peripheral intravenous line until central access can be obtained to avoid delay in restoration of adequate perfusion pressure.
- Based on weak research evidence and consensus (7), corticosteroid therapy is suggested only for patients who have catecholamine-resistant septic shock and suspected or proven adrenal insufficiency.
- Based on strong research evidence (8), rhAPC should not be used to treat children who have septic shock associated with disseminated intravascular coagulopathy.

References
2. American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) of pediatric and neonatal patients: PALS. Pediatrics. 2006;117: e1005–e1028
PIR Quiz

Quiz also available online at http://pedsinreview.aappublications.org.

1. A 3-day-old girl presents with difficulty breathing and looking dusky for the past 6 hours. The infant was born after a normal vaginal delivery and weighed 3.2 kg at birth. Examination reveals a pale/grayish-looking child whose respirations are labored, accompanied by grunting. Rectal temperature is 36.0°C, respiratory rate is 80 breaths/min, heart rate is 160 beats/min, and blood pressure is 50/30 mm Hg. Peripheral pulses are poor but equal in all extremities. Chest auscultation reveals a gallop rhythm and normal breath sounds. Her abdomen is distended, and the liver is palpated 4 cm below the right costal margin. No improvement is noted after an intravenous bolus of 20 mL/kg saline, endotracheal intubation, and mechanical ventilation with 40% oxygen. Antibiotics are administered. Pulse oximetry shows 90% oxyhemoglobin saturation. Blood gas analysis shows pH of 7.12, Pco2 of 60 torr, and Po2 of 74 torr. Which of the following is the next most appropriate step?
   A. Administer sodium bicarbonate.
   B. Begin infusion of norepinephrine.
   C. Increase FiO2 to increase oxyhemoglobin saturation above 95%.
   D. Initiate infusion of prostaglandin E1.
   E. Initiate inhaled nitric oxide therapy.

2. A 3-month-old boy presents with difficulty breathing and looking dusky for the past 6 hours. The infant has had difficulty feeding and episodes of high-pitched cry several times a day for the past 2 weeks. Examination reveals a pale/grayish-looking child whose respirations are labored, accompanied by grunting. Rectal temperature is 36.0°C, respiratory rate is 50 breaths/min, heart rate is 160 beats/min, and blood pressure is 90/60 mm Hg. His extremities are cold, with a capillary refill time of 4 seconds. Chest auscultation reveals a gallop rhythm and bilateral wheezes. The liver is palpated 4 cm below the right costal margin. Chest radiograph shows moderate cardiomegaly and pulmonary congestion. Electrocardiography shows prominent Q waves; ST segment elevation; and inverted T waves in leads I, aVL, V5, and V6. Which of the following mechanisms is the best explanation for these manifestations?
   A. Closure of the ductus arteriosus.
   B. Decrease in pulmonary arterial pressure.
   C. Decreased ventricular lusitropy.
   D. Inflammation of cardiac myocytes.
   E. Pericardial tamponade.

3. A 12-year-old boy presents after being struck by a car while crossing a street. He is unresponsive, with a respiratory rate of 15 breaths/min, heart rate of 50 beats/min, and blood pressure of 76/36 mm Hg. Physical examination reveals bruises and abrasions across the neck, chest, and abdomen. Chest auscultation documents clear breath sounds with equal air entry bilaterally. Rapid sequence intubation is performed, followed by mechanical ventilation. Rapid intravenous infusion of 100 mL/kg 0.9% saline is administered over 1 hour. Repeat assessment shows a heart rate of 52 beats/min and blood pressure of 80/40 mm Hg. His extremities are warm with brisk capillary refill time. Which of the following is most likely associated with these findings?
   A. Abdominal solid organ injury.
   B. Adrenal insufficiency.
   C. Intracranial hemorrhage.
   D. Myocardial contusion.
   E. Sympathetic tone loss.

4. A 5-year-old girl presents with fever and decreasing responsiveness for 12 hours. She is obtunded and moaning incoherently. Her rectal temperature is 40.0°C, respiratory rate is 26 breaths/min, heart rate is 136 beats/min, and blood pressure is 78/50 mm Hg. Her extremities are cool with prolonged capillary refill time. Multiple areas of ecchymosis and purpura are noted over her face, trunk, and extremities. After rapid sequence intubation, mechanical ventilation, intravenous fluid expansion with 0.9% saline (80 mL/kg), and administration of dopamine, epinephrine, and dobutamine, her blood pressure improves to 100/70 mm Hg. Her extremities, however, remain cold with poor perfusion. Which of the following is the next most appropriate therapy?
   A. Activated protein C.
   B. Arginine vasopressin.
   C. Hydrocortisone.
   D. Phenylephrine.
   E. 25% albumin.
### Shock

Phoebe Yager and Natan Noviski

*Pediatrics in Review* 2010;31;311

DOI: 10.1542/pir.31-8-311

| Updated Information & Services | including high resolution figures, can be found at: http://pedsinreview.aappublications.org/content/31/8/311 |
| References | This article cites 9 articles, 3 of which you can access for free at: http://pedsinreview.aappublications.org/content/31/8/311#BIBL |
| Subspecialty Collections | This article, along with others on similar topics, appears in the following collection(s):  
  - Cardiovascular Disorders http://pedsinreview.aappublications.org/cgi/collection/cardiovascular_disorders  
  - Infectious Diseases http://pedsinreview.aappublications.org/cgi/collection/infectious_diseases  
  - Fluid and Electrolyte Metabolism http://pedsinreview.aappublications.org/cgi/collection/fluid_electrolyte_metabolism  
  - Critical Care http://pedsinreview.aappublications.org/cgi/collection/critical_care |
| Permissions & Licensing | Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: /site/misc/Permissions.xhtml |
| Reprints | Information about ordering reprints can be found online: /site/misc/reprints.xhtml |