Physiology and classification of shock in children

Authors
Wendy J Pomerantz, MD, MS
Mark G Roback, MD

Section Editor
Susan B Torrey, MD

Deputy Editor
James F Wiley, II, MD, MPH

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INTRODUCTION — This topic will review the physiologic determinants and classification of shock. The initial evaluation and management of shock in children, in general, and hypovolemic and septic shock, specifically, are discussed separately. (See "Initial evaluation of shock in children" and "Initial management of shock in children" and "Hypovolemic shock in children: Initial evaluation and management" and "Systemic inflammatory response syndrome (SIRS) and sepsis in children: Definitions, epidemiology, clinical manifestations, and diagnosis".)

DEFINITION — Shock is a physiologic state characterized by a significant, systemic reduction in tissue perfusion, resulting in decreased tissue oxygen delivery. Although the effects of inadequate tissue perfusion are initially reversible, prolonged oxygen deprivation leads to generalized cellular hypoxia and derangement of critical biochemical processes, including [1,2]:

- Cell membrane ion pump dysfunction
- Intracellular edema
- Leakage of intracellular contents into the extracellular space
- Inadequate regulation of intracellular pH

These abnormalities rapidly become irreversible and result sequentially in cell death, end-organ damage, failure of multiple organ systems, and death [3-5]. Mortality from shock is less among children than adults. For children with severe sepsis, mortality is about 10 percent, in comparison to 35 to 40 percent within one month of the onset of septic shock for adults [6,7]. Nevertheless, outcomes for children with shock (in terms of morbidity and cost) are significant. Furthermore, unique physiologic responses to poor perfusion among children make it a challenge for clinicians to recognize shock early (before hypotension develops), when responses to treatment are more favorable. (See "Initial evaluation of shock in children", section on 'Evaluation'.)

PHYSIOLOGIC DETERMINANTS — Parameters that determine adequate oxygen delivery to tissues include blood flow to tissues (cardiac output), the regional balance between blood flow and metabolic demand, and the oxygen content of blood (hemoglobin concentration and percentage of hemoglobin saturated with oxygen) [3]. Physiologic variables that the body can manipulate to compensate for compromised perfusion include:

- Cardiac output (volume of blood flow per unit of time) is the product of stroke volume times heart rate. Hence, tachycardia is a common sign of decreased perfusion and early shock. Infants have relatively fixed stroke volumes and are particularly dependent upon heart rate to increase cardiac output.

- Stroke volume is determined by preload, cardiac contractility, and afterload. Compensatory mechanisms that improve stroke volume include increased venous smooth muscle tone (improves preload by shunting blood to the heart) and increased cardiac contractility (resulting in more complete emptying of the ventricles).

- Increased systemic vascular resistance (vasoconstriction) maintains perfusion pressure (measured as blood pressure) despite decreased cardiac output. In addition, blood is shunted away from peripheral structures (including skin, muscle, kidneys, and splanchnic organs) to the heart and central nervous system. As a result, children with compensated shock typically have normal blood pressures, despite signs of poor perfusion (such as decreased peripheral pulses and tachycardia). (See "Initial evaluation of shock in children", section on 'Evaluation'.)

While decreased perfusion directly reflects decreased cardiac output, the increased cardiac output observed in hyperdynamic shock states also is associated with decreased effective tissue perfusion [8]. This decreased effective perfusion derives from a complex interaction of numerous humoral and microcirculatory processes resulting in patchy, uneven local regional blood flow and a derangement of cellular metabolic processes [9].

STAGES OF SHOCK — The shock syndrome is characterized by a continuum of physiologic stages beginning with an initial inciting event that causes a systemic disturbance in tissue perfusion. Subsequently, shock may progress through three stages if not successfully treated, culminating in end-organ damage, irreversible shock, and death [2,10].

Compensated shock — During compensated shock, the body's homeostatic mechanisms rapidly compensate for diminished perfusion and systolic blood pressure is maintained within the normal range [3]. Heart rate is initially increased. Signs of peripheral vasoconstriction (such as cool skin, decreased peripheral pulses, and oliguria) can be noted as perfusion becomes further compromised.

Hypotensive shock — During this stage, compensatory mechanisms are overwhelmed. Signs and symptoms of organ dysfunction (such as altered mental status as the result of poor brain perfusion) appear. Systolic blood pressure falls, although children who have lost as much as 30 to 35 percent of circulating blood volume can typically maintain normal systolic blood pressures [3,11]. Once hypotension develops, the child's condition usually deteriorates rapidly to cardiovascular collapse and cardiac arrest.

Although hypotension is generally a late finding among children with shock, those with early distributive shock (as with sepsis) may have hypotension because of decreased systemic vascular resistance (SVR). Vital organ perfusion is initially maintained by increased cardiac output.

Irreversible shock — During this stage, progressive end-organ dysfunction leads to irreversible organ damage and death. The process is often irreversible, despite resuscitative efforts.

CLASSIFICATION — Four broad mechanisms of shock are recognized: hypovolemic, distributive, cardiogenic, and obstructive. Each type is characterized by one primary physiologic derangement (table 1).

For any given condition that can cause shock, the classification may be mixed. Patients with distributive shock, in particular, often have multiple physiologic abnormalities. As an example, children with distributive shock from sepsis may also have volume loss (from vomiting, diarrhea, poor intake, or increased insensible fluid loss from tachypnea and fever) and myocardial depression from the effect of inflammatory mediators released in response to infection [3].

Hypovolemic shock — Hypovolemic shock is the most common type of shock encountered in children. It results from decreased preload from extravascular fluid loss (such as with diarrhea or osmotic diuresis) or intravascular fluid loss (as with capillary leak or hemorrhage). Because preload is one of the determinants of stroke volume, cardiac output falls when preload drops. (See "Hypovolemic shock in children: Initial evaluation and management".)

Distributive shock — Distributive or vasodilatory shock results from a decrease in SVR, with abnormal distribution of blood flow within the microcirculation and inadequate tissue perfusion. It can lead to functional hypovolemia with decreased preload [3,5,8]. Distributive shock generally is associated with a normal or increased cardiac output.

Causes of distributive shock include:

- Sepsis is the most common etiology of distributive shock among children. (See "Systemic inflammatory response syndrome (SIRS) and sepsis in children: Definitions, epidemiology, clinical manifestations, and diagnosis".)
- Anaphylaxis is an immediate, potentially life-threatening systemic reaction to an exogenous stimulus, typically an allergic, IgE-mediated immediate hypersensitivity reaction. Anaphylactoid responses are clinically indistinguishable reactions that occur by nonantigen/antibody-mediated mechanisms. (See "Anaphylaxis: Emergency treatment".)
- Neurogenic shock is a rare, usually transient disorder that follows acute injury to the spinal cord or central nervous system, resulting in loss of sympathetic venous tone.

Cardiogenic shock — Cardiogenic shock results from pump failure, manifested physiologically as decreased systolic function and depressed cardiac output [12]. Cardiogenic shock is uncommon among children (table 2), as compared with adults, among whom ischemic heart disease is the major cause. The mechanisms of cardiogenic shock are diverse and can be divided into two general categories: cardiomyopathies and arrhythmias [13,14].

- **Cardiomyopathies** – Primary myocardial injury is an uncommon cause of shock in children. Causes of myopathic pump failure include familial, infectious, infiltrative, and idiopathic cardiomyopathies. Prolonged ischemia, cardiopulmonary bypass, and the myocardial depression of late sepsis all can contribute to secondary myocardial dysfunction.
- **Arrhythmias** – Structural heart disease, drug intoxications, and hypothermia are leading causes of arrhythmia in children. Both atrial and ventricular arrhythmias can cause cardiogenic shock.
    - Ventricular fibrillation and pulseless ventricular tachycardia abolish cardiac output, while diminished ventricular filling time during ventricular tachycardia decreases preload and stroke volume substantially. (See "Management and evaluation of wide QRS complex tachycardia in children".)
    - Prolonged unrecognized supraventricular tachycardia (as can occur with the initial presentation for infants) can decrease cardiac output. (See "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia".)
    - Bradyarrhythmias and complete heart block can result in shock caused by chronotropic (heart rate) insufficiency, independent of stroke volume. (See "Bradycardia in children".)

Obstructive shock — Obstructive shock and impaired cardiac output result when blood flow is physically obstructed. Acquired causes of obstructive shock include cardiac tamponade, tension pneumothorax and massive pulmonary embolism. Infants with ductal-dependent congenital heart lesions, such as coarctation of the aorta and hypoplastic left ventricle syndrome, may present in shock when the ductus arteriosus closes during the first few weeks of life [15]. (See "Approach to the septic-appearing infant", section on 'Initial management decisions'.)

COMMON FEATURES — The clinical presentation of shock is variable, but several features are common. These include tachycardia and signs of compromised organ perfusion (skin, brain, and kidneys). Children often present before hypotension develops. (See "Initial evaluation of shock in children".)

- **Tachycardia** – Tachycardia is typical. Although tachycardia is an important early indicator of shock, it is a nonspecific finding. Many common conditions in children such as fever, pain, and anxiety can cause tachycardia without circulatory compromise. A normal heart rate with signs of compensated shock can occur with spinal cord injury and bradycardia can occur as the result of hypoxia, some ingestions (such as beta blockers and calcium channel blockers), or as an agonal event for patients with shock from any cause (table 3).
- **Skin changes** – In many shock states, regulatory processes compensate for decreased effective tissue perfusion. Potent vasoconstrictive mechanisms redirect blood from the peripheral, splanchnic, and renal vessels to maintain coronary and cerebral perfusion. As a result, the skin is typically cool, clammy, pale, or mottled.

  Notable exceptions are the flushed, hyperemic skin of early distributive shock and the peripheral vasodilation of terminal shock states associated with failure of mechanisms that maintain increased peripheral vascular resistance (irreversible shock).
- **Impaired mental status** – Children with impaired cerebral perfusion may be initially listless or agitated and not interacting with caregivers. Mental status typically deteriorates to obtundation and coma as the shock state worsens.
- **Oliguria** – Oliguria with decreased glomerular filtration rate results from the shunting of renal blood flow to other vital organs and the fall in intraglomerular pressure, which normally drives glomerular filtration.
- **Lactic acidosis** – Progressive tissue hypoperfusion is associated with the development of lactic acidosis. Two

factors contribute to this abnormality: increased lactic acid production caused by inadequate delivery of oxygen and decreased clearance of lactate by the liver, kidneys, and skeletal muscle [16]. (See "Causes of lactic acidosis".) The early stages of sepsis often are associated with a respiratory alkalosis caused by primary hyperventilation [17]. (See "Simple and mixed acid-base disorders".)

- **Hypotension** – Hypotension is typically a **late** finding among children in shock. Compensatory vasoconstriction is often so pronounced that systemic blood pressure can be maintained within the normal range, despite significant circulatory compromise. In this situation, the main clinical manifestations of shock are tachycardia and signs of organ hypoperfusion.

  For children, hypotension is defined as a systolic blood pressure that is less than the fifth percentile of normal for age [3]:

  - Less than 60 mmHg in term neonates (0 to 28 days)
  - Less than 70 mmHg in infants (1 month to 12 months)
  - Less than 70 mmHg + (2 x age in years) in children 1 to 10 years
  - Less than 90 mmHg in children 10 years of age or older

SUMMARY — Shock is a physiologic state characterized by a significant, systemic reduction in tissue perfusion, resulting in decreased tissue oxygen delivery. Unique physiologic responses to poor perfusion among children make it a challenge for clinicians to recognize shock early (before hypotension develops), when responses to treatment are more favorable.

- Physiologic variables that the body can manipulate to compensate for compromised perfusion include cardiac output (stroke volume times heart rate), stroke volume (determined by preload, cardiac contractility, and afterload), and systemic vascular resistance (SVR). (See 'Physiologic determinants' above.)
- During compensated shock, the body's homeostatic mechanisms rapidly compensate for diminished perfusion and systolic blood pressure is maintained within the normal range. Once hypotension develops, the child's condition usually deteriorates rapidly to cardiovascular collapse and cardiac arrest. Progressive end-organ dysfunction leads to irreversible organ damage and death. (See 'Stages of shock' above.)
- Types of shock include hypovolemic shock (characterized primarily by reduction in preload, as the result of fluid loss), distributive shock (decreased SVR with abnormal distribution of blood flow from sepsis, anaphylaxis, or nervous system injury), and cardiogenic shock (pump failure as the result of cardiomyopathies, arrhythmias, or obstruction). (See 'Classification' above.)
- Common features of shock include tachycardia and signs of compromised organ perfusion (skin, brain, and kidneys). Children often present before hypotension develops. (See 'Common features' above.)

REFERENCES

1. Barber AE, Shires GT. Cell damage after shock. New Horiz 1996; 4:161.
2. Kristensen SR. Mechanisms of cell damage and enzyme release. Dan Med Bull 1994; 41:423.
3. Pediatric Advanced Life Support Provider Manual, Chameides L, Samson RA, Schexnayder SM, Hazinski MF (Eds), American Heart Association, Dallas 2011. p.69.
4. Bell LM. Shock. In: Textbook of Pediatric Emergency Medicine, 6th, Fleisher GR, Ludwig S, Henretig FM (Eds), Lippincott Williams & Wilkins, Philadelphia 2010. p.46.
5. Tobin RT, Wetzel RC. Shock and multiple system organ failure. In: Textbook of Pediatric Intensive Care, Rogers

MC (Ed), Williams & Wilkins, Baltimore 1996. p.555.

6. Watson RS, Carcillo JA, Linde-Zwirble WT, et al. The epidemiology of severe sepsis in children in the United States. Am J Respir Crit Care Med 2003; 167:695.
7. Bone RC. Toward an epidemiology and natural history of SIRS (systemic inflammatory response syndrome). JAMA 1992; 268:3452.
8. Chittock DR, Russell JA. Oxygen delivery and consumption during sepsis. Clin Chest Med 1996; 17:263.
9. Hinshaw LB. Sepsis/septic shock: participation of the microcirculation: an abbreviated review. Crit Care Med 1996; 24:1072.
10. Abboud FM. Pathophysiology of hypotension and shock. In: The Heart, Hurst JW (Ed), McGraw-Hill, New York 1982. p.452.
11. Schwaitzberg SD, Bergman KS, Harris BH. A pediatric trauma model of continuous hemorrhage. J Pediatr Surg 1988; 23:605.
12. Rodgers KG. Cardiovascular shock. Emerg Med Clin North Am 1995; 13:793.
13. Witte MK, Hill JH, Blumer JL. Shock in the pediatric patient. Adv Pediatr 1987; 34:139.
14. Bengur AR, Meliones JN. Cardiogenic shock. New Horiz 1998; 6:139.
15. Pediatric Advanced Life Support Provider Manual, Chamedes L, Samson RA, Schexnayder SM, Hazinski MF (Eds), American Heart Association, Dallas 2012. p.79.
16. Levraut J, Ciebiera JP, Chave S, et al. Mild hyperlactatemia in stable septic patients is due to impaired lactate clearance rather than overproduction. Am J Respir Crit Care Med 1998; 157:1021.
17. Simmons, DH, Nicoloff, J, Guze, LB. Hyperventilation and respiratory alkalosis as signs of gram negative bacteremia. JAMA 1960; 174:219.

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GRAPHICS

Hemodynamic profiles of the types of shock in children

Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion	Tissue perfusion
Clinical measurement	**Clinical signs* or central venous pressure (if measured)**	**Cardiac output or index ¶**	**Systemic vascular resistance**	**Capillary refill time Δ**	**Mixed venous oxygen saturation ◊**
Hypovolemic	↓	↓	↑	↑	Low
Cardiogenic	↑	↓	↑	↑	Low
Distributive	↓ or ↔	↑	↓	↓ (initial)	High
Obstructive	↑	↓	↑	↑	Low

* Clinical signs of decreased preload include tachycardia, tachypnea, decreased or absent peripheral pulses; normal or weak central pulses; capillary refill time >2 seconds; skin that is pale, mottled, cold or diaphoretic; dusky or pale extremities, altered mental status, decreased urine output, and flat jugular veins. Clinical signs of increased preload include jugular venous distension, pulmonary edema, and hepatomegaly. These patients are also typically tachycardic and poorly perfused. Refer to topics on evaluation of shock in children.

¶ Cardiac index (cardiac output per body surface area) is typically what is measured during clinical care.

Δ In patients with shock, capillary refill time >2 seconds is associated with low mixed venous oxygen saturation while flash capillary refill suggests increased mixed venous oxygen saturation.

◊ A low mixed oxygen saturation is <70 percent when measured through a triple lumen catheter and <65 percent when measured through a pulmonary artery catheter.

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Causes of cardiogenic shock and obstructive shock in children

Trauma
Hemopericardium with tamponade*
Blunt cardiac injury
Myocardial contusion
Traumatic aneurysm
Traumatic septal defect
Chamber rupture
Valvular rupture
Tension pneumothorax*
Tension pneumopericardium*
Congenital heart disease
Critical aortic stenosis*
Critical coarctation of the aorta*
Mitral stenosis*
Mitral atresia*
Interrupted aortic arch*
Hypoplastic left heart*
Pulmonary hypotension
Severe heart failure secondary to congenital heart disease
Postoperative cardiac surgery
Dysrhythmias
Supraventricular tachycardia
Ventricular tachycardia
Atrioventricular block
Junctional ectopic tachycardia
Bradycardia
Massive pulmonary embolus*
Drug toxicity
Beta blockers
Barbiturates
Chemotherapeutic agents
Calcium channel blockers
Radiation

Metabolic derangements
Acidosis
Hyperkalemia
Hypocalcemia
Congenital organic acidemias
Late septic shock
Hypoxic or anoxic/ischemic injury
Hypothermia
Viral myocarditis
Cardiomyopathies
Dilated cardiomyopathies
Infiltrative cardiomyopathies
Mucopoylsaccharidosis
Glycogen storage diseases
Ischemic heart disease
Anomalous left coronary artery
Kawasaki disease
Myocardial infarction
Thyrotoxicosis
Pheochromocytoma

*Denotes that the condition primarily causes obstructive shock. All other conditions primarily cause cardiogenic shock.

Data from:

1. *Witte MK, Hill JH, Blumer JL. Adv Pediatr 1987; 34:139.*
2. *Perkin RM, Levin DL. Shock. In: Essentials of Pediatric Intensive Care, Levin DL, Morriss FC (Eds), Quality Medical Pub, St. Louis, MO 1990. p.78.*
3. *Tobias JD. Shock in children: The first 60 minutes. Pediatr Ann 1996; 25:330.*
4. *Bengur AR, Meliones JN. Cardiogenic shock. New Horiz 1998; 6:139.*
5. *Bell LM. Shock. In: Textbook of Pediatric Emergency Medicine, Fleisher GR, Ludwig S, Henretig FM (Eds), Lippincott Williams & Wilkins, Philadelphia 2000. p.47.*
6. *Williams TM. Indiana Med 1988; 81:18.*
7. *Hazinski MF, Barkin RM. Shock. In: Pediatric Emergency Medicine: Concepts and Clinical Practice, Barkin RM (Ed), Mosby-Yearbook Inc, St. Louis, MO 1997. p.118.*

Graphic 67841 Version 7.0

Heart rates in normal children

Age	Awake rate	Mean	Sleeping rate
Newborn to 3 months	85-205	140	80-160
3 months to 2 years	100-190	140	75-160
2 years to 10 years	60-140	80	60-90
>10 years	60-100	75	50-90

Data from Gillette, PC, Garson, A Jr, Porter, CJ, McNamara, DG. Dysrhythmias. In: Adams, FH, Emmanouilides, GC, Riemenschneider, TA (Eds), Moss' heart disease in infants, children and adolescents, 4th ed, Williams &Wilkin, Baltimore 1989.

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