

## Types of Shock in Pediatrics — Management Guidelines

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**Shock in children is a common presentation to the emergency department. Shock can be classified according to the etiology into hypovolemic, cardiogenic, distributive, and obstructive. Hypovolemic shock due to hemorrhage or intravascular fluid losses and septic shock are the most commonly seen types of shock in children. The diagnostic workup and the physical examination of the child should aim to assess the severity of shock and to identify the most probable etiology. Securing the airway and breathing should have priority in the management of shock in children. The circulation should be improved by fluid expansion with fluid replacement therapy or inotropic therapy. Administration of antibiotics in septic shock should be started as early as possible.**



### Overview

Shock can be defined as the **inadequate delivery of glucose or oxygen to peripheral tissues and organs** in the body. This is associated with **acute energy failure**. When the cardiopulmonary system can no longer adequately supply the mitochondria with glucose and oxygen to create adenosine triphosphate (ATP), a shock state has developed. Additionally, **mitochondrial failure** due to inborn errors of metabolism might also be associated with shock at the cellular level.

**Two major types:**

Compensated	Uncompensated
Early	Late
Blood pressure is still normal	Blood pressure is low
Heart rate is elevated	End-organ damage is usually present

Because of the decreased glucose or oxygen delivery to the peripheral tissues, the patient can develop **central nervous system injury, respiratory failure, renal or hepatic dysfunction**, and **gastrointestinal ischemia**. If left untreated, shock can be **fatal** in children.

The main types of shock include:

- [Cardiogenic shock](#) (due to heart problems)
- [Hypovolemic shock](#) (caused by too little blood volume)
- [Anaphylactic shock](#) (caused by allergic reaction)
- [Septic shock](#) (due to infections)
- Neurogenic shock (caused by damage to the nervous system)

**Overview:**

Hypovolemic	Distributive (vasodilation)	Cardiogenic	Obstructive
<ul style="list-style-type: none"> <li>• Dehydration</li> <li>• Fluid loss</li> <li>• Bleeding</li> <li>• Third spacing</li> </ul>	<ul style="list-style-type: none"> <li>• Sepsis</li> <li>• Neurogenic</li> <li>• Anaphylaxis</li> </ul>	<ul style="list-style-type: none"> <li>• Cardiomyopathy</li> <li>• Arrhythmia</li> </ul>	<ul style="list-style-type: none"> <li>• Tamponade</li> <li>• Pulmonary embolism</li> <li>• Tension pneumothorax</li> </ul>

## Epidemiology of Shock in Children

Shock in children can be considered as one of the most common presentations that are **life-threatening** to the emergency pediatric department. Approximately, 37% of children who present to the emergency department are found to be in shock due to various causes and etiologies.

**Mortality** increases significantly in children who present with shock compared to those who have the same disease but present without shock. The most common cause of shock in children is sepsis, followed by hypovolemic shock, distributive shock and, finally, cardiogenic shock.

The prognosis of children presenting in shock has improved over the last decade, mainly due to the introduction of new classes of antibiotics and our recent advances in the understanding of the pathophysiology of sepsis and septic shock.

## Etiology of Shock in Children

Shock can be classified into **hypovolemic, cardiogenic, distributive** and **obstructive** based on the etiology.

### Hypovolemic shock

**Hypovolemic shock** is the **most common type** of shock to be seen in children and is characterized by a decreased cardiac filling, decreased end-diastolic volume and a decreased stroke volume and cardiac output. This shock is characterized with fluid losses caused by diarrhea and vomiting. These losses are often exacerbated by decreased oral intake as well.

A common cause of hypovolemic shock in children is **hemorrhage** (due to trauma, plasma losses due to burns, environmental exposure and peritonitis, as well as an increased urine loss as seen in diabetic ketoacidosis.) Other possible causes of hypovolemic shock in children include **intravascular volume loss** due to **gastroenteritis**, **burns** and **diabetes insipidus**. Due to an increase in sympathetic discharge and catecholamine release, peripheral vasoconstriction and tachycardia are often adequate in mild or moderate volume loss to preserve relatively normal blood pressure. The diastolic component of the blood pressure may be the most noticeably decreased.

## Cardiogenic shock

**Cardiogenic shock** can result from **congenital heart diseases** or **cardiomyopathies**. They are characterized by decreased cardiac output due to impaired systolic function of the heart and not because of decreased filling.

## Distributive shock

**Distributive shock** happens when the patient has a significant increase in peripheral vascular vasodilation and a decrease in systemic vascular resistance (distributive shock occurs when there is a maldistribution of intravascular volume). The most common causes of distributive shock in children are **sepsis** and **anaphylaxis**. Additionally, during the acute stage of **high-level spinal cord injury**, the child can present with distributive shock due to **sympathetic nervous system dysfunction**.

## Obstructive shock

**Obstructive shock** is the **least common** cause of shock in children and can be caused by an **acute obstruction to the pulmonary or systemic blood flow**. (This occurs when blood is unable to enter or leave the heart, despite normal intravascular volume and cardiac function. Both cardiac and pulmonary causes exist for obstructive shock, such as cardiac tamponade, tension pneumothorax, pulmonary hypertension, and coarctation of the aorta). The common causes of acute obstruction of pulmonary and systemic blood flow are cardiac tamponade, tension pneumothorax and massive pulmonary embolism.

Additionally, children with some **congenital cardiovascular malformations**, such as coarctation of the aorta or severe aortic valve stenosis, can also develop obstructive shock.

## Endocrinological shock

Children who have either recently completed a prolonged course of steroid therapy or are on chronic steroid replacement therapy are at high risk for endocrinological shock.

## Pathophysiology of Shock

The different consequences of shock can be attributed to either the **inadequate delivery of substrates**, such as glucose and oxygen, or **the removal of toxins from peripheral tissues**.

In a normal physiologic state, the cellular metabolism is dependent on glucose and oxygen where adenosine triphosphate can be generated by the mitochondria via aerobic

metabolism and the Krebs cycle.

When shock develops, the body can try to compensate via **gluconeogenesis** and **glycogenolysis**, but this is usually a limited compensation that fails. Due to the absence of oxygen in the shock state, **pyruvate** is converted to **lactate** instead of acetyl-CoA. This pathway generates two adenosine triphosphate molecules per one molecule of glucose and is associated with the **accumulation of lactate**.

The **inadequate production of adenosine triphosphate** and the **production of lactate** at the cellular level is associated with **impaired cell membrane ion pump function** and **acidosis**. **Cellular edema** eventually happens and **cellular death** can ensue if the shock state is not corrected.

Therefore, for cellular shock to happen, an impairment must occur in local tissue blood flow, the content of oxygen in the delivered blood, or the degree of oxygen demand in the peripheral tissue.

In normal physiologic states, we can compensate to increased oxygen demand by increasing the heart rate and the cardiac stroke volume. In shock states, we might try to compensate to the increased oxygen demand by increasing the oxygen extraction ratio but the total arterial flow of oxygen becomes less controlled. Increasing oxygen extraction usually fails in shock state and is associated with the **accumulation of lactate** in the blood and in **hypoxic injury** to the cells.

## Clinical Presentation of Shock in Children

An important part of history-taking in a child who presents with shock is the identification of the etiology of shock. Children who present with **vomiting**, **diarrhea** or both and have shock most likely have **hypovolemic shock** due to **intravascular fluid loss**.

Children with **penetrating trauma** can have **external hemorrhage** and might present with **hemorrhagic shock**.

On the other hand, children with **blunt trauma** can develop **internal bleeding**, which can also cause **hemorrhagic shock**.

Children who have a **high-grade fever** or who are **hypothermic** might have **septic shock**. Neonates and infants younger than 3 months can present with septic shock without a fever. Neonates who present with **hepatomegaly**, a **cardiac murmur** and shock most likely have **cardiogenic shock** due to a **congenital heart anomaly** that is ductal dependent.

In addition to the symptoms and signs of the most likely etiology, one should also look for symptoms and signs due to the shock itself. Children with shock are usually **lethargic**, have **decreased urinary output**, and might present with **poor feeding** or a **decreased level of consciousness**.

A **physical examination** is very important in any child who presents with symptoms and signs suggestive of shock. The goal of your physical examination should be to recognize the severity of the shock and further elucidate the probable causes of shock.

Shock in infants and young children is usually characterized by **tachycardia**, **decreased urinary output**, **altered mental status**, **weak peripheral pulses**, and a **capillary refill time of more than 2 seconds**.

**Cool clumsy extremities** can be seen in cases of severe shock. The child can have

**hyperthermia** due to **dehydration** or an **infectious process** or **hypothermia**.

Hypothermic children might not have tachycardia. **Tachypnea** and eventually **respiratory failure** can be seen in children with shock.

**Measuring the blood pressure** of the child who is in shock is very important as it can differentiate between compensated, decompensated and irreversible shock.

**Compensated shock** can be defined as a shock state that has all the signs and symptoms of shock except for hypotension. When the child develops **hypotension**, it means that he or she has presented late to the emergency department and urgent intervention is needed. Children with hypotension and multi-organ failure might be in the irreversible stage.

The American Heart Association has determined different cut-offs to define hypotension in children. Table 1 summarizes the fifth percentile systolic blood pressures for age which can be used to define hypotension in a child who presents with shock.

<b>Neonate</b>	60 mmHg
<b>1 month to 1 year</b>	70 mmHg
<b>1 year to 10 years</b>	70 mmHg + (2 x age in years) mmHg
<b>More than 10 years</b>	90 mmHg

**Table 1: Fifth percentile of systolic blood pressure in children based on their age**

## Diagnostic Workup for Shock in Children

Any child who presents with shock should be offered the **routine ABC treatment**:

Securing the airway, breathing and circulation before any further diagnostic workup is carried out. Once the child is stable, the diagnostic evaluation should aim to answer two main questions: **What is the severity and stage of shock, and, what is the most likely etiology of shock.**

The first line laboratory investigations in any child with shock are **serum levels of glucose, arterial blood gases, serum lactate levels, a complete blood count, prothrombin and partial thromboplastin times, fibrinogen and d-dimer levels, and fluid cultures.**

Additionally, based on the most expected cause, a **chest radiograph, cardiac output monitoring** or **B-type natriuretic peptide (BNP) levels** might need to be determined.

A **comprehensive metabolic panel (CMP)** should be ordered in any child who is in shock. The CMP can reveal **metabolic acidosis**, which can happen due to severe lactic acidosis, a sign of severe shock. **Hypovolemic shock** can present with **hypernatremia** on the CMP. Elevated blood urea nitrogen, creatinine, aspartate transaminase, and alanine transaminase suggest **kidney or hepatic dysfunction** due to **hypoxic-ischemic end-organ injury.**

**Near-infrared spectroscopy (NIRS)** is useful in the determination of the level of oxygenation in peripheral tissues and organs such as the kidneys. NIRS has become available in many pediatric intensive care units and is safe and non-invasive.

Because **sepsis** has been defined as the most common cause of shock in children and, due to its clear association with significant mortality and morbidity, **Sepsis Biomarkers Risk Models** were extensively studied in the recent decade. Five serum proteins were identified to be associated with significant mortality in children with sepsis: **C-C**

**chemokine ligand 3, heat shock protein 70 kDa 1B, interleukin-8, elastase 2 and lipocalin 2.**

## Treatment of Shock in Children

The goal of the treatment of shock in children should be to **correct the shock status of decreased oxygen and other substrate delivery** to the end-organs.

Based on this goal, any therapeutic plan for the management of shock should aim to achieve a normal mental status, a normal blood pressure for age, normal heart rate for age, a capillary refill time less than 2 seconds, a urine output greater than 1 ml/kg/h, normal serum glucose levels, normal serum ionized calcium levels and a decreasing serum lactate level.

**Central venous oxygen saturation** of more than 70% should be achieved while **managing septic shock** for optimum outcome.

When a child with shock presents to the emergency department, the first step in the management plan should be to determine the **mental status** of the child and the **degree of impaired peripheral perfusion**. The **airway should be secured** before attempting to restore a normal circulation.

Once the airway is secured and deemed patent, the next goal should be to **improve the peripheral and central circulation**. **Fluid expansion** is the mainstay of treatment and can be achieved by the **administration of 20 cc/kg isotonic saline or colloid** as a bolus.

In addition to the initial fluid resuscitation therapy, one should also **correct hypoglycemia and hypocalcemia**. Hypoglycemia can be corrected by the administration of dextrose water at 5 to 10 ml/kg of D10W, 2 to 4 ml/kg of D25W, or 1 to 2 ml/kg of D50W.

Children with hypocalcemia should receive calcium chloride 10% at the dose of 10 to 20 mg/kg and at a rate that is less than 100 mg/min. Children with normal ionized calcium levels while in shock should not receive calcium as this has been associated with increased mortality.

During the **early stage of fluid resuscitation** of a child in shock, the initial bolus of 20 ml/kg isotonic saline should be repeated up to three times over the period of 15 minutes. If the child remains in shock after three bolus infusions, it is very likely that the child is **hemorrhaging**.

In that case, **whole blood or packed red blood cells** might be indicated. Children who develop **rales** or **acute hepatomegaly** during the acute management of shock should be discontinued from fluid resuscitation therapy and **inotropic therapy** should be initiated.

Children with **cardiogenic shock** should receive an initial bolus of 5 to 10 ml/kg of isotonic saline instead of the typical 20 ml/kg dose. **Inotropic therapy** should be initiated in this cohort of patients as early as possible.

If, after 15 minutes of initial fluid resuscitation, the child starts improving, then the child should be admitted to the **pediatric intensive care unit** for further management. Children, who have **fluid refractory shock** should be initiated on **dopamine therapy** and a central venous access to assess **central venous pressure** should be obtained.

Children, who do not respond to **dopamine** or **fluid therapy** might benefit from **epinephrine** or **norepinephrine** administration.

When **septic shock** is suspected, **empirical antibiotic therapy** should be initiated within the first hour of the diagnosis of sepsis. Delayed antibiotics administration has been associated with **increased mortality** and **multi-organ failure** in children with septic shock. Whenever possible, **blood cultures** should be obtained before initiating antibiotic therapy.

The antibiotic regimen is dependent on the child's age. Neonates usually receive a combination of **ampicillin and gentamicin**. Infants and young children should receive a **third-generation cephalosporin combined with vancomycin**. Intravenous immunoglobulins are useless in the management of septic shock in children.

Children who fail to respond to inotropic therapy and fluid replacement therapy might have **pericardial effusion**, **pneumothorax** or **pulmonary embolism** as the etiology of their shock state. In that case, the urgent correction of these etiologies might be life-saving. Children without any apparent cause of fluid and inotropic refractory shock might have an **endocrine emergency**, such as hypothyroidism or adrenal insufficiency.

The administration of corticosteroids in children with septic shock is not supported by evidence. Children with **adrenocortical failure or infarction** should receive **hydrocortisone** in the dose of 50 to 100 mg/m<sup>2</sup>/day intravenously. The determination of **absolute or relative adrenal insufficiency** is difficult in children with shock, but some experts prefer to start corticosteroid replacement therapy when baseline cortisol levels are less than 20 microgram/dL.

## References

Pasman, Eric. **Shock In Pediatrics: Background, Pathophysiology, Etiology**. Emedicine.medscape.com. N.p., 2017. Web. 16 Apr. 2017.

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