

## PedsCases Podcast Scripts

This is a text version of a podcast from [PedsCases.com](http://PedsCases.com) on “[Approach to Shock](#).” These podcasts are designed to give medical students an overview of key topics in pediatrics. The audio versions are accessible on iTunes or at [www.pedcases.com/podcasts](http://www.pedcases.com/podcasts).

### **Approach to Shock**

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My name is Dustin Jacobson, a 3<sup>rd</sup> year pediatrics resident from the University of Toronto. This podcast was supervised by Dr. Suzanne Beno, a staff physician in the division of Pediatric Emergency Medicine at the University of Toronto. Today, we'll discuss an approach to shock in children.

First, we'll *define* shock and understand it's pathophysiology.

Next, we'll *examine* the subclassifications of shock.

Last, we'll *review* some basic and more advanced treatment for shock

But first, let's start with a case. Jonny is a 6-year-old male who presents with lethargy that is preceded by 2 days of a diarrheal illness. He has not urinated over the previous 24 hours. On assessment, he is tachycardic and hypotensive. He is febrile at 40 degrees Celsius, and is moaning on assessment, but spontaneously breathing. We'll revisit this case including evaluation and management near the end of this podcast.

The term “shock” is essentially a ‘catch-all’ phrase that refers to a state of inadequate oxygen or nutrient delivery for tissue metabolic demand. This broad definition incorporates many causes that eventually lead to this end-stage state.

Basic oxygen delivery is determined by cardiac output and content of oxygen in the blood. Blood flow and therefore oxygen delivery is modulated by systemic vascular resistance (SVR). And, in cases that meet the definition, or approach the definition for shock, compensatory mechanisms exist to increase SVR, which results in maintenance of blood pressure for vital organs (i.e. the heart and brain). However, even blood pressure that meets the normal values, (adjusted for age) may not meet increased metabolic demands. Therefore, hypotension can be present but is not a prerequisite for the definition of ‘shock.’ This is especially true in children, where blood pressure typically remains normal until late stages of shock. It's clear then that shock is a continuum, from early changes that our body has mechanisms to compensate for (termed compensated shock), all the way to tissue ischemia and irreversible tissue damage.

The many causes for shock are generally subclassified as hypovolemic, cardiogenic, septic and distributive or neurogenic shock. Let's talk about each of these in detail.

**Hypovolemic shock** is the most common cause of shock occurring in children and results from conditions such as bleeding, burns or gastrointestinal illnesses. Clinical signs and symptoms

can include tachycardia, tachypnea, impaired mental status, oliguria, delayed capillary refill and signs of low volume status, including orthostatic hypotension. Acidosis is also common.

**Cardiogenic shock** is due to a failure of cardiac output. This failure can be a result of arrhythmias, primary decreases in myocardial contractility such as in myocarditis, and other rare causes. Signs and symptoms of cardiogenic shock can include tachycardia or tachypnea, which are common to hypovolemic shock. Some distinguishing features to suggest an underlying primary cardiac cause may include a heart murmur or gallop rhythm, jugular venous distension, pulmonary rales and hepatomegaly. Additionally, point of care ultrasound (POCUS) is an evolving tool for the diagnosis of cardiogenic shock. Specifically, measures to estimate cardiac contractility and/or inferior vena cava size can definitively assess for cardiac function and volume status. A rare cause of cardiogenic shock can exist 'outside of the heart.' Specifically, impairment of filling by outside pressure in the form of cardiac tamponade or bilateral pneumothoraces or air trapping can decrease stroke volume and therefore, cardiac output.

**Distributive shock** is caused primarily by inappropriate vascular tone. The most common reason for this is seen in anaphylaxis, where an IgE mediated reaction results in the release of histamine and other vasodilators. Spinal trauma or anesthesia can result in loss of sympathetic tone; termed **neurogenic shock**. However, the latter of these conditions of deranged vascular tone is distinguished from anaphylaxis because of the absence of reflexive tachycardia. In rare cases, septic shock can present primarily with hypotension related to low vascular tone.

Moving onto **septic shock** which is defined in the pediatric population as a systemic inflammatory response accompanied by tachycardia and inadequate perfusion. Most importantly, one can only use the term sepsis when this systemic response is triggered by an infection. Cold vs warm shock refers to two types of presentations common to septic shock. 'Cold shock' describes a scenario wherein low cardiac output and high SVR results in cool extremities, prolonged capillary refill time and poor peripheral pulses; this is the more common presentation. In 'warm shock,' a scenario of high cardiac output and low SVR results in warm extremities, 'flash' capillary refill and 'bounding pulses.' In both types of shock, the primary issues of inadequate tissue perfusion for metabolic demands, and tachycardia are dominant. And, a patient may transition between these two forms of shock within the same course of illness.

Now onto treatment: the most important aspect of managing shock is rapid assessment, recognition and resuscitation. Hence, ABCDs (or airway, breathing, circulation & disability) are critical, along with the placement of appropriate monitors. Going through this acronym more explicitly, supplemental oxygen should be administered in most patients. Consider intubation for patients unable to protect their airway, those in which one is suspicious for impending respiratory failure, and when non-invasive oxygenation does not meet the requirements for tissue perfusion. Early intubation may be also considered to decrease metabolic demands. In cardiogenic shock, positive pressure ventilation can help decrease afterload on the left side of the heart but transitioning to positive pressure ventilation without adequate volume repletion can lead to decreased venous return and worsening cardiac output. If intubating, careful selections of medications is critical to avoid those that may exacerbate shock. Namely, benzodiazepines, opioids and propofol can all decrease blood pressure and result in circulatory collapse. Therefore, use of etomidate (not in septic shock), and/or ketamine as induction agents may be more prudent in unstable patients; and occasionally the only safe decision is to proceed with an intubation without the use of medications at all.

Although airway and breathing are earlier in the ABC acronym, equally important is maintaining appropriate circulation which should be accomplished in parallel to respiratory interventions. Rapid vascular access is critical, preferably with multiple wide bore intravenous lines if possible. But, one must recognize there are challenges with obtaining vascular access even in small children who are not volume deplete; thus *some access is better than no access*, and early use of umbilical venous catheters (in neonates) and intraosseous lines (in infants or children) should be considered. Central venous catheters for resuscitation, diagnosis of etiology and monitoring of therapy are helpful, but prolonged attempts to obtain these lines should not supersede rapid vascular access via intraosseous routes.

Once access is established, fluid resuscitation is next on the list. Current 2015 pediatric advanced life support (PALS) guidelines recommend an initial 20 ml/kg bolus of isotonic fluid for most patients. However, use of very liberal fluid administration, which was more traditionally taught as paramount, is coming more into question in recent years because of the risk of pulmonary edema and/or worsening of conditions associated with cardiogenic shock. There is much on-going interest and research in optimizing fluid volumes in hemorrhagic shock associated with trauma. Therefore, the decision to use copious fluids during resuscitation requires frequent reassessment and if possible, access to critical care facilities. More sophisticated equipment is often needed to monitor and guide further fluid resuscitation including central venous pressure, cardiac index and others. That being said, in many cases of hypovolemic or septic shock, patients may require up to 200 ml/kg of isotonic fluids.

Further, the choice of isotonic fluid is essential. Generally, crystalloids are suitable for the vast majority of presentations (for example, normal saline or ringer's lactate). Packed red blood cells along with other blood products (i.e. platelets and plasma) should be utilized early in cases of hypovolemic shock caused by acute blood loss.

Often going hand-in-hand with adequate fluid resuscitation is the use of inotropic agents. Agents such as dobutamine, dopamine and epinephrine work to increase cytoplasmic calcium concentration and increase myocardial contractility. Dopamine was considered first-choice for hypovolemic or septic shock, but recent evidence suggests epinephrine is likely superior and can be initiated through a peripheral intravenous line. Sometimes the choice for inotropic medications may rely on center preference and ease of use. The important point is the recognition that an inotropic agent is needed.

Often, inotropic agents are used in combination with vasopressor agents. Vasopressors are agents that lead to peripheral vasoconstriction, hence, increasing SVR. At higher doses, dopamine and epinephrine can act to increase SVR while dobutamine can have the opposite effect. Therefore, for states of low cardiac output and low SVR, dopamine and epinephrine may be helpful, however, in cases of acute cardiac failure, associated with low cardiac output and high SVR, dobutamine would be more helpful. Other vasopressors include phenylephrine and norepinephrine which increase SVR. Norepinephrine can also exert inotropic and chronotropic effects at higher doses.

Clearly, cardiogenic shock is managed different than other etiologies of shock, even though it may present similarly. In cardiogenic shock, vasodilators rather than vasopressors are often used to decrease afterload and improve coronary artery perfusion. Examples of vasodilators include nitroprusside and nitric oxide. Milrinone is another important medication that acts to vasodilate but also has inotropic activity. Prostaglandin E1 is another vasodilator, however its use is primarily restricted to maintaining a patent ductus arteriosus in ductal dependent cardiac

lesions in neonates. All of these agents may be combined with other inotropic agents to maintain systemic perfusion. This fine tuning is often very complex, and requires intensive monitoring, so is best accomplished in a critical care unit.

Corticosteroids are one last measure that may be considered in cases of catecholamine-resistant shock and/or in cases of potential adrenal insufficiency. Catecholamine-resistance refers to shock that does not improve with traditional inotropes.

As part of the initial resuscitation and management for patients presenting in septic shock, broad spectrum antibiotics should be given within the first hour of presentation, often referred to as the 'golden hour.' Along with antibiotics, obtaining cultures from blood, urine and potentially cerebrospinal fluid are important, but should not delay therapy. Often, a lumbar puncture to obtain CSF is deferred if the patient is unstable.

For patients whom are refractory to resuscitation, one last measure for support can include extracorporeal membrane oxygenation (ECMO). An ECMO circuit refers to a piece of equipment which delivers oxygen into the blood and pumps the blood through the body; taking over the job of the lungs and heart. ECMO is used as a last resort when the patient's condition cannot be supported adequately by conventional resuscitation. And, because these patients are in such dire straits when thinking about ECMO, overall survival rates are poor.

The D of ABCD refers to disability. Perform a quick neurological screen, including checking for pupil size or reactivity to look for a clue of the underlying diagnosis. Lastly, 'don't forget the glucose' – always check a blood glucose level in pediatric patients who experience a sudden deterioration or when there's an altered mental status.

Now that you know how to manage shock, let's come back to the case. Jonny is a 6-year-old male with likely hypovolemic shock secondary to a diarrheal illness. He is lethargic, tachycardic and hypotensive. As he is spontaneously breathing and protecting his airway, you further evaluate his breathing status and circulation. While you apply 100% oxygen via a non-rebreather mask, you instruct team members to insert two large bore IV catheters. If this is unsuccessful, you have an intraosseous insertion kit nearby. After access is secured, you start a 20 ml/kg normal saline bolus. Jonny's heart rate comes down slightly, and his blood pressure improves slightly. He has no pulmonary crackles on exam and his liver is non-palpable. Over the next 30-60 minutes, he receives two more boluses for a total of 60 ml/kg. During this time, he remains febrile and is given broad spectrum empiric antibiotics after blood and urine cultures are taken. A lumbar puncture is deferred due to Jonny's unstable status. Jonny's hypotension resolves, his mental status improves as he becomes awake and alert, he voids, his skin color normalizes, he becomes afebrile and his central and peripheral capillary refill is less than 2 seconds. Just in case of further hypotension or deterioration, an epinephrine infusion is prepared.

Before we go, let's summarize the key take away messages from this podcast.

- First, shock is a state of insufficient supply of oxygen for metabolic demand; subclassified into various etiologies including hypovolemic, cardiogenic, septic and distributive and neurogenic shock.
- Basic supportive care for children in shock includes managing the ABCDs which may include supplemental oxygen, securing an airway, fluids, and of course, frequent re-assessments.
- Inotropic or vasoactive medications may be needed along with appropriate antibiotic therapy and/or corticosteroids.

- Finally, ECMO may be considered in centers where its available and in select cases, may be appropriate.

Thank you for listening to this PedsCases podcast on shock in children.

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