Emergency Department Management of Pediatric Shock

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KEYWORDS

• Pediatric • Children • Shock • Hypotension • Sepsis • Vasopressors
• Emergency department

KEY POINTS

- Clinical history and physical examination findings are crucial for the early recognition and classification of shock in the pediatric patient.
- Hypotension is a late and ominous finding in the pediatric patient in shock.
- Rapid fluid resuscitation is the first line of treatment in most forms of shock.
- Three 20 mL/kg isotonic crystalloid boluses should be given within the first 20 to 60 minutes after shock is identified.
- Epinephrine is usually the preferred vasopressor in pediatric shock and should be started peripherally if central access is not present.

INTRODUCTION

Shock is a state of acute energy failure stemming from a decrease in adenosine triphosphate production and subsequent failure to meet the acute metabolic demands of the body. More simply put, it is a state of inadequate oxygen supply to meet the body's cellular demands. Hypoxemia or decreased perfusion results in decreased oxygen delivery to the tissues, causing a shift from more efficient aerobic pathways to anaerobic metabolism, resulting in the production of lactic acid. As oxygen deprivation persists, cellular hypoxia leads to the disruption of critical biochemical processes, eventually resulting in cell membrane ion pump dysfunction, intracellular edema, inadequate regulation of intracellular pH, and cell death.

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Oxygen delivery to the tissues is determined by cardiac output and arterial oxygen content. Cardiac output depends on heart rate and stroke volume. 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, which is greatly affected by systemic vascular resistance [SVR]); contractility (the force generated by the ventricle during systole), and lusitropy (the degree of myocardial relaxation during diastole). In children, compared with adults, cardiac output is more dependent on heart rate than stroke volume owing to myocardial immaturity, which limits the ability to increase contractility. Arterial oxygen content depends on hemoglobin concentration, arterial oxygen saturation, and the arterial partial pressure of oxygen, with most oxygen being carried on hemoglobin and a small portion delivered as dissolved O_2.[1]

Under normal conditions of increased oxygen demand, such as exercise, oxygen delivery must increase by redistribution of blood flow. Similarly, in pathologic instances of increased oxygen demand or decreased oxygen delivery (shock), initial compensatory mechanisms occur to preserve tissue perfusion. In compensated shock, vital organ function is maintained and blood pressure remains normal. In uncompensated shock, hypotension develops and organ and cellular function deteriorate. Left untreated, uncompensated shock progresses to irreversible shock, characterized by irreversible organ failure, cardiovascular collapse, cardiac arrest, and death.

Pediatric shock results in a significant amount of morbidity and mortality worldwide. Sepsis and hypovolemia owing to infectious gastroenteritis are leading causes of child mortality worldwide, with an estimated 3 to 5 billion cases of acute gastroenteritis and nearly 2 million deaths occurring each year in children under 5 years of age, with 98% of those deaths occurring developing countries.[2] In developed countries like the United States, shock is also a common occurrence in the emergency department (ED). These children have a higher mortality rate compared with patients not in shock (11.4% vs 2.6%). The presence of shock is also associated with worse outcomes in a variety of emergency conditions, including traumatic brain injury and cardiac arrest.[3,4]

CLASSIFICATIONS OF SHOCK

Several classifications of shock exist (**Table 1**). Rapid identification of the etiology may help to guide specific therapies.

Table 1
Categories of shock

Category	Hemodynamics	Causes
Hypovolemic	↓Preload, ↑SVR, ↓CO	Gastrointestinal loses, renal loses, hemorrhage, third spacing, burns
Distributive	↓Preload, ↓↓SVR, ↓↑CO	Sepsis, anaphylaxis, neurogenic shock
Cardiogenic	↑Preload, ↑SVR, ↓CO	Congenital heart disease, arrhythmia, cardiomyopathy, myocarditis, severe anemia
Obstructive	↓↑Preload, ↑SVR, ↓CO	Pulmonary embolus, pericardial tamponade, tension pneumothorax, certain congenital heart lesions

Abbreviations: CO, cardiac output; SVR, systemic vascular resistance.

Hypovolemic Shock

Hypovolemia is the most common cause of shock in children[5] and is a leading cause of child mortality worldwide. Hypovolemic shock occurs owing to inappropriately low intravascular blood volume (either owing to intravascular volume loss or hemorrhage), leading to decreased cardiac output. Additionally, hemorrhagic shock decreases oxygen-carrying capacity secondary to direct loss of available hemoglobin.

Intravascular volume loss can occur owing to gastrointestinal, renal, skin (ie, burns), or interstitial (ie, third spacing) losses. Hypovolemia can develop rapidly! Children with gastroenteritis can lose a significant percentage of their circulating volume within a few hours. Even if there is ongoing vomiting or diarrhea, it is usually preferable to attempt oral rehydration if dehydration is mild to moderate. Several studies including large metaanalyses have shown oral rehydration to be highly successful (<5% failure rate) and resulting in shorter ED stays and fewer adverse events compared with intravenous (IV) hydration.[6] If a patient shows signs of decreased end-organ function, however, forego attempts at oral rehydration and proceed to IV resuscitation. Capillary leak syndrome owing to sepsis, burns, or other systemic inflammatory diseases can result in profound intravascular volume loss in patients that may otherwise seem to be edematous and volume overloaded.

Hemorrhage may occur from traumatic or nontraumatic bleeding. Hemorrhagic shock can be further broken down into stages of severity based on percent volume loss and physical examination findings (**Table 2**). In an infant/toddler in shock with unclear etiology, consider occult hemorrhage owing to nonaccidental trauma.

Distributive Shock

In distributive shock, normal peripheral vascular tone becomes inappropriately relaxed. In this state, vasodilation results in effective hypovolemia, although a net fluid loss may not have actually occurred. Common causes of distributive shock include sepsis, anaphylaxis, neurologic injury (ie, spinal shock), or drug-related causes. In

Table 2
Classification of pediatric hemorrhagic shock by clinical signs

	Class I Very Mild Blood Loss (<15%)	Class II Mild Blood Loss (15%–30%)	Class III Moderate Blood Loss (30%–40%)	Class IV Severe Blood Loss (>40%)
HR	Normal to mildly increased	Tachycardic	Tachycardic	Severely tachycardic
Pulse quality	Normal	Peripheral pulses decreased	Peripheral pulses decreased	Central pulses decreased
Respiratory rate	Normal	Tachypneic	Tachypneic	Severely tachypneic
Mental status	Normal/slightly anxious	Anxious/irritable	Irritable/confused	Confused/lethargic/obtunded
Urine output	Normal	Decreased	Decreased	Anuric
Skin	Warm/pink	Cool/mottled	Cool/mottled/pallor	Cold/pallor/cyanotic

Adapted from American College of Surgeons. Advanced trauma life support (ATLS) study guide. Chicago (IL): American College of Surgeon; 2012; with permission.

sepsis, massive inflammatory response along with nitric oxide and cytokine release lead to peripheral vasodilation. In anaphylaxis, mast cell degranulation leads to vasodilatory cytokine release. In spinal shock, injury to the cranial portion of the spinal cord disrupts the sympathetic chain of the autonomic nervous system, resulting in unopposed parasympathetic vasodilation. Spinal shock, unlike most types of shock, often presents with bradycardia owing to unopposed vagal effects.

Sepsis is a common clinical syndrome that complicates severe infection and is characterized by immune dysregulation, systemic inflammation, microcirculatory derangements, and end-organ dysfunction. Sepsis is 10 times more common in children under 1 year than in older children and adolescents.[7] Pediatric sepsis is commonly encountered in the ED, and is a major cause of morbidity, mortality, and health care use costs worldwide. The American College of Critical Care Medicine (ACCM) defines septic shock as a clinical diagnosis made when children have suspected infection manifested by hypothermia or hyperthermia and clinical signs of inadequate tissue perfusion including any of the following: decreased/altered mental status, abnormal capillary refill time (CRT) or pulse characteristic, or decreased urine output (<1 mL/kg/h). Hypotension is not required for the clinical diagnosis of septic shock.[8]

Septic shock can present in one of two ways: cold shock or warm shock. Cold shock is characterized by high SVR resulting in cool/cold extremities, delayed CRT (<2 seconds), diminished peripheral pulses or differential between peripheral and central pulses, and narrow pulse pressure. Warm shock is characterized by low SVR, with warm/dry extremities with brisk ("flash") CRT, tachycardia, and bounding pulses with a wide pulse pressure.

Cardiogenic Shock

Cardiogenic shock can result from a variety of conditions that impair cardiac output. In children, cardiac failure is most commonly due to congenital heart disease, cardiomyopathies, or myocarditis. Additionally, arrhythmias can result in decreased cardiac output and shock.

Categories of heart failure and cardiogenic shock can be classified according to the presence/absence of 2 traits: venous congestion (owing to increased filling pressures) and hypoperfusion (owing to decreased cardiac output or myocardial contractility). This concept is summarized in **Box 1**. The presence of venous congestion is considered "wet" and the absence is described as "dry," and hypoperfusion is "cold" and normal perfusion is "warm." The wet patient may have findings including edema, hepatomegaly, ascites, jugular venous distension, S3 gallop, or crackles on lung auscultation owing to pulmonary edema. The cold patient may have cool extremities, weak pulses with narrow pulse pressure, delayed CRT, altered mental status, or hypotension.[9,10]

Obstructive Shock

Obstructive shock occurs when either pulmonary or systemic blood flow is impaired, resulting in impaired cardiac output. Causes of obstruction to heart function may be intracardiac or extracardiac and may be congenital or acquired. Examples include obstructive congenital heart lesions, cardiac tamponade, tension pneumothorax, massive pulmonary embolism, severe pulmonary hypertension, and hypertrophic cardiomyopathy. Obstructive shock in infants occurs when congenital lesions interfere with the outflow of blood from the heart, requiring the systemic output to be supplied by the pulmonary artery system via the ductus arteriosus. When the ductus closes within the first few days or weeks after birth, these infants present with severe shock. Obstructive shock generally requires prompt recognition with medical management

Box 1
Hemodynamic profiles in pediatric heart failure, classified by presence of hypoperfusion and/or venous congestion (increased filling pressures)

Warm and dry:

Normal perfusion and no congestion.

Well-compensated but may have significant cardiac dysfunction.

Cold and dry:

Poor perfusion without venous congestion. Decompensating. Sick appearing.

Increased peripheral vascular resistance.

May have oliguria and altered mental status.

Warm and wet:

Normal perfusion with venous congestion.

Still partially compensated.

May benefit from diuretics or inodilators.

Cold and wet:

Poor perfusion with venous congestion.

The sickest group of all.

Usually requires inotropes.

May require mechanical support.

(eg, initiation of prostaglandin therapy for a ductal-dependent lesion) and/or procedural management (eg, pericardiocentesis for tamponade or tube thoracostomy for tension pneumothorax).

RECOGNITION

Clinical history is important in children presenting to the ED in shock, and it may help to classify the etiology of shock and help direct therapies. Attention should be paid to past medical history and medication use (especially immunosuppression or steroid use). All infants under 3 months of age presenting in shock should be considered septic until proven otherwise. A history of fever or trauma may be particularly elucidative; however, often the history of a child in shock is often nonspecific with symptoms such as lethargy, fussiness, poor feeding, or decreased urine output.

Children are usually able to compensate for shock with tachycardia and increased SVR to maintain cardiac output and critical organ perfusion. Tachycardia is the most common presenting physical examination finding in pediatric shock. Persistent tachycardia in a calm, afebrile child should be concerning to the emergency provider and should prompt further investigation. For normal heart rates and blood pressure by age, see **Table 3**.[11] Increased SVR manifests as delayed CRT and diminished peripheral pulses. A recent metaanalysis showed that children with prolonged CRT have a 4-fold greater risk of dying compared with children with normal CRT. They found CRT to be highly specific, but not sensitive, for mortality.[12] Another study showed the combination of prolonged CRT and hypotension has a staggering mortality rate of 26.9%.[13]

When compensatory mechanisms fail, hypotension occurs. Guidelines define hypotension as a systolic blood pressure of less than the 5th percentile for age.[5] In addition

Table 3
Pediatric heart rate ranges and hypotensive systolic blood pressure levels by age

Age	HR (bpm)	Hypotensive SBP (mm Hg)
<1 mo	110–180	<60
1–12 mo	100–170	<70
1–2 y	85–150	<70 + (2 × age in y)
3–5 y	70–140	<70 + (2 × age in y)
6–10 y	60–110	<70 + (2 × age in y)
>10	50–100	<90

to hypotension, a child with decompensated shock will present with signs of inadequate end-organ perfusion, including depressed mental status, decreased urine output, metabolic acidosis, tachypnea, weak central pulses, and worsening peripheral perfusion. These signs of hypoperfusion are highly specific for the development of organ dysfunction, even in the absence of hypotension.[14]

ULTRASOUND EXAMINATION

Another useful tool increasingly used for the assessment of children in shock is point-of-care ultrasound (POCUS) examination.[15] The focused assessment with sonography for trauma (FAST) examination is used routinely in both pediatric and adult trauma to identify hemoperitoneum, hemopericardium, and hemothorax (plus pneumothorax in the extended e-FAST). One small study found that, when combined with increased liver transaminases of greater than 100 IU/L, the specificity of the FAST examination was 98%, suggesting a negative FAST and transaminases of less than 100 IU/L have a low likelihood of significant intraabdominal injury and should prompt patient observation instead of abdominal computed tomography scanning.[16] Although standard measurements of the inferior vena cava and aorta are not established in children (as they are in adults), in the evaluation of pediatric hypovolemic shock, both the ratio of the aorta to inferior vena cava and the dynamic assessment of inferior vena cava collapsibility have been studied and both metrics may correlate with hydration status.[17–19]

In the adult emergency medicine/critical care literature, several POCUS algorithms for the assessment of shock exist, and may dramatically affect treatment decisions and improve survival.[20–24] Evidence for the use of POCUS for the assessment of pediatric cardiac function, volume status, and shock management has lagged behind that for adult patients, yet the concepts remain similar. Clearly, further studies are needed in this area.

TREATMENT

Because shock is a problem of inadequate oxygen delivery, every child in shock should be given supplemental oxygen. Place the child on continuous cardiorespiratory and pulse oximetry monitors and obtain peripheral IV access as soon as possible. Check blood sugar and correct hypoglycemia if present. Hypocalcemia (ionized calcium <1.1 mmol/L) may contribute to cardiac dysfunction and should also be corrected.

Fluid Resuscitation

Isotonic crystalloid solutions are the fluid of choice for resuscitation of a child in shock. Crystalloids are preferred because of their safety, effectiveness, low cost, and wide

availability.[25] In less common circumstances, such as in resource-limited settings in developing countries with a high incidence of malaria, anemia, and malnutrition, take caution with IV fluid resuscitation and consider use of colloid (5% albumin) or early transfusion for suspected anemia.[26,27] Treat signs of shock with a fluid bolus of 20 mL/kg, even if blood pressure is normal, and give additional boluses if systemic perfusion fails to improve. In neonates or children with suspected cardiogenic shock, use 10 mL/kg boluses and reassess the patient frequently for signs of volume overload, including hepatomegaly, S3 gallop, or pulmonary rales/crackles. Volume resuscitation in hypovolemia and sepsis commonly requires 40 to 60 mL/kg, but may require as much as 200 mL/kg. POCUS may be useful to help determine if shock is still volume responsive. It is generally accepted that children remaining in shock after 60 mL/kg of IV fluid should be started on vasopressors.

Fluid administration in shock should be as rapid as possible. In infants and children with smaller gauge IVs, a "push/pull" method should be used. Push/pull uses a 3-way stopcock to manually draw a large syringe of fluid from the IV bag (pull) and then rapidly deliver it to the patient (push), and then repeat this process until the full volume is delivered. In 1 trial, fluid administration rates were equivalent in children using a pressure bag versus push/pull system, and both were faster than gravity or an IV infusion pump. Investigators have shown that 20 mL/kg of fluid can be delivered in 5 minutes or less via pressure bag or push methods.[28]

Although placement of a central venous line (CVL) is common in resuscitation in adults, this is unnecessary in children, at least in the initial stages. For the management of a child in shock, the goal should be placement of PIVs of the largest bore possible. If the child is in extremis and without access, intraosseous access should be placed without delay.

Vasoactive Medications

When shock remains refractory to fluid resuscitation, vasoactive infusions should be initiated (**Box 2**). Although infusion of vasoactive medications through a CVL is preferred, placement may be difficult in children. If the child is in fluid-refractory shock,

Box 2
Usual dosing ranges for vasoactive medications

Inotropes

Epinephrine 0.05 to 1.00 (or more) μg/kg/min

Dopamine: 5 to 20 μg/kg/min

Dobutamine 5 to 20 μg/kg/min

Vasopressors

Norepinephrine 0.05 to 0.50 (or more) μg/kg/min

Dopamine 10 to 20 μg/kg/min

Vasopressin 0.0005 to 0.0100 U/kg/min

Inotropes increase cardiac contractility. Vasopressors cause vasoconstriction, increasing systemic vascular resistance. Some medications fit into both categories. Start at the low end of the range and titrate rapidly until shock reversal is achieved. If administering via peripheral an intravenous line, dilute the solution (usually 10× the usual central concentration). Additional "driver" fluid (3–5 mL/h of saline) may be needed if the infusion rate is very low (<1 mL/h).

start vasopressors through whatever line is available (peripheral IV access, intraosseous access, or a CVL). A recent small study of peripheral vasoactive medication use in children in a pediatric intensive care unit found IV infiltration and extravasation to occur in only 2% of patients, with none requiring medical or surgical intervention.[29] Another larger study in adults found similarly low rates of complications with the peripheral administration of vasoactive medications, including norepinephrine, dopamine, and phenylephrine.[30] If a peripheral IV is used for vasopressor administration, the medication solution should be diluted and the IV site should be assessed frequently for problems. The use of peripheral vasopressors may be particularly relevant in children requiring transport to a higher level of care. Transport should not be delayed for CVL placement.[31]

The choice of which vasoactive medication to use depends on the clinical picture. Dopamine has long been the initial medication of choice in pediatric shock; however, several recent studies in both adults and children have challenged this dogma.[32–34] In adults, dopamine is associated with increased mortality and occurrence of arrhythmias compared with norepinephrine.[32] Two recent pediatric studies randomized epinephrine versus dopamine use in septic shock. One showed children receiving epinephrine versus dopamine for fluid-refractory septic shock had a lower mortality rate (7% vs 20%). Both groups used peripheral IVs for the initiation of vasoactive medications until central lines could be placed.[33] The other study did not show a difference in mortality, but children in the epinephrine group had faster resolution of shock and less organ dysfunction than those receiving dopamine.[34] In response to this study and other data, the newest ACCM guidelines recommend epinephrine as first-line treatment for cold fluid-refractory shock, with dopamine use (5–10 μg/kg/min) reserved for when epinephrine is unavailable.[8]

Warm shock is seen much less commonly in children than adults (for whom warm shock predominates). For children in warm shock, norepinephrine is recommended as first-line therapy. Dopamine may be used if norepinephrine is unavailable, and generally requires higher doses than in cold shock (10–20 μg/kg/min). In adults with septic shock, vasopressin levels are frequently low and this finding is thought to contribute to vasodilation. This state has not been found consistently in children, and trials of vasopressin for shock have failed to show benefit.[35] However, vasopressin remains available as an adjunctive therapy for refractory vasodilatory shock not responsive to norepinephrine.

Intubation

Airway management and ventilatory support is often necessary in children in shock. Often underrecognized, intubation of a child in shock may be indicated for hemodynamic instability alone. A significant portion of a child's oxygen consumption (up to 40%) goes into the work of breathing. Support with mechanical ventilation can reduce this oxygen consumption and divert critical cardiac output to vital organs. Care must be taken to fluid resuscitate (and sometimes even start on peripheral vasoactive medication) as best as possible before intubation because initiation of positive-pressure ventilation will decrease venous return and exacerbate hypotension. In a child with decreased cardiac function, the increased intrathoracic pressure associated with mechanical ventilation will afterload reduce the left ventricle and improve cardiac output.

Consider the etiology of shock when choosing intubation medications. Although etomidate has been shown to facilitate endotracheal intubation in infants and children with minimal hemodynamic effect, it is not recommended for use in patients with suspected sepsis owing to its adrenal-suppressive effects. In children and

adults with septic shock, use of etomidate is associated with increased mortality.[36,37] Ketamine has a favorable hemodynamic profile, but without the adrenal suppression, and is the recommended choice for children in septic shock. For shock without sepsis, such as in trauma, the choice of either medication is reasonable.

Antibiotics

When sepsis is suspected, administer broad-spectrum antibiotics within the first hour of presentation. In a study examining adult patients with sepsis, each hour of delay in antibiotic administration was associated with a mean decrease in survival of 7.6%.[38] If possible, obtain cultures to identify the source of infection before antibiotic delivery. Antibiotics should not be delayed if there is difficulty obtaining specimens. Factors such as local antibiotic resistance patterns, recent antibiotic use, existing immunosuppression, drug allergies, and suspected source of infection may influence what antibiotic is chosen.

Steroids

If shock persists despite escalating vasoactive medication doses (catecholamine-resistant shock), consider the adjunctive use of stress-dose corticosteroids. Patients with known or suspected adrenal insufficiency (ie, steroid use within the last 6 months, known pituitary or adrenal abnormalities, or sepsis with purpura fulminans) should receive stress-dose hydrocortisone as soon as possible after shock is identified. Evidence for the use of steroids in pediatric shock is limited and demonstrates conflicting results. In 1 study, children with sepsis who received corticosteroids had no improvement in mortality, days of vasoactive infusion, or hospital duration of stay.[39] A recent metaanalysis showed no difference in mortality rates between those who did and did not receive steroids.[40] For catecholamine-resistant shock, hydrocortisone dosing of 50 to 100 mg/m^2/d or 2 to 4 mg/kg/d is generally used, although some investigators advocate for doses as high as 50 mg/kg/d in refractory shock.[8] Ideally, a baseline cortisol level should be drawn before hydrocortisone dosing.

RESUSCITATION ENDPOINTS

Reversal of shock depends on the reestablishment of sufficient oxygen delivery to the body. The Surviving Sepsis Campaign identifies these therapeutic endpoints for resuscitation of pediatric shock: restoration of a CRT of less than 2 seconds, normal blood pressure for age, normal pulses, warm extremities, normal urine output, and normal mental status.

Goal-Directed Therapy

In addition to clinical resuscitation endpoints, the Surviving Sepsis Campaign and ACCM recommend that resuscitation of children in septic shock should target a mixed venous saturation (SvO_2) of 70% or greater, a perfusion pressure (mean arterial pressure – central venous pressure) of 55 + 1.5 $\times$ age in years, and cardiac index between 3.3 and 6.0 L/min/m^2. Low cardiac output is associated with increased mortality in children with septic shock; a cardiac index between 3.3 and 6.0 is associated with the best outcomes in pediatric septic shock patients compared with patients without shock for whom a cardiac index above 2.0 L/min/m^2 is sufficient. Cardiac output measurement can be measured invasively or noninvasively with a variety of devices. Additionally, to maximize oxygen and glucose delivery to help reverse

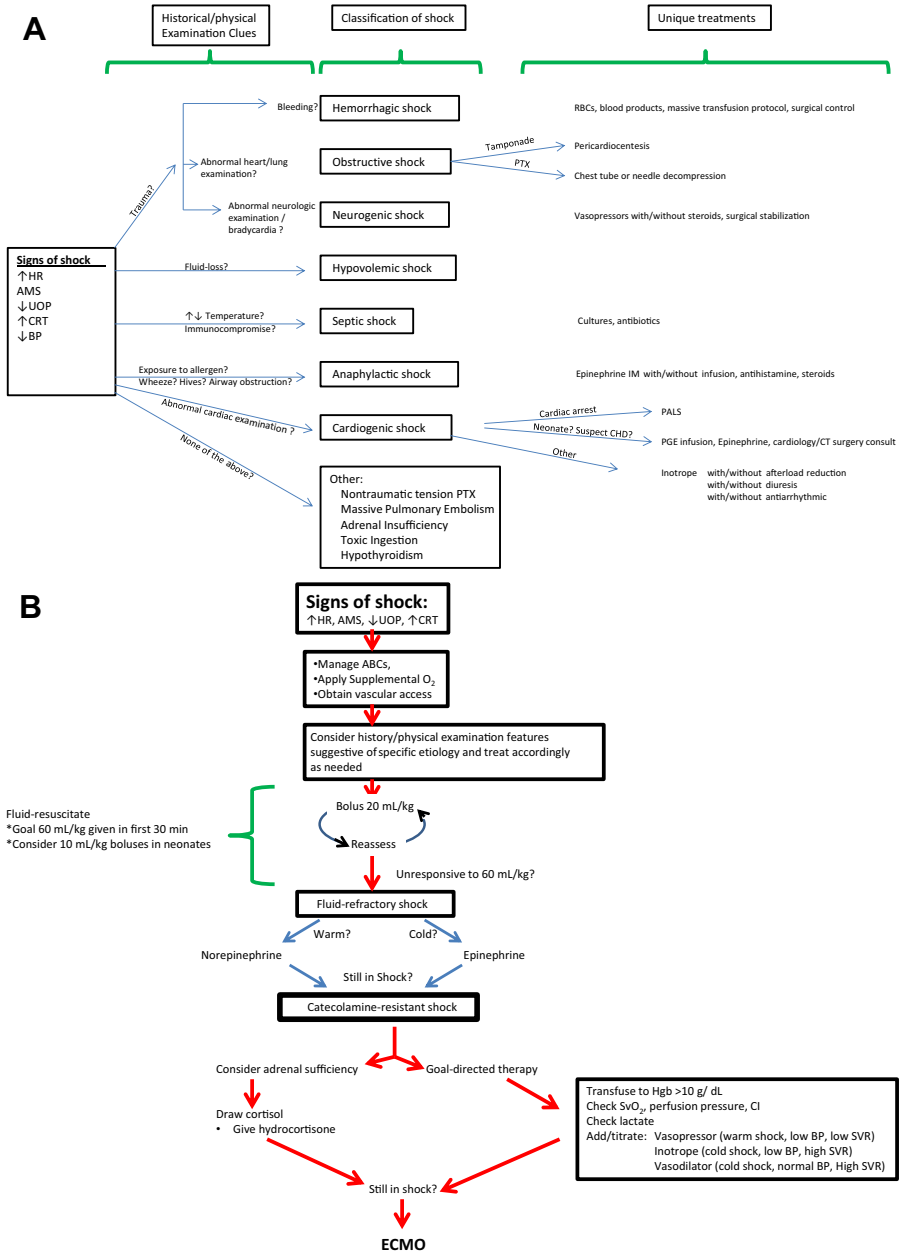

Fig. 1. Algorithmic approach to the pediatric shock patient. (A) Recognition/classification of pediatric shock. (B) Treatment of pediatric shock. For all patients, (1) Manage ABC's. (2) Apply supplemental O2 & obtain vascular access. (3) Use history/physical exam +/- POCUS to classify shock and guide treatment. (4) Frequently reassess response to treatment. (5) Clinical goals = normalization of heart rate, mental status, perfusion, blood pressure, urine output. Signs of shock: ↑HR = tachycardia; ↓UOP = decreased urine output; ↑CRT = delayed capillary refill time; ↓BP = hypotension; ABCs, airway, breathing, circulation; AMS, altered mental status; BP, blood pressure; CHD, congenital heart disease; CI, cardiac index; CT, computed

shock, the ACCM recommends transfusion to a hemoglobin concentration of greater than 10 g/dL and the administration of maintenance fluids containing D10 (D10 normal saline or D10 ½ normal saline).[8,41] In the Surviving Sepsis Campaign's nonpediatric recommendations, a lactate concentration 4 mmol/L or greater is identified as a key marker of tissue hypoperfusion, and normalization of lactate is a key resuscitation goal. Several pediatric studies have shown that increased lactate levels and failure to clear lactate correlate with mortality and organ dysfunction.[42,43] Lactate clearance, however, was notably excluded from the pediatric guidelines as a resuscitation endpoint based on the observation that many children in shock have normal lactate levels as well as the fact that lactate may be increased for many reasons other than cellular hypoxia.[8]

Targeted resuscitation has its foundation in the classic Rivers' early goal-directed therapy (EGDT) trial, which showed a significant mortality benefit when specific resuscitation goals were used in the ED management of adults with septic shock.[44] A pediatric trial of EGDT found significant mortality reduction and decreased organ dysfunction when resuscitation was titrated using SvO_2 goals.[45] However, EGDT (particularly the requirement for invasive CVP measurement and continuous SvO_2 monitoring) has lost some support after 3 recent large methodologically robust trials in adults with septic shock comparing EGDT with usual care showed no benefit in either mortality or secondary clinical and economic outcomes.[46]

Resuscitation to specific EGDT goals may eventually go by the wayside in pediatric algorithms, but for now the ACCM continues to advocate for the titration of therapies to SVO_2, perfusion pressure, and cardiac index goals. In the initial ED management, if invasive monitoring is not used, then usual care must mean vigilant, attentive care. Early recognition with prompt delivery of IV fluids and antibiotics and frequent reassessment is critical. Consider trending lactate levels and using noninvasive methods such as POCUS to assess the adequacy of resuscitation.

PUTTING IT ALL TOGETHER

When a child presents to the ED with tachycardia and signs/symptoms of shock, the most immediate concern should be stabilization of the airway, breathing, and circulation, followed by a rapid assessment of historical clues, physical examination findings, and laboratory studies that may aid classification and help to guide treatment. Refer to **Fig. 1** for an algorithmic approach to pediatric shock management. Some types of shock require specific therapies. Most shock requires some degree of fluid resuscitation, but be cautious if there is concern for a cardiogenic etiology. If shock remains refractory to fluids, add inotropes and/or vasopressors. If catecholamine-resistant shock occurs, advanced hemodynamic monitoring may be required to help to titrate therapies. Consider hydrocortisone supplementation. At multiple points along the way, POCUS may assist diagnosis and help to guide therapies including assessment of preload, fluid responsiveness, and cardiac function. At each step, reassess for response to treatment.

tomography; ECMO, extracorporeal membrane oxygenation; Hgb, hemoglobin concentration; IM, intramuscular; PALS, pediatric advanced life support guidelines; PGE, prostaglandin E infusion; POCUS, point of care ultrasound examination (used to help diagnose reasons for shock and assess volume responsiveness and cardiac function); PTX, tension pneumothorax; RBCs, red blood cells; SvO_2, venous oxygen saturation; SVR, systemic vascular resistance.

SUMMARY

Shock is an unstable pathophysiologic state of inadequate tissue perfusion that must be identified and treated promptly. Failure to recognize and reverse shock can have catastrophic results. In the ED, initial therapies should be titrated to normalize vital signs and physical examination abnormalities. If initial resuscitation with fluids and vasoactive medications do not reverse the shock state, advanced hemodynamic monitoring may be required to guide treatment (goal-directed therapy). Early recognition and resuscitation can improve mortality and outcomes for pediatric shock patients.

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