Initial evaluation of shock in children

AUTHOR: Mark Waltzman, 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 initial evaluation of children with shock and focus on the clinical features that identify and classify shock in resource-abundant settings and can be used to evaluate response to treatment. Additional considerations for the recognition of shock in children in resource-limited settings are provided separately. (See "Shock in children in resource-limited settings: Recognition".)

The physiology, classification, and initial management of pediatric shock, including the evaluation and management of specific types of pediatric shock are discussed separately:

- (See "Pathophysiology and classification of shock in children".)
- (See "Shock in children in resource-abundant settings: Initial management".)
- (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis".)
- (See "Children with early and life-threatening sepsis in resource-abundant settings: Rapid recognition and initial resuscitation (first hour)" and "Sepsis and septic shock in children in resource-abundant settings: Ongoing management after resuscitation".)
- (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management".)

DEFINITION

Shock is a dynamic and unstable pathophysiologic state characterized by inadequate tissue perfusion. Shock develops as the result of conditions that cause decreased intravascular volume, abnormal distribution of intravascular volume, and/or impaired cardiovascular

function. Aggressive treatment within the first few hours after presentation may prevent the invariable progression and poor outcome that characterize the natural clinical course of shock, manifested clinically by end-organ damage, failure of multiple organ systems, and death. (See "Pathophysiology and classification of shock in children", section on 'Physiology' and "Pathophysiology and classification of shock in children", section on 'Stages of shock'.)

EPIDEMIOLOGY

Clinical experience suggests that frequent causes of shock among children include hypovolemia from diarrheal disease or traumatic hemorrhage and sepsis:

- Hypovolemia from gastroenteritis is the most common cause of pediatric shock worldwide [1]. Widespread use of oral rehydration therapy has substantially reduced mortality in resource-poor countries, although this intervention continues to be underutilized in some developed countries. (See "Oral rehydration therapy", section on 'Efficacy'.)

- Trauma, including hemorrhagic shock, also remains a significant cause of death in children [2].

- In addition, sepsis occurs frequently in children around the world, especially low birth weight newborns and infants younger than one month of age, immunosuppressed patients, and children with chronic debilitating disease. (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Epidemiology'.)

Cardiogenic and obstructive shock occur much less frequently in children. However, clinicians must consider these etiologies since they are typically not fluid responsive and require specific therapies [3].

Common causes of cardiogenic shock in children include (table 1) [3]:

- Sepsis (see "Children with early and life-threatening sepsis in resource-abundant settings: Rapid recognition and initial resuscitation (first hour)", section on 'Patients with fluid-refractory shock')

- Congenital heart disease and heart failure (see "Heart failure in children: Etiology, clinical manifestations, and diagnosis")

- Myocarditis (see "Clinical manifestations and diagnosis of myocarditis in children", section on 'Clinical manifestations')

- Cardiomyopathy (see "Familial dilated cardiomyopathy: Prevalence, diagnosis and treatment", section on 'Diagnosis of familial DCM')
- Poisoning or drug toxicity (table 2)
- Brady- or tachyarrhythmias (see "Pediatric advanced life support (PALS)", section on 'Bradycardia algorithm' and "Pediatric advanced life support (PALS)", section on 'Tachycardia algorithm')
- Thoracic trauma with blunt cardiac injury (see "Overview of intrathoracic injuries in children", section on 'Blunt cardiac injury')

Obstructive shock can be caused by cardiac tamponade, tension pneumothorax, ductal dependent congenital cardiac lesions, or massive pulmonary embolism (table 1). (See "Pathophysiology and classification of shock in children", section on 'Obstructive shock'.)

PATHOPHYSIOLOGY

The physiologic determinants and stages of shock are discussed separately. (See "Pathophysiology and classification of shock in children", section on 'Physiology' and "Pathophysiology and classification of shock in children", section on 'Stages of shock'.)

Shock can develop from a variety of conditions that result in the following:

- Insufficient circulating blood volume (preload)
- Changes in vascular resistance (afterload)
- Heart failure (contractility)
- Obstruction to blood flow

The pathophysiologic consequences of inadequate circulation may be compounded by conditions (such as fever from infection or increased work of breathing from injury) that increase tissue metabolic needs.

Deleterious effects of decreased tissue perfusion include [4]:

- Poor perfusion of vital organs results in impaired function. For example, inadequate perfusion of the brain and kidneys can cause depressed mental status and low urine output, respectively.
- Lactic acid accumulates as cells switch to anaerobic metabolism to generate energy. Increased lactic acid in tissues causes metabolic acidosis, which interferes with cell and organ function.

- Hypoperfusion initiates inflammatory events (such as the activation of neutrophils and release of cytokines) that disrupt the microcirculation and contribute to tissue injury. Adrenergic stress responses that are activated to compensate for decreased tissue perfusion and increased metabolic demand include the following:

  - Blood flow to vital organs is preserved through stimulation of the heart (tachycardia and increased contractility) by the sympathetic nervous system and increased peripheral vasoconstriction (increased systemic vascular resistance [SVR] and venous tone) mediated by the sympathetic nervous and renin-angiotensin systems.

  - Hormones such as catecholamines, corticosteroids, and glucagon initiate increased liver glycolysis and lipolysis to maintain cell energy sources, causing an increase in lactic acid production.

The classification of shock is based upon the physiologic mechanisms that result in decreased tissue perfusion (table 3). This classification has important consequences for management decisions. (See "Pathophysiology and classification of shock in children", section on 'Classification' and "Shock in children in resource-abundant settings: Initial management", section on 'Further management by type of shock'.)

Classification of pediatric shock and typical causes include the following [3]:

- **Hypovolemic shock** – Hypovolemic shock is the most common type of shock in children. Causes include fluid and electrolyte loss (as from gastroenteritis or osmotic diuresis), hemorrhage (as from trauma), capillary leak (as from bowel obstruction or burns), inadequate fluid intake, and insensible losses (eg, fever, tachypnea, or burns). (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'Etiology'.)

  By definition, preload is decreased in hypovolemic shock. SVR may be increased as the result of compensatory mechanisms. Cardiac contractility is typically normal. (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'Pathophysiology'.)

- **Distributive shock** – Distributive shock physiologically refers to a condition in which SVR is initially decreased. It may occur as the result of sepsis, anaphylaxis, or neurologic injury. (See "Pathophysiology and classification of shock in children", section on 'Distributive shock'.)

  With sepsis and anaphylaxis, volume depletion may also develop because of losses related to the underlying infection (septic shock), or inflammatory cascade (anaphylaxis). Both processes are associated with increased capillary permeability with

loss of plasma from the intravascular space into the tissues. Myocardial dysfunction can also contribute to poor tissue perfusion.

In septic shock, abnormal distribution of blood flow as the result of changes in vasomotor tone causes inappropriate tissue perfusion (such as decreased splanchnic circulation with increased flow to skin and muscle). SVR may be low, producing increased blood flow to skin and a wide pulse pressure (warm shock) or SVR may be increased, in which case, blood flow to skin is decreased and the pulse pressure is narrow (cold shock). (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Clinical manifestations'.)

Neurogenic shock may develop in a child with a high spinal cord injury (above the sixth thoracic spinal level) [3]. Uncontrolled vasodilation occurs as the result of the sudden loss of sympathetic tone. Compensatory sympathetic mechanisms (such as tachycardia and peripheral vasoconstriction) are absent. (See "Evaluation and acute management of cervical spine injuries in children and adolescents", section on 'Physical examination'.)

- **Cardiogenic shock** – Cardiogenic shock results from pump failure because of intrinsic cardiac disease (eg, congenital heart disease, myocarditis, myocardial contusion, myocardial ischemia, cardiomyopathy, or arrhythmia (table 1)). Physiologic features of cardiogenic shock include tachycardia, increased SVR, and decreased cardiac output [3]. (See "Pathophysiology and classification of shock in children", section on 'Cardiogenic shock'.)

- **Obstructive shock** – Obstructive shock describes physical obstruction of systemic blood flow from the heart which causes abrupt impairment of cardiac output (table 1). Causes of obstructive shock include cardiac tamponade, tension pneumo- or hemothorax and massive pulmonary embolism. Infants with ductal-dependent congenital heart lesions, such as coarctation of the aorta and hypoplastic left ventricle syndrome, may also present in shock when the ductus arteriosus closes during the first few weeks of life. Conditions that cause obstructive shock must be recognized quickly because they generally require specific treatment. (See "Pathophysiology and classification of shock in children", section on 'Obstructive shock' and "Approach to the ill-appearing infant (younger than 90 days of age)", section on 'Initial stabilization'.)

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]. (See 'Clinical classification of shock' below.)

EVALUATION

Children can compensate for circulatory dysfunction (primarily by increasing heart rate, systemic vascular resistance [SVR], and venous tone) and maintain normal blood pressures despite significantly compromised tissue perfusion. Consequently, hypotension is a very late and ominous finding [3,5]. The challenge for the clinician is to recognize children in shock early (before they develop hypotension), when they are more likely to respond favorably to treatment.

Although the cause of shock may not be initially apparent, treatment must begin immediately. A systematic approach to the evaluation of children with evidence of poor perfusion typically identifies features of the history, physical examination, and ancillary studies that suggest the underlying condition (algorithm 1).

The goals of the initial evaluation of shock in children include:

- Immediate identification of life-threatening conditions (eg, tension pneumothorax, hemothorax, cardiac tamponade, or pulmonary embolism)
- Rapid recognition of circulatory compromise
- Early classification of the type and cause of shock

The initial management of undifferentiated shock is provided in the algorithm and discussed in detail separately (algorithm 2). (See "Shock in children in resource-abundant settings: Initial management".)

A general approach to the assessment of airway, ventilatory, and circulatory function in children is reviewed separately. (See "Initial assessment and stabilization of children with respiratory or circulatory compromise", section on 'Initial assessment' and "Technique of emergency endotracheal intubation in children" and "Assessment of systemic perfusion in children".)

Rapid assessment — The pediatric assessment triangle (PAT) provides a quick evaluation of appearance, breathing, and circulation for acutely ill or injured children that should identify conditions that require immediate intervention.

Features of the PAT that are specific for the evaluation of shock include:

Appearance — Significant changes in appearance (such as poor tone, unfocused gaze, or weak cry) may be indicators of decreased cerebral perfusion. Subtle differences in appearance (such as decreased responsiveness to caretakers or painful procedures) may also be important indicators of shock.

Breathing — A child with depressed mental status as the result of shock may not be able to maintain a patent airway. Tachypnea without respiratory distress can develop in response to metabolic acidosis. Children with cardiogenic shock typically have some increased work of breathing in addition to tachypnea.

Children with severe respiratory distress and signs of circulatory compromise may have obstructive shock and require life-saving interventions to treat one of the following:

- **Tension pneumothorax** – Signs of tension pneumothorax include respiratory distress, decreased breath sounds over the involved hemithorax, subcutaneous air, and distended neck veins. Children with tension pneumothorax frequently have sustained thoracic trauma, although a spontaneous pneumothorax can also develop tension. Treatment consists of needle decompression followed by chest tube thoracostomy. (See "Thoracic trauma in children: Initial stabilization and evaluation", section on 'Initial rapid assessment' and "Thoracic trauma in children: Initial stabilization and evaluation", section on 'Chest decompression'.)

- **Cardiac tamponade** – Features that may be seen with cardiac tamponade include respiratory distress, muffled heart tones, pulsus paradoxus, and distended neck veins. Cardiac tamponade can occur as the result of penetrating thoracic trauma or from the accumulation of fluid as the result of infection, malignancy, or following cardiac surgery (movie 1). Pericardiocentesis is emergently indicated. (See "Causes of acute respiratory distress in children", section on 'Cardiac tamponade' and "Emergency pericardiocentesis".)

- **Ductal-dependent congenital heart disease** – Infants with ductal-dependent congenital heart disease may have respiratory distress, circulatory collapse, cyanosis not responsive to oxygen administration, cardiomegaly, and/or a pulse or blood pressure gradient between the upper and lower extremities which may occur abruptly as the ductus arteriosus closes. Although timing varies, it is most common in neonates between one and three weeks after birth. Identification and management of infants with congenital heart disease is discussed separately. (See "Evaluation of suspected critical congenital heart disease (CHD) in the newborn" and "Cyanotic congenital heart disease (CHD) in the newborn: Causes, evaluation, and initial management", section on 'Initial management'.)

- **Massive pulmonary embolism (PE)** – Findings that suggest PE include cyanosis, respiratory distress, and pleuritic chest pain. PE occurs uncommonly in children. There is typically a predisposing condition (such as a central venous access device or an inherited hypercoagulable state). (See "Venous thrombosis and thromboembolism (VTE) in children: Risk factors, clinical manifestations, and diagnosis", section on 'Pulmonary embolism'.)

Circulation — Poor perfusion can often be identified rapidly, before a blood pressure measurement is taken. Features of circulation that should be quickly evaluated include:

- **Quality of central and peripheral pulses** – Decreased intensity of distal pulses in comparison to central pulses suggests peripheral vasoconstriction and compensated shock. (See "Assessment of systemic perfusion in children", section on 'Pulses'.)

  Bounding pulses may be present in patients with distributive ("warm") shock.

- **Skin temperature** – Skin may be mottled or cool in children with compensated shock, but this finding can also be influenced by environmental temperature.

- **Capillary refill** – Capillary refill greater than two seconds suggests shock [6]. The usefulness of capillary refill is limited by interobserver variability and by the effect of environmental temperature. (See "Assessment of systemic perfusion in children", section on 'Capillary refill time'.)

  Flash capillary refill (<1 second) may be present in patients with distributive ("warm") shock.

- **Heart rate** – Tachycardia is frequently present (table 4) although a normal or low heart rate with signs of compensated or hypotensive shock can occur with cervical or high thoracic spinal cord injury.

  Hypoxia and some poisonings (eg, ingestion of beta blockers, calcium channel blockers, cardiac glycosides, opioids, or benzodiazepines) can cause bradycardia (table 2). Bradycardia can also be an agonal event for patients with shock from any cause.

- **Blood pressure** – 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. Ideally, shock is recognized before hypotension occurs by identifying tachycardia and signs of organ hypoperfusion such as skin changes (prolonged capillary refill) and decreased urine output.

  For children, hypotension is defined as a systolic blood pressure that is less than the fifth percentile of normal for age. We use the age-related criteria provided by the Pediatric Advanced Life Support Course (see "Pathophysiology and classification of shock in children", section on 'Common features'):

  - Term neonates (0 to 28 days): <60 mmHg
  - Infants (1 month to 12 months): <70 mmHg

- Children 1 to 10 years old: <(70 mmHg + [2 x age in years])
- Children ≥10 years old: <90 mmHg

Point of care ultrasonography (POCUS), where available, may also provide essential information to guide life-saving procedures (such as relief of tension pneumothorax or cardiac tamponade) during the rapid assessment of children with undifferentiated shock. The bulk of evidence supporting the role of POCUS in the initial assessment of undifferentiated shock is for adult patients (see "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock", section on 'Initial diagnostic evaluation'). However, expertise in POCUS for children is increasing, providing at least anecdotal evidence that it may also be useful for children [7]. (See 'Ancillary studies' below.)

A more general discussion of the PAT is provided separately. (See "Initial assessment and stabilization of children with respiratory or circulatory compromise", section on 'Pediatric assessment triangle'.)

History — The following historical features may identify the condition causing shock:

- A history of fluid loss (due to gastroenteritis, diabetic ketoacidosis, or a gastrointestinal bleed) is consistent with hypovolemic shock. If available through review of medical records or caregiver report, a pre-illness weight, when compared with the weight on presentation, can provide an estimate of the amount of fluid loss. (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'History'.)
- Children who have been injured may have hypovolemic shock from hemorrhage (eg, solid organ injury from blunt abdominal trauma), obstructive shock (eg, tension pneumothorax or cardiac tamponade), and/or neurogenic shock (eg, spinal cord injury). (See "Trauma management: Approach to the unstable child", section on 'Circulation'.)
- Fever and/or immunocompromise (due to chemotherapy, sickle cell disease, or inherited immunodeficiencies) may indicate septic shock. (See "Children with early and life-threatening sepsis in resource-abundant settings: Rapid recognition and initial resuscitation (first hour)", section on 'Rapid recognition'.)
- A history of exposure to an allergen (eg, a bee sting or food) suggests anaphylactic shock.
- Shock may develop as the result of exposure to toxins (eg, iron, arsenic, beta blockers, calcium channel blockers, or cardiac glycosides).
- Patients with chronic heart disease (eg, cardiomyopathy or complex congenital heart disease) may develop cardiogenic shock.

- Adrenal crisis must be considered in a patient at risk for adrenal insufficiency (eg, patients receiving chronic steroid therapy, hypopituitarism, neonates with congenital adrenal disease, or sepsis) (table 5). (See "Clinical manifestations and diagnosis of adrenal insufficiency in children" and "Treatment of adrenal insufficiency in children".)

Physical examination — A complete physical examination, including vital signs with pulse oximetry, should be performed (table 6 and table 4).

An accurate weight is essential for determining fluid requirements and medication doses. The child's length provides a reasonable estimate of weight when a weight cannot be measured. (See "Initial assessment and stabilization of children with respiratory or circulatory compromise", section on 'Estimation of weight'.)

Abnormal vital signs provide essential information regarding the severity, classification, and cause of shock.

- **Respiratory rate** – Children with shock are usually tachypneic. Capnography may be helpful for identifying hyperventilation in response to metabolic acidosis [8]. A falsely low EtCO2 can also occur in the setting of poor pulmonary perfusion as a component of the shock state. (See "Carbon dioxide monitoring (capnography)".)

- **Heart rate** – In children, sinus tachycardia is a consistent sign of shock (except for patients with cardiogenic shock from a bradyarrhythmia or spinal cord injury). For patients with compensated shock, it may be the only abnormal vital sign. Other causes of tachycardia with poor perfusion in children include:

  - **Supraventricular tachycardia (SVT)** – With SVT, the heart rate is typically >220 beats per minute for infants, >180 beats per minute for older children and adolescents, and relentlessly regular (table 7 and waveform 1). Pediatric treatment is discussed in the algorithm and provided separately (algorithm 3). (See "Management of supraventricular tachycardia (SVT) in children".)

  - **Ventricular tachycardia (VT)** – VT is an uncommon rhythm in children that can be identified by electrocardiogram (waveform 2). Initial pediatric treatment is provided in the algorithm and discussed separately (algorithm 3). (See "Management and evaluation of wide QRS complex tachycardia in children", section on 'Stable patient'.)

- **Blood pressure** – Children with shock may have normal blood pressures. Hypotension must be rapidly identified, because those with low blood pressures typically deteriorate rapidly to cardiovascular collapse and cardiopulmonary arrest [3]. (See 'Circulation' above.)

Measurement with a manual cuff may be more accurate for children with circulatory compromise. Blood pressures determined with automated office devices can be higher than those using manual devices, particularly for hypotensive patients [9]. For children with normal systolic blood pressures, the classification of shock may be suggested by changes in the pulse pressure:

- Narrow pulse pressure (typically <30 mmHg in older children and adults) occurs when diastolic blood pressure is increased as the result of a compensatory increase in SVR (such as with hypovolemic and cardiogenic shock).
- Widening of pulse pressure (typically >40 mmHg in older children and adults; lower pulse pressures may reflect widening in infants and neonates) can be seen when diastolic blood pressure is decreased as the result of decreased SVR (as can occur with distributive shock).

- **Temperature** – Fever (or hypothermia in young infants) is often consistent with septic shock.

Additional features of the physical examination that suggest the etiology of shock include the following:

- **Stridor, wheezing, or abnormal breath sounds** – Children with stridor or wheezing may have anaphylaxis. Those with crackles may have a pneumonia (septic shock) or heart failure (cardiogenic shock). Those with asymmetric breath sounds may have a tension pneumothorax. Airway obstruction from other causes (such as foreign body aspiration or status asthmaticus) may lead to cardiovascular collapse from hypoxemia.
- **Distended neck veins** – Distended neck veins suggest an abnormality of cardiac contractility with heart failure, or obstruction to venous return caused by cardiac tamponade or tension pneumo- or hemothorax.
- **Abnormal heart sounds** – Cardiogenic shock is suggested by cardiac murmurs or a gallop rhythm. Muffled heart tones suggest pericardial fluid and, when accompanied by pulsus paradoxus, identify cardiac tamponade.
- **Pulse differential** – Decreased pulses and/or blood pressure in the lower extremities when compared to the upper extremities suggests coarctation of the aorta or other structural heart disease.
- **Hepatomegaly** – Hepatic congestion and resulting hepatomegaly can be seen with heart failure.
- **Abnormal abdominal findings** – Abdominal distention, masses, or tenderness is consistent with an abdominal catastrophe such as bowel obstruction, perforation, or

peritonitis. Inflicted injury should be considered in the absence of a plausible history of trauma.

- **Abnormal skin findings** – Urticaria or facial edema suggests anaphylaxis but is **not** a consistent finding in severe reactions. Purpura can be seen with septic shock. Bruises and/or abrasions may be noted with trauma.

Ancillary studies — Selected ancillary studies may be useful for successfully treating shock, identifying the etiology, and monitoring response to treatment. Ancillary studies should be simultaneously obtained with rapid assessment and treatment based upon the most likely etiology for shock as follows:

- **Hypovolemic shock without hemorrhage** (see "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'Ancillary data') – Suggested studies for patients with significant hypovolemic shock reflecting more than 10 percent dehydration include:

  - Rapid blood glucose
  - Serum electrolytes
  - Blood lactate
  - Urine dipstick
  - Chest radiograph for patients without rapid improvement after 60 mL/kg of isotonic fluid administration
  - Two view abdominal radiograph for patients with abdominal findings that suggest possible perforation or obstruction

  Serum lactate is a marker of tissue perfusion and has been used to measure the severity of shock in patients with sepsis. Lactate clearance has been used to monitor the response to therapy. Although not specifically studied in hypovolemic shock, blood lactate may also be an indicator of severity of illness and lactate clearance a measure of response to fluid therapy. The role of serum lactate and other studies for children with suspected septic shock are discussed in more detail separately. (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Laboratory studies' and "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Imaging'.)

  A urine dipstick reading provides a quick measure of specific gravity, ketones, and glucose. Glycosuria with ketonuria suggests diabetic ketoacidosis.

  A chest radiograph in children with suspected hypovolemic shock who do not rapidly improve after up to 60 mL/kg of isotonic fluid administration is also suggested. If the heart size is small, then additional bolus fluid administration is indicated. In contrast, if the heart is big, then fluid therapy should be moderated and additional types of shock

(eg, septic or cardiogenic shock) may be present and warrant specific therapy. (See "Children with early and life-threatening sepsis in resource-abundant settings: Rapid recognition and initial resuscitation (first hour)" and "Shock in children in resource-abundant settings: Initial management", section on 'Further management by type of shock'.)

For patients with abdominal findings and possible third spacing, plain radiography of the abdomen may identify signs of bowel obstruction or perforation. Additional studies are usually warranted to establish a definitive diagnosis (eg, ultrasound for intussusception or upper gastrointestinal series for malrotation). (See "Intussusception in children", section on 'Evaluation' and "Acute appendicitis in children: Diagnostic imaging" and "Intestinal malrotation in children", section on 'Diagnosis'.)

- **Hypovolemic shock with hemorrhage** – In addition to the above studies, patients with hemorrhagic hypovolemic shock warrant the following:
  - Hematocrit
  - Arterial or venous blood gas measurements
  - Type and cross match (patients with trauma and hemorrhagic shock)
  - Coagulation studies (platelet count, prothrombin time [PT] with international normalized ratio [INR], and activated partial thromboplastin time [PTT])
  - Chest radiograph
  - Bedside focused assessment with sonography for trauma (FAST)

  For patients with traumatic hemorrhagic shock, a chest radiograph may also identify intrathoracic bleeding (ie, hemothorax) or other obstructive causes for shock, such as tension pneumothorax or pericardial effusion. (See "Trauma management: Approach to the unstable child", section on 'Screening radiographs'.)

  Bedside extended focused assessment with sonography for trauma (e-FAST) can rapidly identify pericardial effusion and serious intraabdominal hemorrhage (movie 2). Additional imaging is typically warranted for these patients to better define sites of bleeding. The approach to imaging in the pediatric trauma patient is discussed separately. (See "Trauma management: Approach to the unstable child", section on 'Adjuncts to the primary survey' and "Trauma management: Approach to the unstable child", section on 'Adjuncts to the secondary survey'.)

- **Sepsis and septic shock** – Suggested laboratory studies for children with sepsis and septic shock are discussed in detail separately and include (see "Children with early and

life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Laboratory studies'):

- Rapid blood glucose
- Arterial or venous blood gas
- Complete blood count with differential
- Blood lactate
- Serum electrolytes
- Blood urea nitrogen and serum creatinine
- Ionized blood calcium
- Serum total bilirubin and alanine aminotransferase
- PT and INR
- PTT
- Fibrinogen and D-dimer
- Blood culture
- Urinalysis
- Urine culture
- Other cultures as indicated by clinical findings (eg, wound or mucosal site cultures for patients with suspected toxic shock syndrome (table 8))
- Diagnostic serologic testing as indicated to identify suspected sources of infection
- Inflammatory biomarkers (eg, C-reactive protein, procalcitonin) in selected cases

Other imaging may be appropriate depending upon clinical findings. For example, computed tomography of the head may be necessary in the patient with evidence of coagulopathy and altered mental status to evaluate for intracranial hemorrhage; ultrasound or computed tomography of the abdomen may be indicated to evaluate for intra-abdominal abscess. (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Imaging'.)

- **Cardiogenic shock** – Children with a history of congenital or acquired heart disease, abnormal cardiac examinations, cardiomegaly on chest radiograph, or who are not improving with initial treatment should receive 12-lead electrocardiograms (ECG) to evaluate for heart failure or signs of ischemia. In previously healthy children who present with cardiogenic shock, multisystem inflammatory syndrome in children (MIS-C) (table 9) and myocarditis are important etiologies to pursue. (See "COVID-19: Multisystem inflammatory syndrome in children (MIS-C) clinical features, evaluation, and diagnosis" and "Clinical manifestations and diagnosis of myocarditis in children".)

  Limited evidence suggests that POCUS performed by well-trained, experienced emergency medicine providers may identify life-threatening conditions (such as myocardial dysfunction or tamponade (movie 1)) and guide critical procedures [10-

13] (see "Emergency pericardiocentesis", section on 'Ultrasound-guided pericardiocentesis technique'). Depending upon specific findings, consultation with a pediatric cardiologist and formal echocardiography may also be warranted.

Cardiac arrhythmias may be suspected based upon heart rate and waveform during continuous monitoring. However, a 12-lead ECG is necessary to fully characterize the arrhythmia. (See "Clinical features and diagnosis of supraventricular tachycardia (SVT) in children", section on 'Diagnosis' and "Management and evaluation of wide QRS complex tachycardia in children".)

- **Obstructive shock** – Although chest radiograph or computed tomography (CT) of the chest can be diagnostic, whenever possible, tension pneumo- and hemothorax should be identified clinically or by emergency bedside ultrasonography (movie 3) and rapidly treated by chest tube thoracostomy. (See "Clinical presentation and diagnosis of pneumothorax", section on 'Diagnostic imaging' and "Thoracostomy tubes and catheters: Indications and tube selection in adults and children", section on 'Indications'.)

  Computed tomographic pulmonary angiography provides definitive diagnostic imaging for pulmonary embolism (PE) but should only be performed in patients who respond to the initial treatment of shock (oxygenation, intravenous fluid resuscitation, and vasopressor support). In patients who remain unstable despite adequate resuscitation and in whom the suspicion for pulmonary embolus is high, definitive testing is typically considered unsafe. Bedside transthoracic or transesophageal ultrasonography may be used in such patients to establish a presumptive diagnosis of PE and used to justify administration of thrombolytic therapy. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Hemodynamically unstable patients'.)

CLINICAL CLASSIFICATION OF SHOCK

Once the initial evaluation is completed, a constellation of clinical characteristics suggest a specific type of shock (algorithm 1). A patient may have more than one type of shock (such as an infant with cardiogenic shock from supraventricular tachycardia (SVT) who is also hypovolemic because he has been unable to drink, or a child with underlying cardiomyopathy who is septic).

Findings that may be helpful include:

- **Hypovolemic shock** – History of volume loss (including hemorrhage associated with trauma), narrow pulse pressure, signs of poor peripheral perfusion (decreased distal

pulses, cool extremities, prolonged capillary refill), small heart on chest radiograph (if one is obtained) (table 10). (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'Evaluation'.)

- **Distributive shock** – Vascular dilation typically causes a widened pulse pressure. Other features associated with specific types of distributive shock include:
  - **Septic shock** – History of fever or immunocompromise, variable peripheral perfusion (vasodilation with warm shock, vasoconstriction with cold shock), purpuric rash, abnormal white blood cell count, disseminated intravascular coagulation. (See "Children with early and life-threatening sepsis: Definitions, clinical manifestations, and diagnosis", section on 'Diagnosis'.)
  - **Anaphylactic shock** – History of exposure to an allergen (such as a bee sting or food), stridor, wheezing, wide pulse pressure, vasodilation, urticaria, facial edema. (See "Anaphylaxis: Acute diagnosis", section on 'Definition'.)
  - **Neurogenic shock** – History of trauma with severe head or cervical spine injury, hypotension with wide pulse pressure, normal heart rate or bradycardia.
- **Cardiogenic shock** – History of heart disease (eg, corrected congenital heart disease or cardiomyopathy), history of palpitations, signs of heart failure (eg, pulmonary rales, hepatomegaly, gallop rhythm, distended jugular veins), or arrhythmia.
- **Obstructive shock** – History of thoracic trauma, deviation of the trachea, and distended neck veins in a patient with tension pneumo- or hemo-thorax; muffled heart sounds and pulsus paradoxus in a patient with cardiac tamponade; abrupt circulatory collapse in a patient with pulmonary embolism caused by predisposition to thrombosis (eg sickle cell disease or congenital thrombophilia); or abrupt onset of new heart murmur, shock, and/or cyanosis in a neonate with a ductal dependent congenital heart lesion (eg, critical coarctation of the aorta or hypoplastic left heart syndrome) within the first few weeks of life due to closure of the ductus arteriosus. (See "Evaluation of suspected critical congenital heart disease (CHD) in the newborn".)

MANAGEMENT

Definitive management of shock depends upon correct classification of the type of shock present and definitive treatment of the underlying etiology. The initial management of undifferentiated shock in resource-abundant settings is provided in the algorithm and discussed in detail separately (algorithm 2). (See "Shock in children in resource-abundant settings: Initial management".)

The initial management of hypovolemic and septic shock as well as the management of shock in children in resource-limited settings are discussed elsewhere.

- (See "Hypovolemic shock in children in resource-abundant settings: Initial evaluation and management", section on 'Management'.)
- (See "Children with early and life-threatening sepsis in resource-abundant settings: Rapid recognition and initial resuscitation (first hour)", section on 'Resuscitation'.)
- (See "Shock in children in resource-limited settings: Initial management".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Shock in children".)

SUMMARY AND RECOMMENDATIONS

- **Pathophysiology** – Shock develops as the result of conditions that cause one or more of the following (table 3) (see 'Pathophysiology' above):
  - Decreased intravascular volume – Hypovolemic shock
  - Abnormal distribution of intravascular volume – Distributive shock
  - Impaired cardiovascular function – Cardiogenic shock
  - Obstruction to cardiac output – Obstructive shock

- **Evaluation** – The goals of the evaluation of shock are to identify life threatening conditions and to rapidly recognize children with circulatory compromise so that treatment can be initiated before hypotension develops. (See 'Evaluation' above.)

- **Rapid assessment** – Rapid assessment of appearance, breathing, and circulation should identify children with life-threatening conditions, including hypotensive shock, obstructive causes of shock (eg, tension pneumothorax, hemothorax, cardiac tamponade, or pulmonary embolism), and compensated shock. (See 'Rapid assessment' above.)

- **Clinical classification of shock** – Clinical features and ancillary studies often suggest the cause of shock and can be used to guide management decisions (algorithm 1). (See 'Clinical classification of shock' above and 'Ancillary studies' above.)

- **Initial management** – The initial management of undifferentiated shock occurs simultaneously with the rapid assessment and clinical classification of shock and an approach is provided in the algorithm and discussed in detail separately

(algorithm 2). Definitive management of pediatric shock depends upon correct classification of the type of shock based upon clinical findings and definitive treatment of the underlying etiology. (See "Shock in children in resource-abundant settings: Initial management" and "Shock in children in resource-limited settings: Initial management".)

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Topic 6396 Version 31.0

GRAPHICS

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

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

Barbiturates
Chemotherapeutic agents
Calcium channel blockers
Radiation

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

Data from:

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Graphic 67841 Version 7.0

Drug- and toxin-induced changes in blood pressure and pulse

Hypertension with tachycardia	Hypertension with bradycardia	Hypotension with tachycardia	Hypotension with bradycardia
Sympathomimetics	Alpha-adrenergic agonists	Beta-adrenergic agonists	Beta-blockers
Amphetamines	Phenylpropanolamine	Theophylline	Calcium-channel blockers
Cocaine	Phenylephrine	Albuterol	Cardiac glycosides
Ephedrine	Phentermine	Isoproterenol	Digoxin
Pseudoephedrine	Ergot alkaloids	Terbutaline	Digitalis purpurea
Theophylline	Sumatriptan	Caffeine	Oleander
Caffeine	Clonidine (early)	Disulfiram reaction (late)	Red squill
Methylphenidate	Guanfacine	Toxic alcohols	Bufotenin
Cathinones	Imidazolines	Isopropyl alcohol	Clonidine
Anticholinergics	Tetrahydrozoline	Carbon monoxide	Alpha-methyldopa
Antihistamines	Oxymetazoline	Alpha-adrenergic antagonists	Cyanide
TCAs (early)	Cholinergic agents	Phenothiazines	Carbon monoxide (late)
Phenothiazines (some)	Organophosphates	TCAs	Opioids
Antiparkinson agents	Carbamates	Hydralazine	Sedative-hypnotics
Muscle relaxants	Steroid hormones	Heavy metals (acute)	Barbiturates
Clozapine	Glucocorticoids	Iron	Benzodiazepines
Central hallucinogens	Mineralocorticoids	Arsenic	Cholinergics
Designer amphetamines	Estrogen	Colchicine	Organophosphates
Lysergic acid diethylamide (LSD)	Progesterone	Nitrates	Carbamates
Phencyclidine (PCP)	Androgens	Sodium nitroprusside	Antiarrhythmics
Synthetic cannabinoids	Yohimbine		
Envenomations	Heavy metals		
Black widow spider bite	Lead		
Scorpion stings	Disulfiram reaction (early)		
Drug withdrawal states			

MAOIs (foods with tyramine)
Nicotine
Cholinergic agents (sometimes)
Organophosphates
Carbamates
Thyroid hormone

Graphic 51200 Version 8.0

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 timeΔ**	**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.

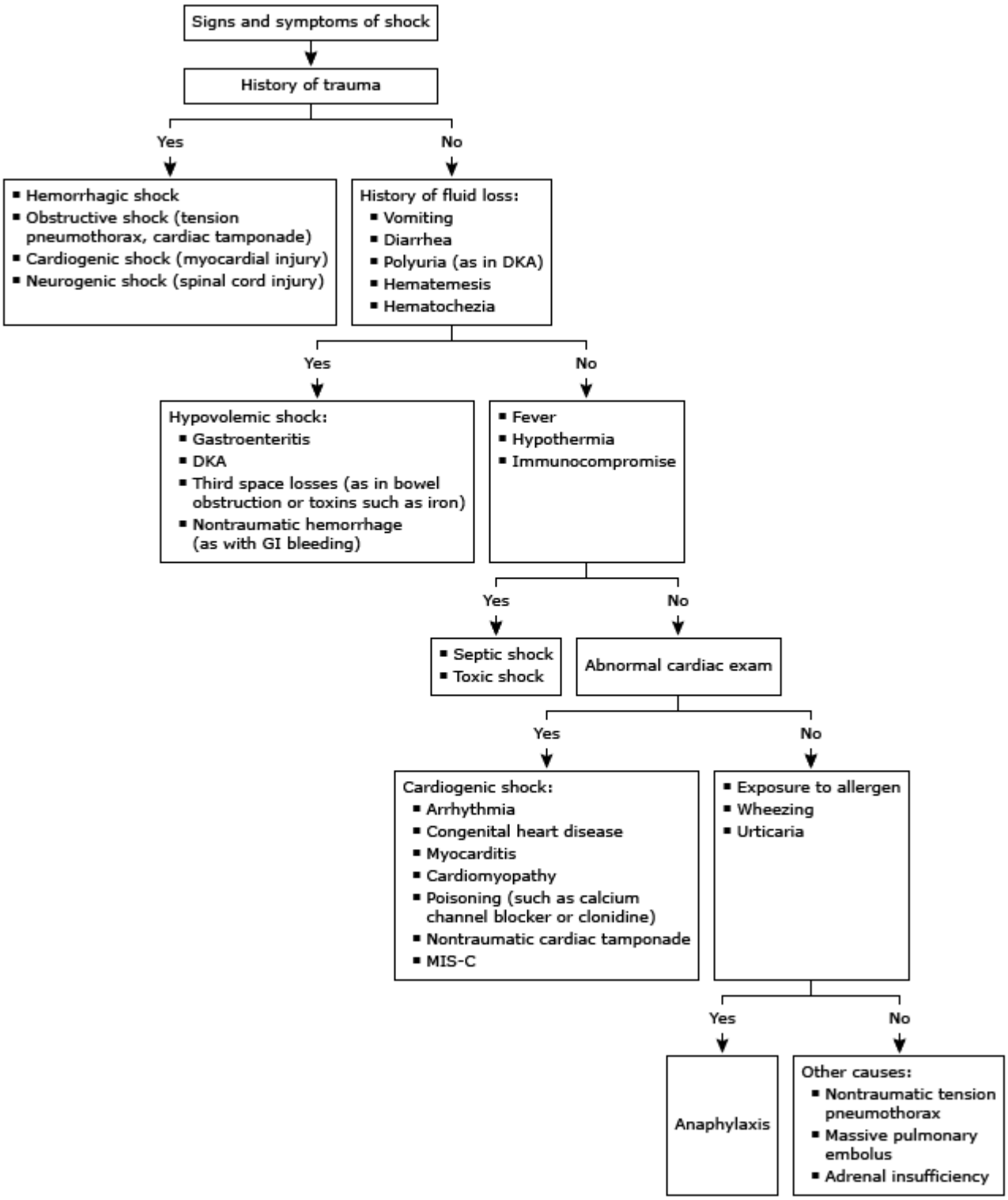
Δ In patients with shock, capillary refill time >2 seconds is associated with low mixed venous oxygen saturation while flash capillary refill suggests increased mixed venous oxygen saturation.

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

§ In patients with obstructive shock caused by tension pneumothorax, the patient typically displays clinical signs of increased preload. However, because blood return to the heart is obstructed by compression or occlusion of the superior and inferior vena cavae, physiologic preload is decreased.

Graphic 73000 Version 8.0

Approach to the classification of undifferentiated shock in children



DKA: diabetic ketoacidosis; GI: gastrointestinal.

Graphic 81356 Version 5.0

Initial shock management in children in settings with access to ICU care


Clinical signs of shock present (2 or more):
Diminished peripheral pulses
Cool, pale, or mottled skin
Altered mental status
Tachycardia or bradycardia
Provide supplemental oxygen, as needed, to avoid hypoxemia
Identify and treat life-threatening conditions:
Arrhythmia
Obstructive shock caused by tension pneumothorax, hemothorax, or cardiac tamponade
Anaphylaxis
Initiate continuous monitoring of HR, BP, and pulse oximetry
Treat respiratory failure with HFNC, NIV, or mechanical ventilation as needed*
Within 5 minutes, obtain venous access:
Preferred: two peripheral IVs of largest caliber that can be reliably inserted
If peripheral access not achieved: place IO cannula or central venous line
Obtain bedside glucose and laboratory studies (refer to inset)¶
If no signs of fluid overload, give IV or IO fluid resuscitation with balanced crystalloid solution (eg, Ringer's lactate) or normal saline as follows:Δ
Hypotensive shock: 20 mL/kg over 5 to 10 minutes
Compensated shock: 10 to 20 mL/kg over 5 to 20 minutes
Identify need for and give time-sensitive treatment based upon underlying condition:
Blood transfusion for hemorrhage
Empiric antimicrobial therapy for septic shock
Dextrose infusion for hypoglycemia
Prostaglandin E1 for ductal-dependent congenital heart disease
Systemic corticosteroids for adrenal insufficiency
Treatment of electrolyte abnormalities (eg, hyperkalemia, hypokalemia, or hypocalcemia)
Evaluate clinical targets; are all of the following present?
BP >5th percentile
Strong central and distal pulses
Warm skin with capillary refill <2 seconds
Alert mental status
Urine output 1 mL/kg or greater after circulating volume is restored
Yes
No
Continue monitoring
Treat underlying condition
Admit to appropriate inpatient setting
Signs of fluid overload (one or more of the following):
Pulmonary rales
Worsening respiratory distress with new or worsening oxygen requirement
Gallop rhythm
Jugular venous distension
Hepatomegaly
Cardiomegaly or pulmonary edema on bedside US or chest radiograph
Yes
No
Probable cardiogenic shock:
Evaluate cardiac function (bedside US, ECHO◇, and pediatric cardiology consultation)
If indicated, give smaller fluid boluses over a longer period of time (eg, 5 to 10 mL/kg over 15 to 30 minutes)§
Start vasoactive therapy¥
For persistent signs of shock, repeat 10 to 20 mL/kg f
up to 60 mL/kg within the first hour
Evaluate clinical targets after each bolus
Signs of shock persist
Re-evaluate presumed cause of shock
For suspected hemorrhagic shock, evaluate for source of bleeding and give blood products
For suspected hypovolemic shock, re-estimate fluid losses and continue fluid replacement
For patients with suspected distributive shock, start vasoactive agents¥
Obtain consultation with a pediatric critical care specialist and arrange transfer to a pediatric ICU
Inset: Initial laboratory stud
All patients:
Rapid blood glucose
Complete blood count with d
Serum electrolytes
Venous or arterial blood gas
Blood lactate
Blood urea nitrogen and seru
Urine dipstick
Septic shock suspected:
Serum total bilirubin
Alanine aminotransferase
Ionized blood calcium
PT and INR
PTT
Fibrinogen and D-dimer
Blood culture
Other cultures as indicated b
Diagnostic serologic testing a
Inflammatory biomarkers (eg
Urinalysis
Urine culture
Hemorrhagic shock suspected:
Type and cross-match
PT and INR
PTT
Additional studies based upo
(refer to UpToDate content o
Other studies:
ECG (arrhythmia or signs of
Bedside US (suspected obstr
Pediatric cardiology specialis
Chest radiograph for patients
60 mL/kg of isotonic fluid ad
Two-view abdominal radiogra
suggest possible perforation

ICU: intensive care unit; HR: heart rate; BP: blood pressure; HFNC: high-flow oxygen by nasal cannula; NIV: noninvasive ventilation; IV: intravenous; IO: intraosseous; US: ultrasound; ECHO: echocardiography; PT: prothrombin time; INR: international normalized ratio; PTT partial thromboplastin time; ECG: electrocardiography; e-FAST: extended focused assessment with sonography for trauma.

* A trial of HFNC or NIV, such as continuous positive airway pressure ventilation or bi-level positive airway pressure ventilation, may avoid the need for endotracheal intubation in selected patients. Patients with hemodynamic instability should receive appropriate interventions to treat shock prior to or during intubation. Refer to UpToDate content on HFNC, NIV, and rapid sequence intubation in children.

¶ Ancillary studies are determined by patient presentation and suspected type or types of shock present. Other laboratory and ancillary studies may also be indicated based upon the suspected underlying condition that is causing shock.

Δ Fluid volume should be calculated based upon ideal body weight (eg, 50^{th} percentile for age).

◇ When performed by trained and experienced physicians, bedside ECHO can provide rapid evidence of myocardial dysfunction, including dysfunction due to obstructive shock.

§ Patients with signs of fluid overload who continue to receive fluid boluses warrant close monitoring for respiratory and cardiac failure. The clinician should have a low threshold for endotracheal intubation and mechanical ventilation to treat pulmonary edema in these patients.

¥ Suggested vasoactive therapy depends upon type of shock and clinical findings; refer to UpToDate topics and graphics on management of shock in children.

Graphic 129655 Version 1.0

Pediatric respiratory rate and heart rate lower limit, normal range, and upper limit by age*

Age	Respiratory rate (breaths/minute)			Heart rate (beats/minute)		
	Lower limit (1st percentile)	Normal range (10th to 90th percentile)	Upper limit (99th percentile)	Lower limit (1st percentile)	Normal range (10th to 90th percentile)	Upper limit (99th percentile
0 to 3 months	25	34 to 57	66	107	123 to 164	181
3 to <6 months	24	33 to 55	64	104	120 to 159	175
6 to <9 months	23	31 to 52	61	98	114 to 152	168
9 to <12 months	22	30 to 50	58	93	109 to 145	161
12 to <18 months	21	28 to 46	53	88	103 to 140	156
18 to <24 months	19	25 to 40	46	82	98 to 135	149
2 to <3 years	18	22 to 34	38	76	92 to 128	142
3 to <4 years	17	21 to 29	33	70	86 to 123	136
4 to <6 years	17	20 to 27	29	65	81 to 117	131
6 to <8 years	16	18 to 24	27	59	74 to 111	123
8 to <12 years	14	16 to 22	25	52	67 to 103	115
12 to <15 years	12	15 to 21	23	47	62 to 96	108
15 to 18 years	11	13 to 19	22	43	58 to 92	104

* The respiratory and heart rates provided are based upon measurements in awake, healthy infants and children at rest. Many clinical findings besides the actual vital sign measurement must be taken into account when determining whether a specific vital sign is normal in an individual patient. Values

for heart rate or respiratory rate that fall within normal limits for age may still represent abnormal findings that are caused by underlying disease in a particular infant or child.

Data from: Fleming S, Thompson M, Stevens R, et al. Normal ranges of heart rate and respiratory rate in children from birth to 18 years of age: A systematic review of observational studies. Lancet 2011; 377:1011.

Graphic 78097 Version 11.0

Rapid overview: Adrenal crisis in children and adolescents

Signs and symptoms that may indicate adrenal crisis:

- Vomiting and diarrhea, sometimes with severe abdominal pain or unexplained fever, weight loss and anorexia.
- Serum electrolyte abnormalities:
  - Hyponatremia with or without hyperkalemia.
  - Metabolic acidosis.
  - Hypoglycemia (especially in young children).
- Hypotension or shock, particularly if disproportionate to apparent underlying illness.

Consider the diagnosis in:

- **Neonates with atypical genitalia and/or electrolyte abnormalities, lethargy, hypoglycemia, hypotension or shock, or failure to thrive**. These may be presenting features of CAH due to 21-hydroxylase deficiency* or (very rarely) other causes of adrenal insufficiency.
  - In the United States, 21-hydroxylase deficiency is part of the newborn screen in all states, so most affected infants will be diagnosed prior to presentation with adrenal crisis. Adrenal crisis usually presents between 10 and 20 days of life. Affected females usually have atypical genitalia (varying severity). Males usually do not have genital abnormalities.
  - The presentation of adrenal crisis in an infant may mimic that of pyloric stenosis. However, infants with pyloric stenosis typically have hypokalemic alkalosis rather than the hyperkalemic acidosis that is typical of adrenal crisis.
- **Any patient with known disorders of adrenal insufficiency** (eg, CAH), especially if exposed to stress (illness).
- **Patients on chronic treatment with corticosteroids**, especially if exposed to stress or during a tapering of the corticosteroid dose.
- **Patients with other autoimmune endocrine deficiencies**, such as type 1 diabetes mellitus, hypothyroidism, or gonadal failure.
- **Critically ill patients with septic shock**, who are unresponsive to fluid resuscitation and inotropic medications (in this case, adrenal crisis can be caused by bilateral adrenal hemorrhage)
- Other patients presenting with the above signs, especially those with **hyperpigmentation or vitiligo**.

Evaluation:

- If adrenal crisis is suspected, then patients should be treated empirically with stress doses of glucocorticoids, as outlined below.
- For patients without a preexisting diagnosis of adrenal insufficiency, draw baseline blood samples prior to the administration of glucocorticoids for subsequent testing for electrolytes,

glucose, cortisol and other adrenal steroids (17-hydroxyprogesterone if CAH is suspected)*, ACTH, and plasma renin activity. Treatment with glucocorticoids can be initiated presumptively once blood samples are drawn. If the blood samples cannot be rapidly obtained and the patient is acutely ill, proceed immediately to treatment.

Treatment:

- **Shock** – Give a bolus of normal saline (0.9%), 20 mL/kg IV over 1 hour. If shock is persistent, repeat up to a total of 60 mL/kg within 2 hours.
- **Hypoglycemia** – Give an initial bolus of 0.5 to 1 g/kg of dextrose IV (maximum single dose 25 g). The glucose bolus is infused slowly, at 2 to 3 mL per minute.
  - For all age groups, this can be given as 25% dextrose solution (D25W), 2 to 4 mL/kg. (D25W is 250 mg dextrose/mL.)
  - An alternative for infants and children up to 12 years of age is to use 10% dextrose solution (D10W), 5 to 10 mL/kg. (D10W is 100 mg dextrose/mL.)
- **Stress glucocorticoids** – Administer hydrocortisone (Solu-Cortef) as a bolus, using a dose of 50 to 100 mg/m^2 IV.[¶ Δ] If body surface area is not available, age-based dosing may be used as follows:
  - Infants and toddlers 0 to 3 years old – 25 mg IV.
  - Children 3 to 12 years – 50 mg IV.
  - Children and adolescents 12 years and older – 100 mg IV.
- Continue glucocorticoids at the same dose given as a constant rate or as 4 divided doses over the following 24 hours. The subsequent dose and duration depend on the patient's clinical status.
- **Electrolytes** – If hyperkalemia is present, perform EKG to evaluate:
  - EKG changes consistent with hyperkalemia – Initially a tall, peaked T wave with shortened QT interval, followed by progressive lengthening of the PR interval and QRS duration.
  - Hyperkalemia typically improves promptly with fluids and hydrocortisone therapy alone. Rarely, severe and symptomatic hyperkalemia requires emergency therapy (ie, IV calcium infusion followed by IV insulin and glucose infusion; refer to UpToDate topic on the management of hyperkalemia in children).
  - Monitor and treat other electrolyte abnormalities and fluid balance.

CAH: congenital adrenal hyperplasia; ACTH: adrenocorticotropic hormone (corticotropin); IV: intravenous; EKG: electrocardiogram; BSA: body surface area; IM: intramuscularly.

* Refer to UpToDate content on classic CAH due to 21-hydroxylase deficiency.

¶ Dosing based on BSA is preferred if the patient's BSA is known or if the BSA can be promptly calculated based on measured height and weight. A BSA calculator is available in the UpToDate program.

Δ Hydrocortisone also may be given IM if IV access is not readily available.

Graphic 61357 Version 28.0

Normal blood pressure in children: 50th to 90th percentiles

Age	Systolic pressure, mm Hg	Diastolic pressure, mm Hg
Birth, 12 hours, <1000 g	39-59	16-36
Birth, 12 hours, 3 kg	50-70	25-45
Neonate, 96 hours	60-90	20-60
Infant, 6 months	87-105	53-66
Toddler, 2 years	95-105	53-66
School age, 7 years	97-112	57-71
Adolescent, 15 years	112-128	66-88

The median (50th percentile) systolic blood pressure for children older than 1 year may be approximated by the following formula: 90 mm Hg + (2 x age in years). The lower limit (5th percentile) of systolic blood pressure can be estimated with this formula: 70mm Hg + (2 x age in years).

A low systolic blood pressure should prompt an immediate evaluation for additional signs of inadequate perfusion, such as diminished mental status, prolonged capillary refill, and tachycardia.

Data from: Vershold, H. Aortic blood pressure during the first 12 hours of life in infants with birth weight 610-4220 grms. Pediatrics 1981; 67:107 and Report of the second Task Force on Blood Pressure Control in Children--1987. Task Force on Blood Pressure Control in Children. National Heart, Lung, and Blood Institute, Bethesda, Maryland. Pediatrics 1987; 79:1.

Graphic 51186 Version 3.0

Rapid overview: Emergency management of supraventricular tachycardia in children

Evaluation

Provide oxygen and ventilation immediately as needed.

Goals – Identify the unstable patient; distinguish SVT from sinus tachycardia.

Clinical assessment

- Potential causes of sinus tachycardia (dehydration, fever, pain, drugs).
- Signs of poor perfusion (poor capillary refill, hypotension, pallor, diminished mental status).
- Signs of heart failure (increased work of breathing, rales, hepatomegaly).

ECG*

- The following are consistent with SVT:
  - Rate – Infants 220 to 280 bpm; children and adolescents 180 to 240 bpm.
  - Relentlessly regular rhythm without variation with respiration or activity.
  - Abnormal P waves (absent or negative in II, III, and aVF).
  - For wide QRS complex, assume the origin is ventricular (although in children, most are SVT with aberrant conduction)¶.

Management

Goal – Terminate the abnormal rhythm. Consult pediatric cardiology early.

Stable	Unstable
▪ Children with SVT who are stable are unlikely to deteriorate suddenly. ▪ Begin treatment with vagal maneuvers and/or adenosine and consult pediatric cardiology.	▪ For unstable patients (depressed consciousness, poor perfusion, hypotension or other signs of shock or severe heart failure), begin treatment to convert to sinus rhythm immediately. ▪ Continue oxygen and ventilation as needed.
▪ **Vagal maneuvers** – For infants, apply bag containing ice water to the face above the nose and mouth for 15 to 30 seconds. Do not obstruct ventilation. In older children, bearing down or blowing into an occluded straw for 15 to 20 seconds provides vagal stimulation. Do **not** use carotid massage or orbital pressure.	▪ **Vagal maneuvers** – Do not delay treatment to administer vagal maneuvers in unstable patients. Attempt while preparing for cardioversion or drug therapy.
▪ **Adenosine** – With continuous ECG monitoring, administer rapidly through IV closest to the central circulation. Initial dose: 0.1 mg/kg (maximum 6 mg); if no response in 2 minutes, repeat dose 0.2 mg/kg	▪ **Without IV/IO in place:** • **Synchronized cardioversion** – Cardiovert immediately all unstable patients without IV access. Use 0.5 to 1 J/kg. If not effective, increase to 2 J/kg.

(maximum 12 mg). Follow each dose immediately with a saline flush of 5 mL[Δ].	▪ **With IV/IO in place[◇]:** • **Adenosine** – If immediately available, adenosine may be given to unstable patients with narrow complex SVT with I access while preparing to cardiovert. Initial dose is 0.1 mg/kg (maximum 6 mg); if no response in 2 minutes, repeat dose 0.2 mg/kg (maximum 12 mg). Follow each dose immediately with a saline flush of 5 mL[Δ]. • **Synchronized cardioversion** – If adenosine is not immediately available o if there is no response to adenosine, synchronized cardioversion should be performed in all unstable patients with I access. Use 0.5 to 1 J/kg. If not effective, increase to 2 J/kg. • **IV antiarrhythmic options for refractory SVT** – Alternative second-line agents that have been used in this setting include IV amiodarone, IV esmolol, IV procainamide, IV sotalol, and IV verapamil (in patients ≥1 year old). Choice of a second-line agent should be guided by expert consultation, given potential proarrhythmic and life-threatening hemodynamic collapse whe administering multiple antiarrhythmic agents.[§]

SVT: supraventricular tachycardia; ECG: electrocardiogram; bpm: beats per minute; IV: intravenous; IO: intraosseous; J/kg: joule per kilogram.

* A 15-lead ECG is preferred. This includes the 12 standard leads plus leads V3R and V4R (right-sided leads analogous to V3 and V4 on the left) and V7 (left posterior axillary line at V4 level). If a 15-lead ECG is not available, a standard 12-lead ECG is acceptable. ECG monitoring should continue during therapeutic maneuvers.

¶ Refer to separate UpToDate content for details of evaluation and management of wide QRS complex tachyarrhythmias in children.

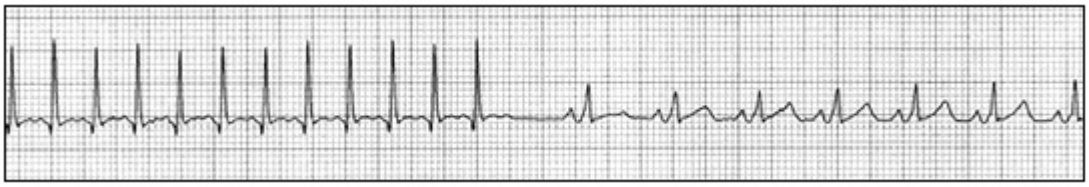
Δ The use of 2 syringes (1 with adenosine and the other with normal saline flush) connected to a stopcock is a useful way of ensuring rapid and effective drug delivery.

◇ IV access is preferred over IO for administration of adenosine and antiarrhythmic drugs. An IO can be used for these agents, but conversion to sinus rhythm with adenosine may not be successful when using IO access.

§ Consultation with a pediatric cardiologist is advised. Refer to UpToDate content on management of tachyarrhythmias in children for further details.

Graphic 53559 Version 17.0

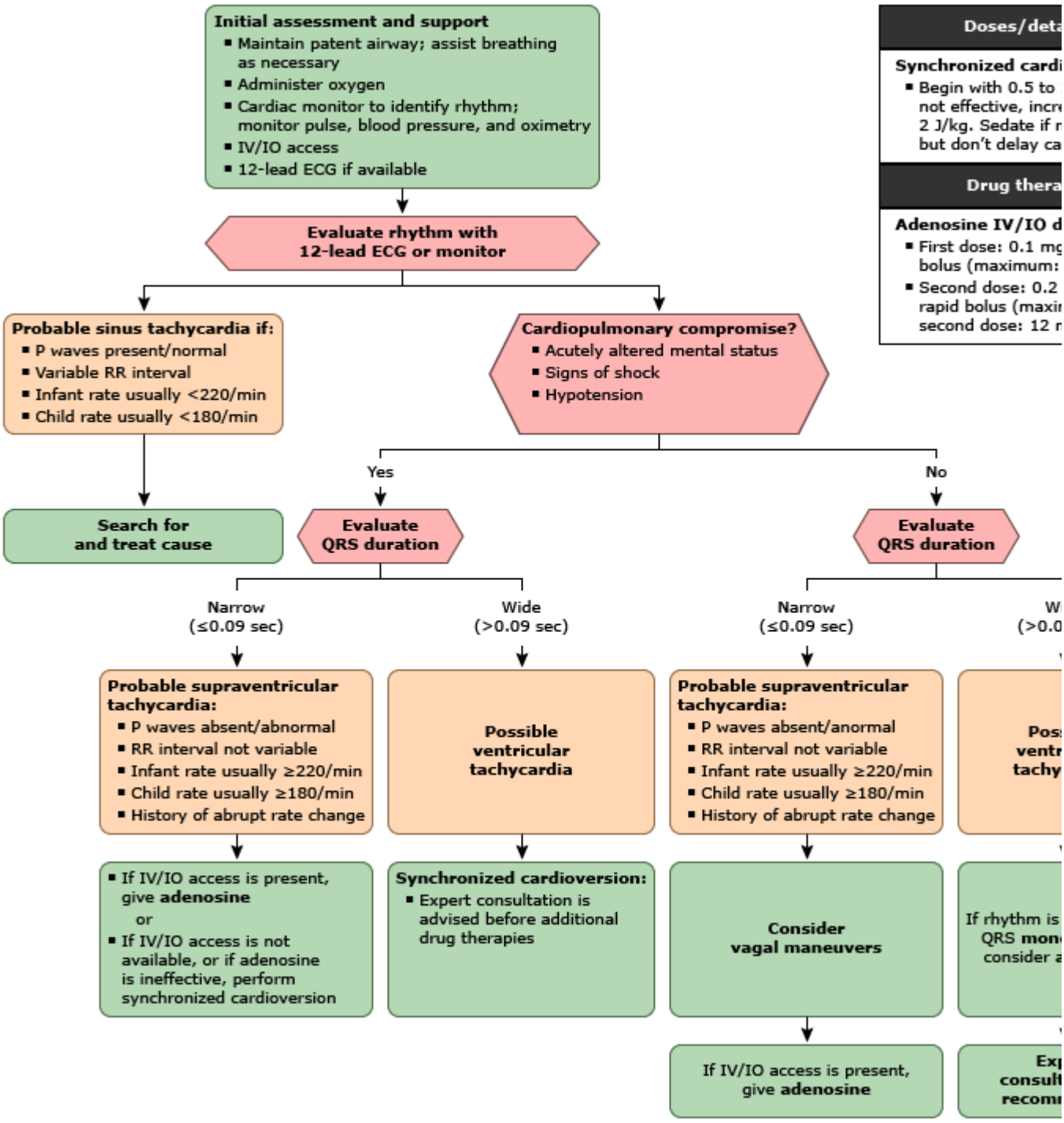
Conversion of supraventricular tachycardia with adenosine administration



Electrocardiogram during conversion of supraventricular tachycardia to sinus rhythm with administration of adenosine. During tachycardia at a rate of 230 beats/min, there is a normal-appearing QRS complex without a delta wave (no ventricular preexcitation), and there is no distinct P wave. After conversion to sinus rhythm, there is a short PR interval (80 milliseconds) and wide up-sloping QRS complex (90 milliseconds) representing ventricular preexcitation, indicative of the Wolff-Parkinson-White syndrome.

Graphic 70842 Version 2.0

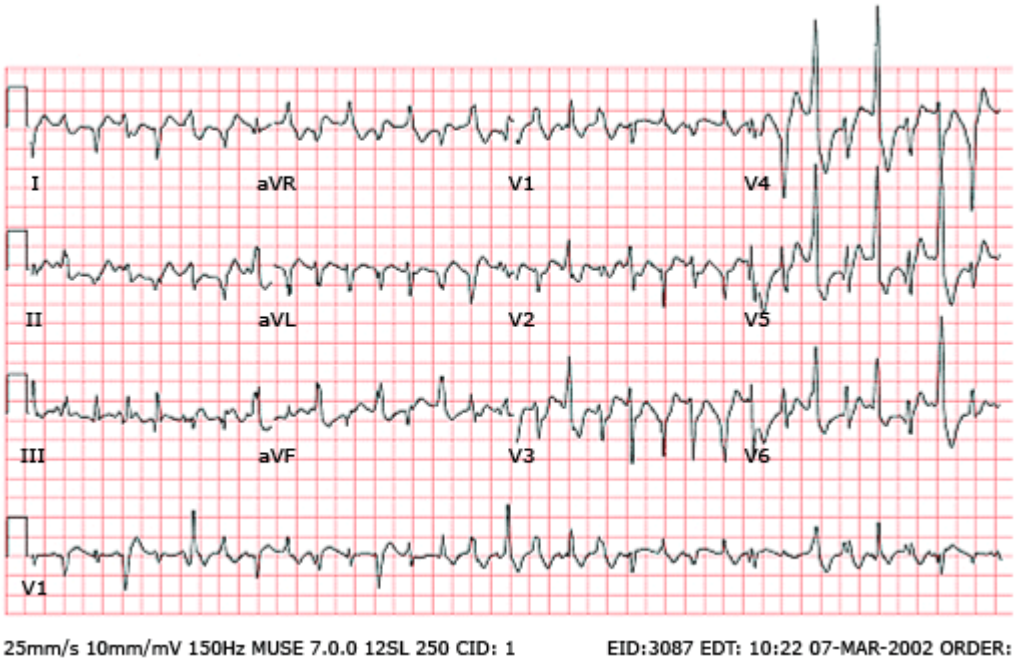
Pediatric tachycardia with a pulse 2020 update



IV: intravenous; IO: intraosseous; ECG: electrocardiogram; J/kg: joules per kilogram.

Graphic 129940 Version 10.0

12-lead electrocardiogram in a child with catecholaminergic polymorphic ventricular tachycardia



This is the electrocardiogram of a 5-year-old child with recurrent exertional syncope. The electrocardiogram demonstrates bidirectional ventricular tachycardia. This child underwent genetic testing and was positive for the RyR2 mutation.

Graphic 76976 Version 11.0

Clinical criteria for staphylococcal toxic shock syndrome (issued by the United States Centers for Disease Control and Prevention)

Clinical criteria
Fever – Temperature ≥38.9°C (102.0°F)
Rash – Diffuse macular erythroderma
Desquamation – 1 to 2 weeks after onset of rash
Hypotension: ▪ **For adults** – Systolic blood pressure ≤90 mmHg ▪ **For children <16 years of age** – Systolic blood pressure less than 5th percentile by age
Multisystem involvement (3 or more of the following organ systems)**:** ▪ **Gastrointestinal** – Vomiting or diarrhea at onset of illness ▪ **Muscular** – Severe myalgia or creatine phosphokinase elevation >2 times the upper limit of normal ▪ **Mucous membranes** – Vaginal, oropharyngeal, or conjunctival hyperemia ▪ **Renal** – Blood urea nitrogen or serum creatinine >2 times the upper limit of normal or pyuria (>5 leukocytes/high-power field) in the absence of urinary tract infection ▪ **Hepatic** – Bilirubin or transaminases >2 times the upper limit of normal ▪ **Hematologic** – Platelets <100,000/microL ▪ **Central nervous system** – Disorientation or alterations in consciousness without focal neurologic signs when fever and hypotension are absent
Laboratory criteria
Cultures (blood or cerebrospinal fluid) negative for alternative pathogens (blood cultures may be positive for *Staphylococcus aureus*)
Serologic tests negative (if obtained) for Rocky Mountain spotted fever, leptospirosis, or measles
Case classification
Probable case – A case which meets the laboratory criteria and 4 of the 5 clinical criteria
Confirmed case – A case which meets the laboratory criteria and all 5 of the clinical criteria, including desquamation (unless the patient dies before desquamation occurs)

The above criteria were established for epidemiologic surveillance; they should be not be used to exclude a case that is highly suspicious for toxic shock syndrome, even if all criteria are not met.

Adapted from: Centers for Disease Control and Prevention. Toxic shock syndrome (other than streptococcal) (TSS): 2011 case definition. Available at: https://ndc.services.cdc.gov/case-definitions/toxic-shock-syndrome-2011/ (Accessed on February 11, 2022).

Graphic 54778 Version 11.0

Clinical manifestations of COVID-19-associated multisystem inflammatory syndrome in children and adolescents

	Frequency* (%)
Presenting symptoms	
▪ Persistent fevers (median duration 4 to 6 days)	100
▪ Gastrointestinal symptoms (abdominal pain, vomiting, diarrhea)	60 to 100
▪ Rash	45 to 76
▪ Conjunctivitis	30 to 81
▪ Mucous membrane involvement	27 to 76
▪ Neurocognitive symptoms (headache, lethargy, confusion)	29 to 58
▪ Respiratory symptoms (tachypnea, labored breathing)	21 to 65
▪ Sore throat	10 to 16
▪ Myalgias	8 to 17
▪ Swollen hands/feet	9 to 16
▪ Lymphadenopathy	6 to 16
Clinical findings	
▪ Shock	32 to 76
▪ Criteria for complete Kawasaki disease met	22 to 64
▪ Myocardial dysfunction (by echocardiogram or elevated troponin/BNP)	51 to 90
▪ Arrhythmia	12
▪ Acute respiratory failure requiring noninvasive or invasive ventilation	28 to 52
▪ Acute kidney injury	8 to 52
▪ Serositis (small pleural, pericardial, and ascitic effusions)	24 to 57
▪ Hepatitis or hepatomegaly	5 to 21
▪ Encephalopathy, seizures, coma, or meningoencephalitis	6 to 7
Laboratory findings	
▪ Abnormal blood cell counts	
• Lymphocytopenia	80 to 95

• Neutrophilia	68 to 90
• Mild anemia	70
• Thrombocytopenia	31 to 80
▪ Elevated inflammatory markers	
• C-reactive protein	90 to 100
• Erythrocyte sedimentation rate	75 to 80
• D-dimer	67 to 100
• Fibrinogen	80 to 100
• Ferritin	55 to 76
• Procalcitonin	80 to 95
• Interleukin 6	80 to 100
▪ Elevated cardiac markers	
• Troponin	50 to 90
• BNP or NT-pro-BNP	73 to 90
▪ Hypoalbuminemia	48 to 95
▪ Mildly elevated liver enzymes	62 to 70
▪ Elevated lactate dehydrogenase	10 to 60
▪ Hypertriglyceridemia	70
Imaging findings	
▪ Echocardiogram	
• Depressed LV function	31 to 58
• Coronary artery dilation/aneurysm	8 to 38
• Other findings can include mitral regurgitation and pericardial effusion	--
▪ Chest radiograph	
• Normal in many patients	--
• Abnormal findings included small pleural effusions, patchy consolidations, focal consolidation, and atelectasis	--
▪ Chest CT	
• Findings generally similar to those on chest radiograph	--

• A few patients had nodular ground-glass opacification	--
▪ Abdominal imaging (ultrasound and/or CT)	
• Findings are nonspecific, including free fluid, ascites, bowel and mesenteric inflammation, including terminal ileitis, mesenteric adenopathy/adenitis, and pericholecystic edema	--

COVID-19: coronavirus disease 2019; BNP: brain natriuretic peptide; NT-pro-BNP: N-terminal pro-BNP; LV: left ventricular; CT: computed tomography.

* The frequencies listed in this table represent the proportion of patients with each finding among those tested or assessed for the finding. Not all patients were tested or assessed for each.

Graphic 128294 Version 8.0

Physical findings of volume depletion in infants and children

Finding	Mild (3 to 5%)	Moderate (6 to 9%)	Severe (≥10%)
Pulse	Full, normal rate	Rapid*	Rapid* and weak **or** absent
Systolic pressure	Normal	Normal to low	Low
Respirations	Normal	Deep, rate may be increased	Deep, tachypnea **or** decreased to absent
Buccal mucosa	Tacky or slightly dry	Dry	Parched
Anterior fontanelle	Normal	Sunken	Markedly sunken
Eyes	Normal	Sunken	Markedly sunken
Tears (in infants)	Present	Decreased	Absent
Skin turgor	Normal	Reduced	Tenting
Skin temperature and appearance	Normal	Cool	Cool, mottled, acrocyanosis
Urine output	Normal or mildly reduced	Markedly reduced	Anuria
Systemic signs	Increased thirst	Listlessness, irritability	Grunting, lethargy, coma

* Tachycardia may be the first sign of hypovolemic shock in infants.

Graphic 76198 Version 10.0

Contributor Disclosures

Mark Waltzman, MD No relevant financial relationship(s) with ineligible companies to disclose. **Susan B Torrey, MD** No relevant financial relationship(s) with ineligible companies to disclose. **James F Wiley, II, MD, MPH** No relevant financial relationship(s) with ineligible companies to disclose.

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