Shock

Nelson textbook of pediatrics ۲۰۲۴

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- acute, life-threatening process of cardiovascular insufficiency characterized by inadequate delivery of oxygen and nutrients(e.g., glucose) by the circulatory system to meet the metabolic demands of vital organs that results in cellular dysfunction and injury.
- imbalance between oxygen/nutrient delivery through the circulation and oxygen/nutrient consumption within organ systems can be compensated for by increasing tissue oxygen extraction.
- Further limitations in delivery of oxygen and nutrients as the shock state progresses, energy production gradually shifts from aerobic metabolism to less efficient anaerobic metabolic pathways
- If this process is not corrected, numerous adverse vascular, inflammatory, metabolic, cellular, and endocrine responses contribute to a state of **multiple organ dysfunction syndrome (MODS)**, cardiovascular collapse, and death

TYPES OF SHOCK

SHOCK	PATHOPHYSIOLOGY	APPEARANCE	EXAMPLES	TREATMENTS
Hypovolemic*	Decreased cardiac output due to volume depletion → decreased preload	Tachycardia and vasoconstriction maintain adequate circulation up to 30% of circulating volume Narrowed pulse pressure, delayed capillary refill, orthostatic changes, late hypotension, AMS, and decreased UOP	Nonhemorrhagic (vomiting and diarrhea) Hemorrhagic (trauma)	IV fluid bolus Vasopressors Blood replacement
Cardiogenic	Decreased cardiac output due to myocardial dysfunction, increased afterload, and/or lack of ventricular filling	Tachycardia, vasoconstriction, cool extremities, narrow pulse pressure, delayed capillary refill, respiratory distress, rales or gallop rhythm, enlarged liver, JVD, cardiomegaly on chest radiograph Significant gradient between upper and lower extremity blood pressures	Cardiomyopathies, infectious myocarditis, and systemic inflammatory process, autoimmune disease, impaired coronary perfusion, cardiopulmonary bypass, acidosis, HIE, and dysrhythmias Infants: ductal-dependent lesions, tachydysrhythmias	Judicious IV fluid resuscitation, ionotropic agents to improve contractility, vasodilators to reduce afterload, and management of tachyarrhythmias PGE for infants <2 mo
Distributive	Decreased cardiac output and systemic vascular resistance due to peripheral vasodilation → decreased afterload and preload, redistribution of blood flow away from vital organs, and loss of sympathetic outflow	Tachycardic, vasodilated, flushed, warm extremities, wide pulse pressure, bounding pulses, flash capillary refill Anaphylaxis: rash; facial swelling; lip, tongue, or airway swelling; bronchospasm; hypotension Spinal shock: unable to raise HR, hypotension Neurogenic: Control ↑ ICP	Septic shock Toxic ingestions Anaphylaxis Spinal cord injuries	IV fluids, vasopressors, antibiotics Specific antidotes Remove triggers, IV fluids, IM epinephrine, antihistamines, vasopressors IV fluids, vasoconstrictors
Obstructive	Decreased cardiac output due to increased afterload of the right ventricle from an obstructive process	Tachycardia, delayed capillary refill, cool extremities, narrow pulse pressure, distended neck veins, distant heart tones, asymmetric breath sounds	Tension pneumothorax Pulmonary embolism Cardiac tamponade	Anticoagulants Drain pericardial effusion Evacuate pneumothorax
Dissociative	High cardiac output failure due to inadequate oxygen-releasing capacity	Tachycardia ± delayed capillary refill, cool extremities, weakened pulse, pallor or	Anemia Carbon monoxide Methemoglobinemia	Gradual fluid replacement and blood replacement 5 mL/dL Oxygen, hyperbaric chamber

- **Septic shock** is generally a unique combination of distributive, hypovolemic, and cardiogenic shock, as well as dissociative shock.

Hypovolemia from insufficient fluid intake, GI losses, and/or vascular leak

Cardiogenic shock results from the myocardial-depressant effects of sepsis,

distributive shock is the result of decreased SVR.

- Degree to which a patient exhibits each of these responses varies, but there are frequently alterations in preload, afterload, and myocardial contractility.

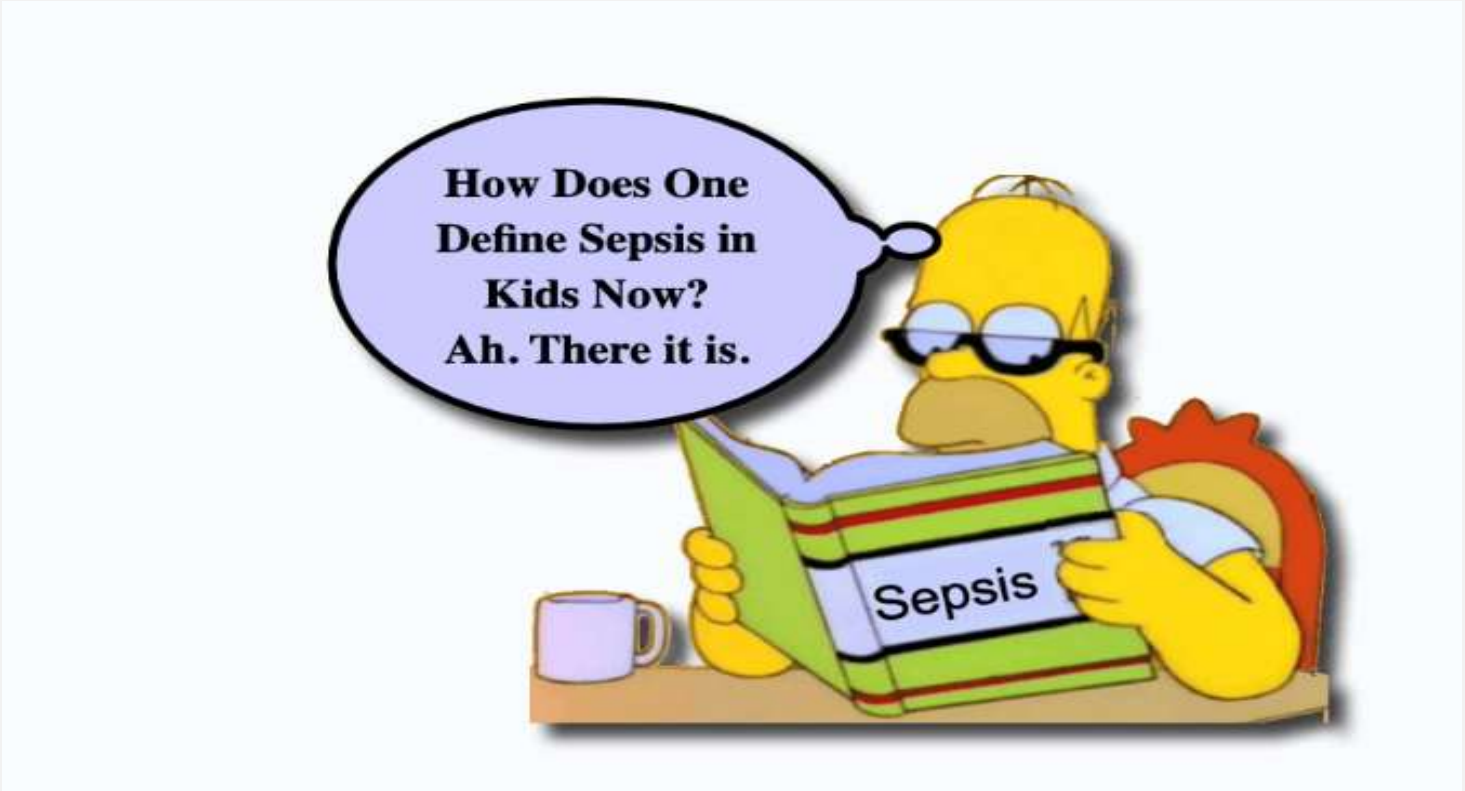
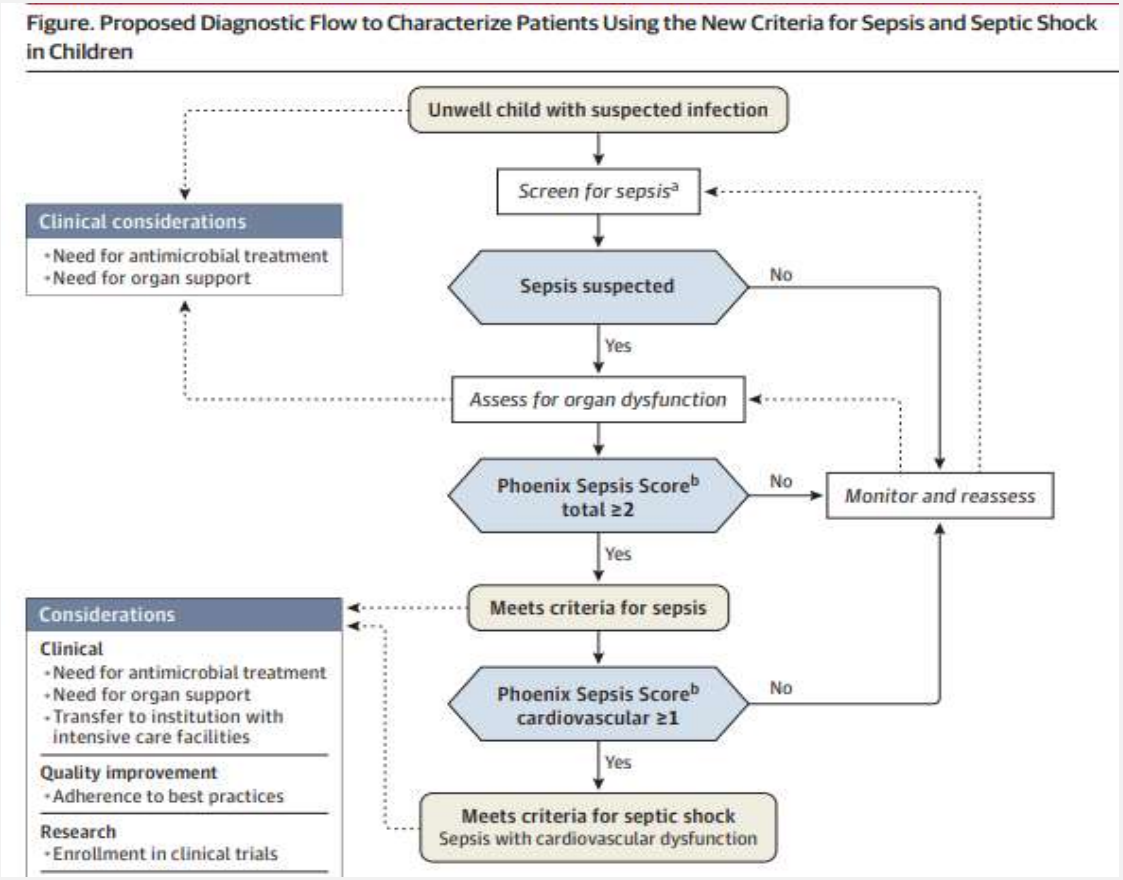

How Does One Define Sepsis in Kids Now? Ah. There it is.
Sepsis

Figure. Proposed Diagnostic Flow to Characterize Patients Using the New Criteria for Sepsis and Septic Shock in Children



The Phoenix Sepsis Score is not intended for early screening or recognition of possible sepsis and management before organ dysfunction is overt

- Uses a composite **۴-organ system model**
  - **Respiratory**
  - **Cardiovascular**
  - **Neurological**
  - **Coagulation Dysfunction**
- **Sepsis** and **Life-threatening Organ Dysfunction** in children with **Suspected/Confirmed Infection** can be identified as a **Phoenix Score of at least ۲ points.**
- "**Severe Sepsis**" is a term that **should NOT be used** any longer.
  - *Essentially, once you have defined organ dysfunction that mets Sepsis criteria, the illness is severe.*
  - SIRS is also not used.
  - **Septic Shock is a subset of Sepsis.**
    - Have **Cardiovascular Dysfunction** and **higher mortality.**
    - Defined by a **Cardiovascular score of at least ۱ point.**

Phoenix Sepsis Score

Table 85.2 The Phoenix Sepsis Score[a]

VARIABLES	0 POINTS	1 POINT	2 POINTS	3 POINTS
RESPIRATORY, 0–3 POINTS	$PaO_2:FIO_2$ ≥400 or $SpO_2:FIO_2$ ≥292[b]	$PaO_2:FIO_2$ <400 on any respiratory support or $SpO_2:FIO_2$ <292 on any respiratory support[b,c]	$PaO_2:FIO_2$ 100–200 and IMV or $SpO_2:FIO_2$ 148–220 and IMV[b]	$PaO_2:FIO_2$<100 and IMV or $SpO_2:FIO_2$ <148 and IMV[b]
CARDIOVASCULAR, 0–6 POINTS		1 point each (up to 3)	2 points each (up to 6)	
	No vasoactive medications[d]	1 Vasoactive medication[d]	≥2 Vasoactive medications[d]	
	Lactate <5 mmol/L[e]	Lactate 5–10.9 mmol/L[e]	Lactate ≥11 mmol/L[e]	
AGE BASED[f]	Mean Arterial Pressure, mm Hg[g]			
<1 mo	>30	17–30	<17	
1 to 11 mo	>38	25–38	<25	
1 to <2 yr	>43	31–43	<31	
2 to <5 yr	>44	32–44	<32	
5 to <12 yr	>48	36–48	<36	
12 to 17 yr	>51	38–51	<38	
COAGULATION, 0–2 POINTS[H]		1 point each (maximum 2 points)		
	Platelets ≥100 × 10^3/μL	Platelets <100 × 10^3/μL		
	International normalized ratio ≤1.3	International normalized ratio >1.3		
	D-dimer ≤2 mg/L FEU	D-dimer >2 mg/L FEU		
	Fibrinogen ≥100 mg/dL	Fibrinogen <100 mg/dL		
NEUROLOGIC, 0–2 POINTS[I]	Glasgow Coma Scale score >10; pupils reactive[i]	Glasgow Coma Scale score ≤10[i]	Fixed pupils bilaterally	
PHOENIX SEPSIS CRITERIA				
Sepsis	Suspected Infection and Phoenix Sepsis Score ≥2 points			
Septic shock	Sepsis with ≥1 cardiovascular point(s)			

- The Phoenix Sepsis Score is **NOT intended to be used as an early screening tool** to help recognize possible sepsis in children.
- **The Score correlates with in-hospital mortality!**
- The Phoenix Sepsis Score is aimed at:
  - **Improving clinical care**
  - **Epidemiological assessment**
  - **Research** on pediatric sepsis and septic shock **globally**

CLINICAL MANIFESTATIONS

- *Shock may initially manifest as only tachycardia, with or without tachypnea.* Progression leads to decreased urine output, poor peripheral perfusion, respiratory distress or failure, alteration of mental status and low BP
- A significant misconception is that shock occurs only with low BP.

Hypotension is often a late finding, especially in infants and young children, because of a complex set of compensatory mechanisms that attempt to preserve BP and peripheral perfusion.

- Hypotension reflects an advanced state of uncompensated shock and is associated with increased morbidity and mortality.

- In septic shock, it is important to distinguish between the inciting infection and the host inflammatory response.
- Normally, host immunity prevents the development of sepsis through activation of the reticular endothelial system along with the cellular and humoral immune systems. This host immune response produces an *inflammatory cascade* mediated by hormones, cytokines, and chemokines.
- If this inflammatory cascade is uncontrolled, derangement of the microcirculatory system leads to subsequent organ and cellular dysfunction.

- **Systemic inflammatory response syndrome (SIRS)**

is an inflammatory cascade that is initiated by the host response to an infectious or noninfectious trigger when the host defense system **does not adequately recognize and/or eliminate the triggering event.**

The inflammatory cascade initiated by shock can lead to hypovolemia, cardiac and vascular failure, acute respiratory distress syndrome (ARDS), insulin resistance, coagulopathy, and unresolved or secondary infection.

Table 85.3 Differential Diagnosis of Systemic Inflammatory Response Syndrome (SIRS)

INFECTION
Bacteremia or meningitis (*Streptococcus pneumoniae, Haemophilus influenzae* type b, *Neisseria meningitidis*, group A *Streptococcus, Staphylococcus aureus*)
Viral illness (influenza, enteroviruses, hemorrhagic fever group, herpes simplex virus, respiratory syncytial virus, cytomegalovirus, Epstein-Barr virus, COVID-19)
Encephalitis (arboviruses, enteroviruses, herpes simplex virus)
Rickettsiae (Rocky Mountain spotted fever, Ehrlichia, Q fever)
Syphilis
Vaccine reaction (pertussis, influenza, measles)
Toxin-mediated reaction (toxic shock, staphylococcal scalded skin syndrome)

CARDIOPULMONARY
Pneumonia (bacteria, virus, mycobacteria, fungi, allergic reaction)
Pulmonary emboli
Heart failure
Arrhythmia
Pericarditis
Myocarditis

METABOLIC-ENDOCRINE
Adrenal insufficiency (adrenogenital syndrome, Addison disease, corticosteroid withdrawal)
Electrolyte disturbances (hyponatremia or hypernatremia, hypocalcemia or hypercalcemia)
Diabetes insipidus
Diabetes mellitus
Inborn errors of metabolism (organic acidosis, urea cycle, carnitine deficiency, mitochondrial disorders)
Hypoglycemia

GASTROINTESTINAL
Gastroenteritis with dehydration
Volvulus
Intussusception
Appendicitis

HEMATOLOGIC/IMMUNE
Anemia (sickle cell disease, blood loss, nutritional)
Methemoglobinemia
Splenic sequestration crisis
Leukemia or lymphoma
Hemophagocytic syndromes
Cytokine release syndrome s/p CAR-T therapy
Immune reconstitution syndrome
Graft-versus-host disease

NEUROLOGIC
Intoxication (drugs, carbon monoxide, intentional or unintentional overdose)
Intracranial hemorrhage
Infant botulism
Trauma (child abuse, accidental)
Guillain-Barre syndrome
Myasthenia gravis

OTHER
Anaphylaxis (food, drug, insect sting, idiopathic)
Hemolytic-uremic syndrome
Kawasaki disease
Erythema multiforme, toxic epidermal necrolysis
MIS-C
Poisoning, iron, cyanide
Toxic envenomation
Systemic JIA
Macrophage activation syndrome
Idiopathic systemic capillary leak (Clarkson) syndrome

DIAGNOSIS

- Shock is a clinical diagnosis that should be primarily based on thorough history and physical examination
- History

recent trauma or bleeding, exposure to new medications or substances, preceding or on current symptoms of infection or fluid losses, the patient's comorbid conditions

- Physical examination

include rapid assessment of vital signs (including pulse oximetry),mental status/level of consciousness, extremity temperature, peripheral and central pulses, capillary refill, and presence of rashes or skin changes

LABORATORY FINDINGS

- early laboratory testing to screen for hyperlactatemia (as a sign of increased anaerobic metabolism), acidemia, and electrolyte disturbances, including hypoglycemia and hypocalcemia.

Laboratory evidence of concurrent MODS :

- a complete blood count, renal function tests, liver function tests, and coagulation profile (including fibrinogen and d-dimer or fibrin split products). Elevated neutrophil counts with increased immature forms (i.e., bands, myelocytes, promyelocytes) and elevated CRP and procalcitonin levels may be seen with infection. blood gas. central venous oxygen saturation (Svo_2)

at least one blood culture should be collected along with other cultures as supported by the history and physical.

Ferritin levels should be determined in all patients with septic shock.

- In addition to the traditional description of septic shock, a subgroup immunophenotype presents with **hyperferritinemia**.
- Hyperferritinemic septic shock may be due to familial primary genetic hemophagocytic lymphohistiocytosis (HLH) or, more often, secondary HLH initiated by an infectious agent or macrophage activation syndrome (MAS) associated with autoimmune diseases.

Considerations in Management of Hyperferritinemic Sepsis Beyond Usual Diagnostic and Therapeutic Measures Used in Sepsis Patients Without Hyperferritinemia

WHAT OCCULT INFECTION IS PRIMING AND WHAT CO-INFECTION IS PRESENT FOR THE HYPERFERRITINEMIC PROCESS?

Perform diagnostic workup for **viruses** (e.g., herpes simplex virus 1, HHV-6, HHV-8, Epstein-Barr virus, adenovirus, cytomegalovirus, parvovirus, Ebola, COVID-19, Dengue, hepatitis A, HIV, severe fever with thrombocytopenia syndrome virus, influenza, hemorrhagic fevers), **parasites** (e.g., toxoplasmosis, leishmaniasis, malaria, scrub typhus, babesiosis), **intracellular bacteria** (e.g., tuberculosis, atypical mycobacteria, mycoplasma, fusobacteria, babesiosis, ehrlichiosis), rickettsia, and **fungi** (e.g., histoplasmosis).

Begin empiric therapy and specific therapies according to context.

Give IVIG if no specific therapy is available to neutralize infection.

IS HEMOLYSIS AND FREE HEMOGLOBIN DRIVING THE HYPERFERRITINEMIC PROCESS?

Measure free hemoglobin and ferritin sequentially.

Minimize blood transfusions.

If unable to reverse hemolysis or hyperferritinemia, consider 5 days of plasma exchange to remove free hemoglobin and ferritin and to resolve inflammation.

IS AN ANTIINFLAMMATORY STRATEGY NEEDED TO SAFELY CONTROL INFLAMMASOME ACTIVATION?

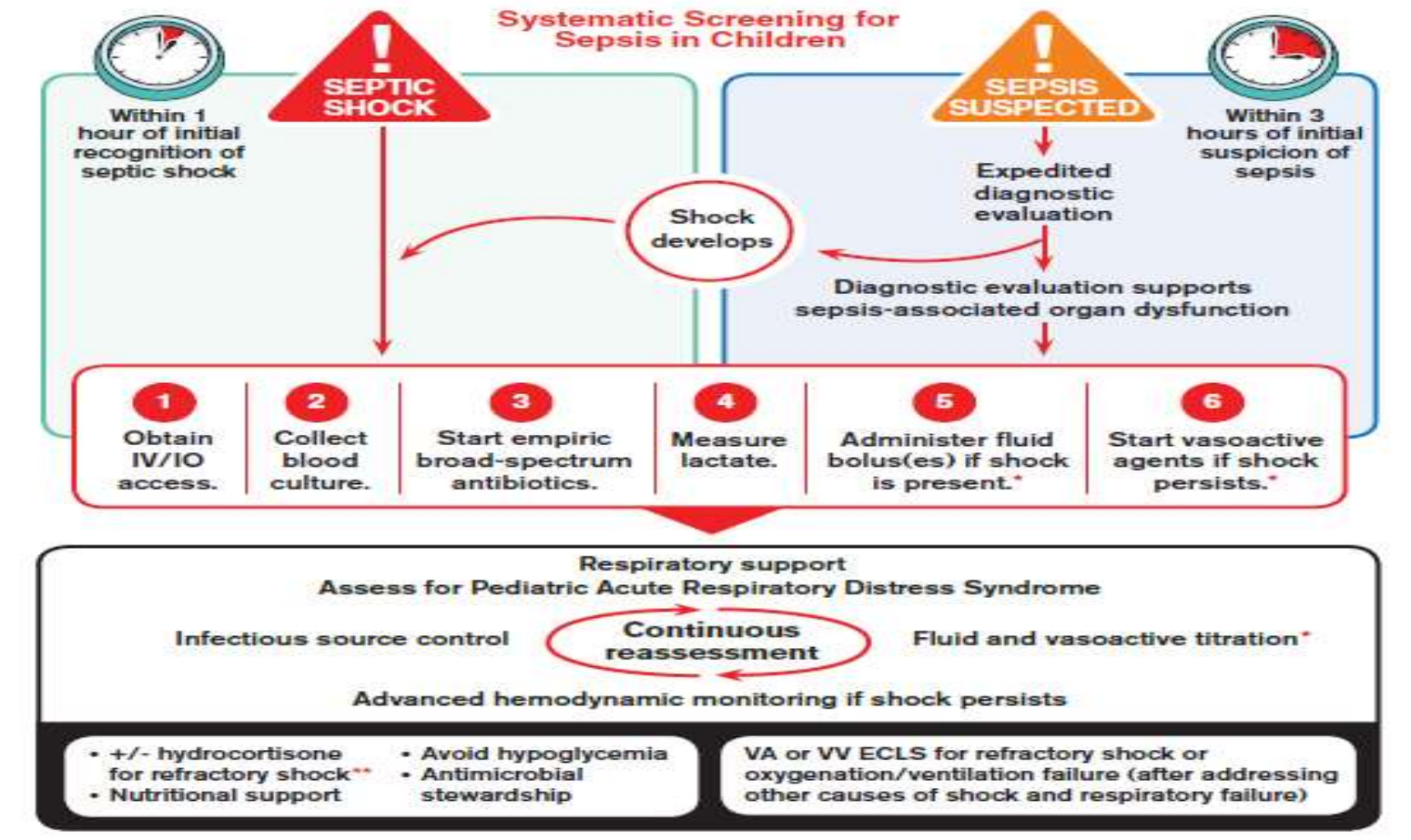
If ferritin is >3,000 ng/mL in the high-resource PICU setting, or >500 ng/mL in the resource-poor PICU setting, then mortality risk is increased.

Methylprednisone, IVIG, and interleukin 1 receptor antagonist (anakinra) can be safely given to reduce mortality risk in these children.

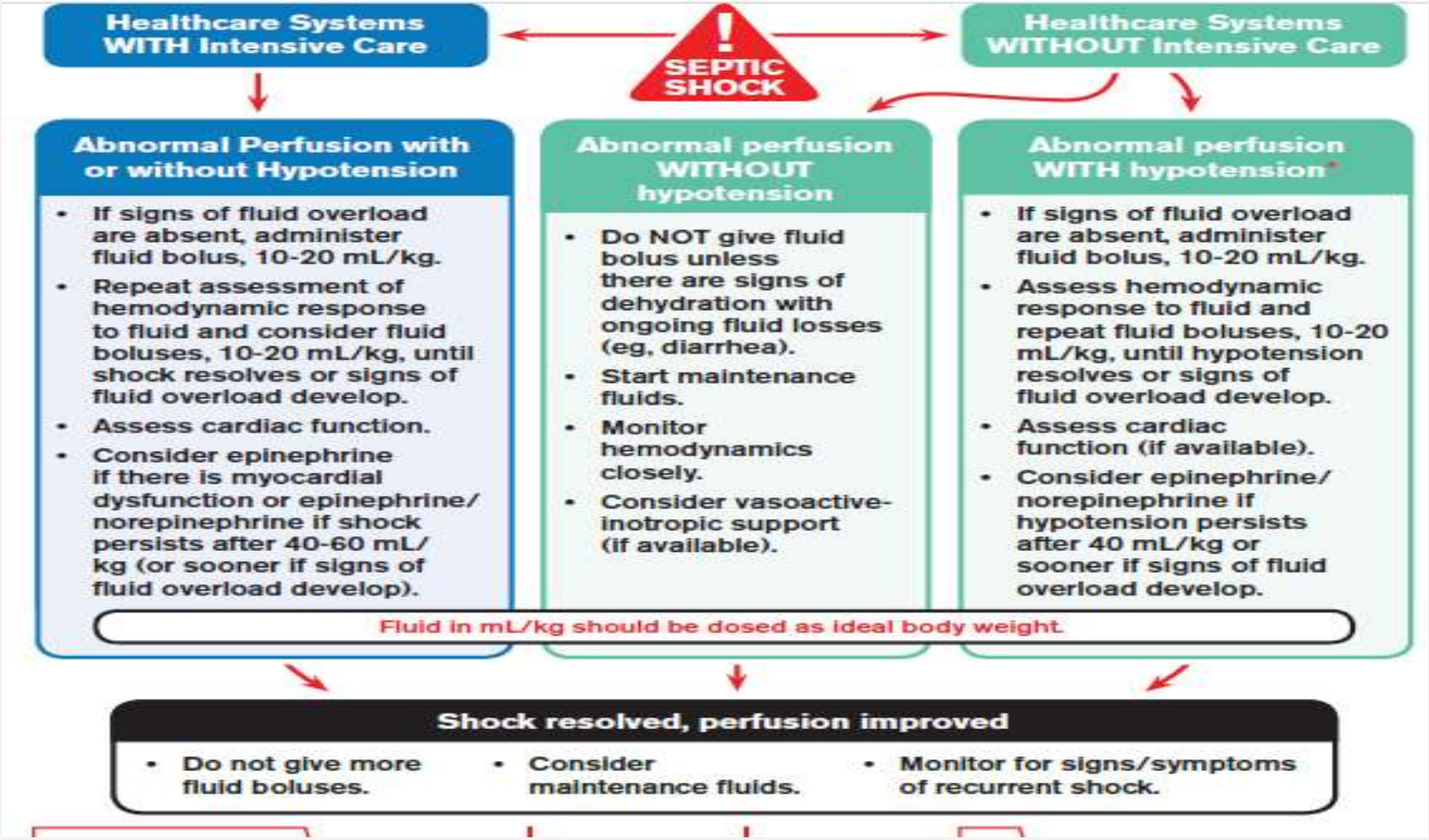
General Principles of Shock Management

- Restoration of oxygen delivery includes ensuring airway patency and adequacy of breathing, providing supplemental oxygen if hypoxemia ($Spo^{۲}$ <۹۲–۹۴%)
- Establishing vascular access
- reversing the underlying etiology of shock
- expanding the circulating blood volume
- supporting the cardiac and vascular system with appropriate vasoactive agents when necessary
- frequently reassessing the patient's response to initial therapy

Initial Resuscitation Algorithm for Children



Fluid and Vasoactive-Inotrope Management Algorithm For Children



Targets for Shock Reversal

- Clinical signs of successful resuscitation include :

a decrease in HR and respiratory rate, increase in BP, improved urine output to >۰/۵ mL/kg/hr, normalization of mental status, decreased capillary refill time, warmth of distal extremities, and improved peripheral pulses.

- laboratory measurements include:

rise in $Svo^{۲}$ to >۷۰% ,decreasing trend in blood lactate levels (normalization of blood lactate to <۲ mmol/L within ۴ hours), Blood pH and base deficit

- serial POCUS to assess volume status, cardiac output(CI= ۳/۳ and ۶/۰ $L/min/m^{۲}$)

Fluid-Refractory and Catecholamine-Resistant Shock

- If shock persists despite volume resuscitation and vasoactive support with an initial catecholaminergic agent

Such children have an increased risk for MODS and death, especially if requirement for vasoactive support is high, blood lactate exceeds ٨ mmol/L (or is rising after ٢ hours of resuscitation), and/or myocardial dysfunction

- Several additional management in this scenario :

evaluation for atypical and/or reversible etiologies

second-line vasoactive medications

stress-dose corticosteroids

extracorporeal membrane oxygenation (ECMO)

PROGNOSIS

- Risk of death involves a complex interaction of factors including:

the underlying etiology

presence of chronic illness

host immune response

timing of recognition and therapy


THANK
YOU!