Pediatric Shock

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OBJECTIVES

Definition
Pathophysiology
Types and Sx
Labs
Management concepts: General and specific

Definition

Shock is a *significant, systemic reduction in tissue perfusion,* resulting in decreased oxygen delivery to tissues

Causes

- Decreased intravascular volume
- Abnormal distribution of intravascular volume
- Impaired cardiovascular function.
- Impaired O2 utilization

Definition

It is a state of cellular and tissue hypoxia as a result of

- Reduced O2 delivery (hypovolemia, heart failure)
- Increased oxygen consumption (severe sepsis)
- Inadequate oxygen utilization (toxins)

Another Definition

Inability to meet the cellular demand for oxygen

Oxygen delivery is less than Oxygen Consumption

Effects of Shock

Prolonged shock may lead to cellular dysfunction and death:

- Membrane ion pump dysfunction
- Intracellular edema
- Leakage of intracellular contents into the extracellular space
- Inadequate regulation of intracellular pH

Physiology

Variables involved:

- Cardiac Output
- Stroke Volume
- Systemic Vascular Resistance
- Oxygen consumption

Important concepts

CO= HR X Stroke Volume

Heart rates in normal children

Age	Awake rate	Sleeping rate
Neonate (28 days or younger)	100-205	90-160
Infant (29 days to 1 year)	100-180	90-160
Toddler (1 to <3 years)	98-140	80-120
Preschool (3 to 5 years)	80-120	65-100
School-aged child (6 to 12 years)	75-118	58-90
Adolescent (>12 years)	60-100	50-90

Data from Pediatric Advanced Life Support Provider Manual, American Heart Association, 2016. p. 48.

Important concepts: Stroke Volume

Stroke volume is determined by :

- Preload
- Cardiac contractility
- Afterload.

Compensatory mechanisms that improve stroke volume include:

- Increased venous smooth muscle tone (improves preload by shunting blood to the heart)
- Increased cardiac contractility (resulting in more complete emptying of the ventricles).

Children can’t modify stroke volume well so rely on heart rate

Preload

- The volume of blood filling the heart (especially the ventricles) at the end of diastole.
- It depends on venous return and blood volume.
- ↑ Preload (e.g. in fluid resuscitation) can improve cardiac output.
- ↓ Preload (e.g. in dehydration or bleeding) can lead to shock.

AfterLoad

The resistance the heart must overcome to pump blood out to the body

Afterload is influenced by

- Systemic vascular resistance (SVR)
- Aortic pressure.

↑ Afterload (e.g. in cold shock or aortic stenosis) makes it harder for the heart to pump, which may reduce cardiac output.

Preload

Filling volume before heart contracts

Affected by hydration, blood volume

Afterload

Resistance against which heart pumps

Affected by SVR, BP, congenital heart lesions

Increased Systemic vascular resistance

This is a ***compensatory*** mechanism to:

Maintain perfusion pressure (measured as blood pressure) despite decreased cardiac output.

Shunt blood toward vital organs such as the heart and CNS and away from peripheral structures (including skin, muscle, kidneys, and splanchnic organs)

*Children with **compensated** shock typically have normal blood pressures, despite signs of poor perfusion (such as decreased peripheral pulses and **tachycardia**).*

How to determine adequate O2 delivery

- Blood flow to tissues (cardiac output CO)
- Balance between blood flow and metabolic demand in tissues
- O2 content of blood (hemoglobin concentration and percentage of hemoglobin saturated with oxygen)
- Ability of Hb to let go of O2 (2,3 DPG Levels)

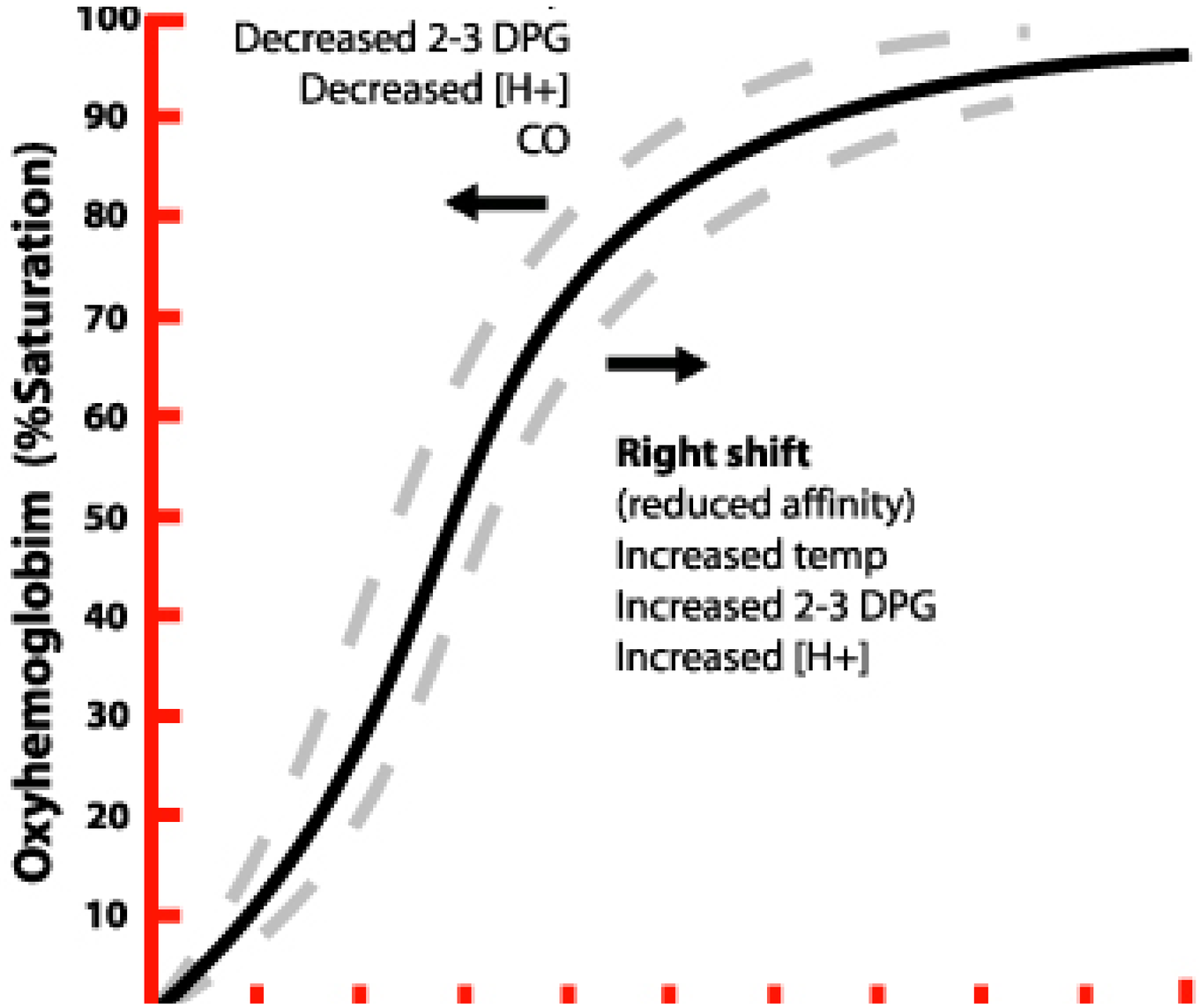
How to determine adequate O2 delivery

2,3-DPG

2,3-DPG is a key regulator inside RBCs that helps hemoglobin “let go” of oxygen where tissues need it.

High levels → more O_2 delivery to tissues

Low levels → hemoglobin holds onto O_2 too tightly.


Oxyhemoglobim (%Saturation)
100
90
80
70
60
50
40
30
20
10
Decreased 2-3 DPG
Decreased [H+]
CO
Right shift
(reduced affinity)
Increased temp
Increased 2-3 DPG
Increased [H+]

🩺 Clinical Situations That Affect 2,3-DPG:

↑ 2,3-DPG (Right Shift)	↓ 2,3-DPG (Left Shift)
Chronic hypoxia (e.g., high altitude, COPD)	Blood stored for transfusion
Anemia	Septic shock (sometimes)
Hyperthyroidism	Hypophosphatemia
Acidosis	Alkalosis
Exercise	Multiple transfusions

How to determine adequate O2 delivery
VO_2 vs DO_2

VO_2 Oxygen consumption by tissues

Indicates how well tissues are extracting and using O_2

DO_2 : Oxygen delivered to tissues

Depends on Hb, CaO_2, CO

Critical Concepts

Normal DO2: 500 ml/min/m2

Normal oxygen extraction : 25-30%

Normal oxygen consumption in an adult is 250 ml/min

Arterial Oxygen Content. What is it?

CaO_2 = Arterial Oxygen Content

- The Total amount of oxygen in arterial blood
- It is Crucial for tissue oxygen delivery (DO_2)

CaO_2

It has 2 parts

1. Hb-bound O_2:
    - $Hb \times SaO_2 \times 1.34$
    - Major contributor 98%
2. Dissolved O_2:
    - $PaO_2 \times FiO_2 \times 0.003$~1–2%)

CaO2 calculation

Formula:

$CaO_2=(Hb\times SaO_2\times 1.34)+(PaO_2\times FiO_2\times 0.003)$

SaO_2: Arterial oxygen saturation

PaO_2: Partial pressure of O_2

FiO_2 :Inspired oxygen fraction

1.34 : O_2 binding capacity of Hb

0.003 :O_2 solubility in plasma

How CaO_2 Components Are Measured

- Hb Hemoglobin (CBC) – venous blood sample
- SaO_2 Arterial oxygen saturation (bound to Hb) Pulse oximetry (SpO_2) or Arterial Blood Gas (ABG)
- PaO_2 Oxygen dissolved in plasma Arterial Blood Gas (ABG)
- FiO_2 Fraction of inspired oxygen Set on O_2 device (e.g., nasal cannula, mask, ventilator)
- 1.34 O_2 binding capacity of hemoglobin (mL O_2/g Hb) Physiological constant (no measurement needed)
- 0.003 Solubility of O_2 in plasma (mL O_2/mmHg) Physiological constant (no measurement needed)
-

CaO_2

CaO_2 = Hb bound O_2+ Plasma Dissolved O_2

$(\underline{Hb} \times \underline{SaO_2} \times 1.34) + (PaO_2 \times FiO_2 \times 0.003)$

$(12 \times 0.95 \times 1.34) + (80 \times 0.21 \times 0.003)$

15.28 + 0.05 = 15.33 ml/dl

Normal CaO_2 :17-20 ml/dl

CaO2 and O2 Delivery

- $DO_2 = CaO_2 \times$ Cardiac Output
- Even high CaO_2 is not enough if cardiac output is low
- All three must be optimized:
  - Oxygen content
  - Cardiac output
  - Tissue perfusion

🩺 CaO_2 in Shock - Key Clinical Relevance

Hypovolemic Shock

↓ Preload → ↓ Cardiac Output (CO) → ↓ Oxygen Delivery (DO_2)

Cardiogenic Shock

↓ Contractility → ↓ CO → Poor oxygen delivery to tissues

Septic Shock (Early Phase)

Maldistribution of blood flow → Tissues not receiving enough oxygen

Anemic Shock

Severe ↓ Hemoglobin (Hb) → ↓ CaO_2, even if SaO_2 and PaO_2 are normal

Hypoxic Shock

↓ PaO_2 → ↓ SaO_2 → ↓ CaO_2 → Inadequate oxygen in the blood

DO2 , VO2 and O2 extraction

Oxygen Dynamics in Pediatrics: Normal vs Shock

Parameter	Normal State	Hypovolemic Shock	Cardiogenic Shock	Septic Shock (Early)	Septic Shock (Late/Cold)	Anemic/ Hypoxic Shock
DO_2 (O_2 delivery)	Normal (~500–600 mL/min/m^2)	↓↓ due to ↓ preload and ↓ CO	↓↓ due to ↓ contractility and CO	Normal or ↑ (high-output state)	↓ due to ↓ CO and vasoconstriction	↓ due to ↓ Hb (anemia) or ↓ PaO_2 (hypoxia)
CaO_2 (arterial O_2 content)	Normal (17–20 mL/dL)	Normal unless bleeding is severe	Normal	May be normal or ↓ (if hypoxia or anemia)	May be ↓ (due to hypoxia or organ failure)	↓↓↓ due to ↓ Hb or ↓ SaO_2/PaO_2
VO_2 (O_2 consumption)	Normal (~6–8 mL/kg/min in infants)	Normal early → ↓ if DO_2 drops below critical	Normal early → ↓ with low output	Normal or ↑ (↑ demand, fever, metabolic stress)	↓ due to cellular dysfunction and hypoperfusion	↓ if O_2 supply is inadequate
O_2 Extraction Ratio (O_2ER)	Normal: 25–30%	↑↑ (compensatory) → up to 50–60%	↑↑ (trying to maintain VO_2)	May be low early (due to maldistribution)	↑↑ later as body extracts max O_2	↑ (if tissues try to compensate) → then ↓ if supply fails

Types of Shock

Hypovolemic
Distributive
Cardiogenic
Obstructive
Toxin induced

The 3 Stages of Shock

Compensated

Hypotensive Shock

Irreversible Shock

Compensated Shock

Normal BP

Increased HR and Decreased perfusion

Homeostatic mechanisms rapidly compensate for diminished perfusion maintaining and systolic blood pressure by

Increasing HR

Increasing PVR as perfusion decreases so signs of peripheral vasoconstriction (such as cool skin, decreased peripheral pulses, lethargy, and oliguria) can be noted as perfusion becomes further compromised.

Hypotensive Shock

Systolic blood pressure falls

Homeostasis is *overwhelmed*.

Signs and symptoms of organ dysfunction (such as coma, respiratory failure, liver dysfunction, coagulopathy, and/or kidney injury) appear.

Although hypotension is generally a late finding among children with shock, those with early distributive shock (as with sepsis) may have hypotension because of decreased systemic vascular resistance (SVR). In these cases vital organ perfusion is initially maintained by increased cardiac output.

Hypotension by age

Hypotensive Shock = systolic bp less than 5th Percentile)

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

Irreversible Shock

Symptoms of Early Compensated Shock

- Tachycardia (elevated heart rate) — one of the earliest signs.
- Tachypnea (increased breathing rate).
- Delayed capillary refill (>2 seconds) & weak or thready peripheral pulses.
- Cool, clammy extremities (vasoconstriction); skin may look pale or mottled.
- Normal blood pressure (or only minimally reduced) — blood pressure often preserved until later.
- Decreased urine output (oliguria) may begin.
- Altered mental status early: irritability, anxiety, fussiness, restlessness.
- Other nonspecific symptoms: poor feeding, lethargy, weakness.

Symptoms of Hypotensive Shock

• Hypotension (low blood pressure)
• Worsening tachycardia
• Marked mental status changes: lethargy, obtundation, confusion, possibly unconsciousness.
• Very weak or absent peripheral pulses; central pulses may also weaken
• Cold extremities, poor capillary refill, mottled or cyanotic skin.
• Reduced urine output (very low output, or anuria).
• Respiratory distress or failure (increased work of breathing, possible irregular breathing).
• Temperature instability: hypothermia especially in infants or septic shock, or sometimes hyperthermia.

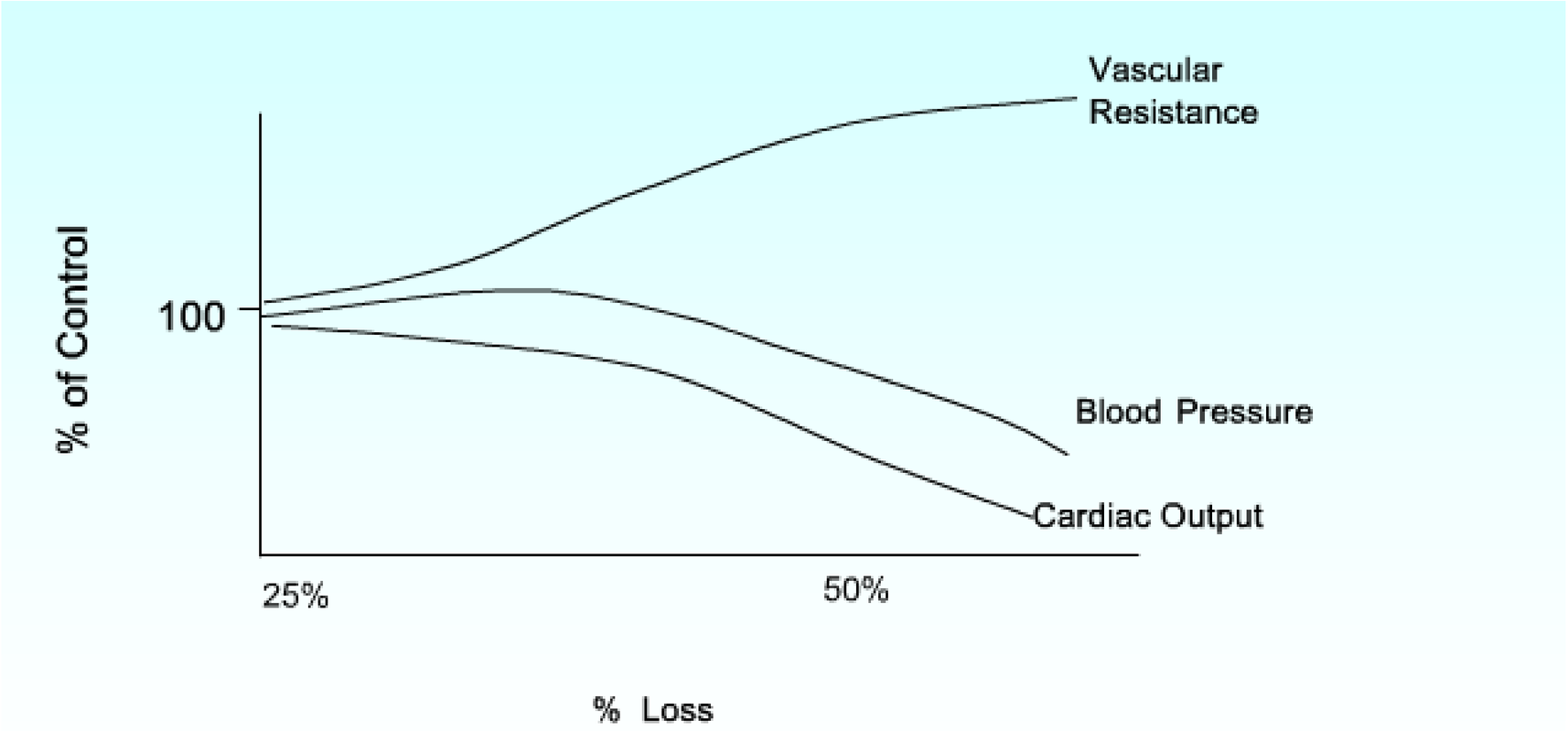
Pearls

- In children, blood pressure is often preserved in the early (compensated) stage; hypotension is a late sign of decompensated shock.
- Because children can compensate well initially, rely on signs of poor perfusion (skin, pulses, cap refill, mental status, urine output) rather than just blood pressure.
- Types of shock (hypovolemic, distributive/septic, cardiogenic, obstructive) may have variations in presentation, especially with skin perfusion (warm vs cold), but the progression from compensated → decompensated follows broadly similar themes.

Hypovolemic Shock

Hemorrhagic : (Think of hidden blood loss like femoral Fracture)
Plasma loss (Burns, Steven Johnson syndrome, Epidermolysis Bullosa)
Loss of water (Gastroenteritis, extreme diuresis in DKA, DI)
Relative hypovolemia (loss of fluid by third spacing)

Hemodynamic Response to Hemorrhage



💧 Hypovolemic Shock

Recent fluid loss (diarrhea, vomiting, bleeding, burns)

- Dry mucous membranes
- Sunken eyes/fontanelle (infants)
- Flat neck veins(older children)
- Cool, pale extremities
- Narrow pulse pressure

Cardiogenic

Congenital heart disease: Ductal dependent lesions such as HLHS, AS, Coarc or interrupted aortic arch

Cardiomyopathies : Dilated, restrictive , hypertrophic

Myocarditis: Infectious (Coxsackie B),Toxins(Cocaine), autoimmune

Abnormal rate or rythm: Extreme bradycardia, SVT, VT

❤️Cardiogenic Shock

- Hepatomegaly
- Gallop rhythm or murmur
- Pulmonary crackles or rales
- Increased work of breathing without improved perfusion
- History of congenital heart disease or recent myocarditis

Distributive shock

Sepsis

SIRS: Systemic Inflammatory Response Syndrome

Anaphylaxis

Neurogenic

Distributive shock

Results from :

- A decrease in systemic vascular resistance
- Abnormal distribution of blood flow within the microcirculation (capillary leaking)

1. Inadequate tissue perfusion.
2. Functional hypovolemia with decreased preload

Distributive shock generally is associated with a normal or increased cardiac output.

🌡️ Distributive Shock (e.g., Septic, Anaphylactic, Neurogenic)

- Bounding pulses (early)
- Warm, flushed skin (early)
- Wide pulse pressure
- History suggestive of infection (fever, rash) or allergy
- Temperature instability (fever or hypothermia)
- Late: cold extremities, weak pulses, hypotension
-

Obstructive

Tension Pneumothorax

Cardiac Tamponade

Pulmonary embolism

 Obstructive Shock

- Distended neck veins (rare in infants)
- Muffled heart sounds (cardiac tamponade)
- Tracheal deviation (tension pneumothorax)
- Sudden respiratory distress
- Chest trauma or known congenital heart defect

Toxic causes of impaired O2 utilization

Cyanide poisoning

Carbon monoxide

Heat stroke:Severe hyperthermia

Toxic causes of impaired O2 utilization: Cyanide

Cyanide poisoning: Cyanide blocks cytochrome oxidase in mitochondria, halting oxidative phosphorylation so cells cannot use oxygen, even if delivery is normal → switch to anaerobic metabolism → lactic acidosis.

Toxic causes of impaired O2 utilization : CO

Carbon monoxide: CO binds to hemoglobin with >200 times the affinity of oxygen, forming carboxyhemoglobin (COHb). Although perfusion may be adequate, oxygen cannot be released to tissues → cellular hypoxia.

Toxic causes of impaired O2 utilization: heat stroke

Severe hyperthermia → direct cellular injury and impaired oxygen utilization.Can cause mitochondrial dysfunction and metabolic derangements → impaired aerobic metabolism.

Types of Shock

modynamic 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 time△	Mixed venous oxygen saturation◇
Hypovolemic	↓	↓	↑	↑	Low
Cardiogenic	↑	↓	↑	↑	Low
Distributive	↓ or ↔	↑	↓	↓ (initial)	High
Obstructive	↑§	↓	↑	↑	Low

Types of Shock

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.

🧪 Labs

CBC

Blood Glucose

Ionized Calcium

Serum lactate

ABG

Sepsis screen

PT/PTT/INR

Labs

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 (ALT)
- Prothrombin and partial thromboplastin times (PT and PTT)
- International normalized ratio (INR)
- Fibrinogen and D-dimer
- Blood culture
- Urinalysis
- Urine culture
- Other cultures as indicated by clinical findings
- Diagnostic serologic testing as indicated to identify suspected sources of infection
- Inflammatory biomarkers (eg, C-reactive protein, procalcitonin) in selected cases

🧪 Supportive Labs/Monitoring

- Elevated Serum Lactate
- Metabolic acidosis
- Decreased SvO_2 or $ScvO_2$
- ECG or CXR abnormalities
- Poor perfusion despite oxygen delivery

🧪 Monitoring : SvO_2 vs $ScvO_2$

- SvO_2 = Mixed venous oxygen saturation
  - Measured in the pulmonary artery via a pulmonary artery (Swan–Ganz) catheter.
  - Reflects the average oxygen saturation of blood returning from the entire body (head, arms, trunk, legs).
  - Considered the gold standard for assessing global tissue oxygen balance.
- $ScvO_2$ = Central venous oxygen saturation
  - Measured in the superior vena cava (usually via a central venous catheter in the internal jugular or subclavian vein).
  - Represents venous return mainly from the upper body and brain.
  - Easier to obtain than SvO_2, often used as a surrogate.

🔷 What does decreased SvO_2 / $ScvO_2$ mean?

Both are indicators of the balance between oxygen delivery (DO_2) and oxygen consumption (VO_2).

- Normal values:
  - SvO_2: ~65–75%
  - $ScvO_2$: ~70–75% (slightly higher, since it excludes lower body venous blood which extracts more O_2)
- Decreased SvO_2 / $ScvO_2$ (<60–65%) suggests:
  - Inadequate oxygen delivery (e.g., low cardiac output, anemia, hypoxemia)
  - Increased oxygen consumption (e.g., fever, seizures, shivering)
  - Impaired oxygen utilization (e.g., sepsis with mitochondrial dysfunction)

🔹 Why important in shock?

- In shock, tissues may extract more oxygen because delivery is insufficient → venous blood returning to the heart is more desaturated.
- Thus, low SvO_2/$ScvO_2$ is an early marker of shock or inadequate resuscitation.
- Conversely, normal or high $ScvO_2$ in septic shock may be misleading — it can mean impaired oxygen extraction at the tissue level.

✅ Clinical use:

- Monitoring $ScvO_2$ is part of goal-directed therapy in pediatric and adult sepsis protocols.
- Target $ScvO_2$ ≥70% is often used as a marker of adequate resuscitation.

Summary – Pediatric Shock Management

- Early recognition = better outcomes
- ABCDE assessment is critical
- Start fluids early (carefully in cardiogenic/septic shock)
- Add vasopressors if needed
- Tailor treatment to shock type
- Monitor response closely

Management Principles

- Shock is a life-threatening condition requiring immediate recognition and intervention.
  - Children compensate well—hypotension is a late sign.
  - Early identification and treatment improve survival.

Initial Management Principles ABCDE

✅ A – Airway

- Ensure airway patency
- Intubate if GCS < 8 or poor respiratory effort

✅ B – Breathing

- Give high-flow oxygen
- Monitor SpO_2 and work of breathing

✅ C – Circulation

- Establish IV or IO access
- Begin fluid resuscitation
- Monitor cap refill, pulse quality, urine output

Initial Priorities (ABCDE Approach)

✅ D – Disability

- Assess consciousness (AVPU or GCS)
- Check blood glucose and treat hypoglycemia

✅ E – Exposure

- Undress to examine fully
- Avoid hypothermia

🎯 Goals of therapy

- Restore perfusion
- Normalize mental status
- Maintain age-appropriate BP
- Achieve good urine output

MANAGEMENT PRINCIPLES

Decrease O2 consumption

- Minimize work of breathing
- Treat Fever
- Treat pain and anxiety
- Treat Seizures
- Treat Infection

Management Basics

Check blood sugar and serum electrolytes including Ca & Mg
If anaphylaxis suspected give epinephrine, diphenhydramine and hydrocortisone.
Conside H2 blocker.
Continous monitoring of HR, RR, BP, SaO2 & Urine output

Fluid Resuscitation

💧 Use isotonic crystalloids (NS or Ringer’s Lactate)

💧 Initial bolus: 10–20 mL/kg over 10–15 minutes(if you suspect cardiogenic shock start with 5-10 ml/kg 💧

Reassess: HR, perfusion, mental status

💧 Up to 60 mL/kg in first hour if needed

⚠️ Caution: in septic or cardiogenic shock, reassess frequently to avoid overload

Fluid Resuscitation

Reasses following each bolus (HR,CRT, Pulses,BP)

May repeat if some improvement noted up to 4-5 boluses

General principles if not responding to Fluid resusitation

If cardiovascular exam not back to normal consider:

- Starting Inotropic support
- Start Emperic Antimicrobial Therapy for suspected Septic Shock
- In Infants with suspected ductus dependant lessions start Prostaglandin E1 drip to reopen the ductus
- For Hemorrhagic Shock give PRBC’s
-

Type-Specific Management

Hypovolemic Shock

- Most common in children (e.g., diarrhea, bleeding)
- Rapid boluses of isotonic fluid
- Blood products if hemorrhage
- Monitor for response (UO, perfusion, mental status)

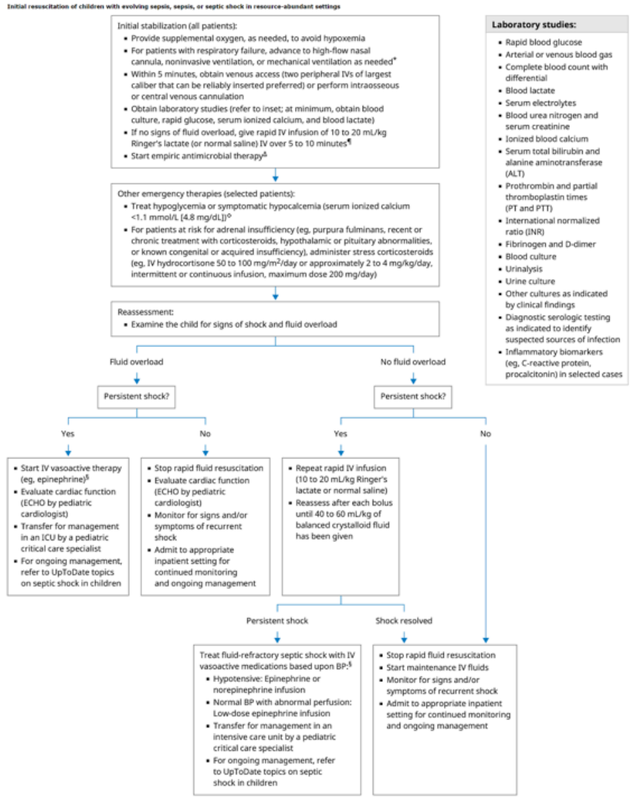
Cardiogenic Shock

- Avoid fluid overload
- Start inotropes early (e.g., milrinone)
- May need PICU, ventilation

Distributive Shock (e.g., Sepsis)

- Early IV antibiotics (within 1 hour!)
- Fluids + vasopressors as needed
- Treat fever, support organs
- Monitor lactate and perfusion

Flow Chart For treatment of sepsis



Vasoactive Drugs (if fluids inadequate)

🩺 Persistent poor perfusion despite fluids

Start vasoactive support:

- Start via central line when possible
- Titrate to maintain perfusion, normal BP for age

Obstructive Shock

Causes:

tension pneumothorax, tamponade, PE

- Immediate intervention (e.g., needle decompression)
- Support circulation and breathing

Dissociative Shock

Cyanide or carbon monoxide poisoning

- High-flow O_2, antidotes if available
- Consider hyperbaric oxygen for CO poisoning

Monitoring

🔍 Continuous reassessment

- Heart rate
- Cap refill
- BP (age-appropriate)
- Urine output ≥ 1 mL/kg/hr
- Mental status
- Serum lactate

Management Refractory Shock

In Refractory shock consider Adrenal Insufficiency or alternative reasons for poor O2 delivery to tissues

Asses End Point Organ perfusion

- Level of Conciousness
- BP, Pulses
- Urine Output

Management Refractory Shock

If shock still not responding consider stat Echo to assess function and volume status

To *Improve contractility* consider Milrinone Or Dobutamine

If Contractility poor despite inotropic support consider afterload reduction with vasodilators such as Nitroprusside

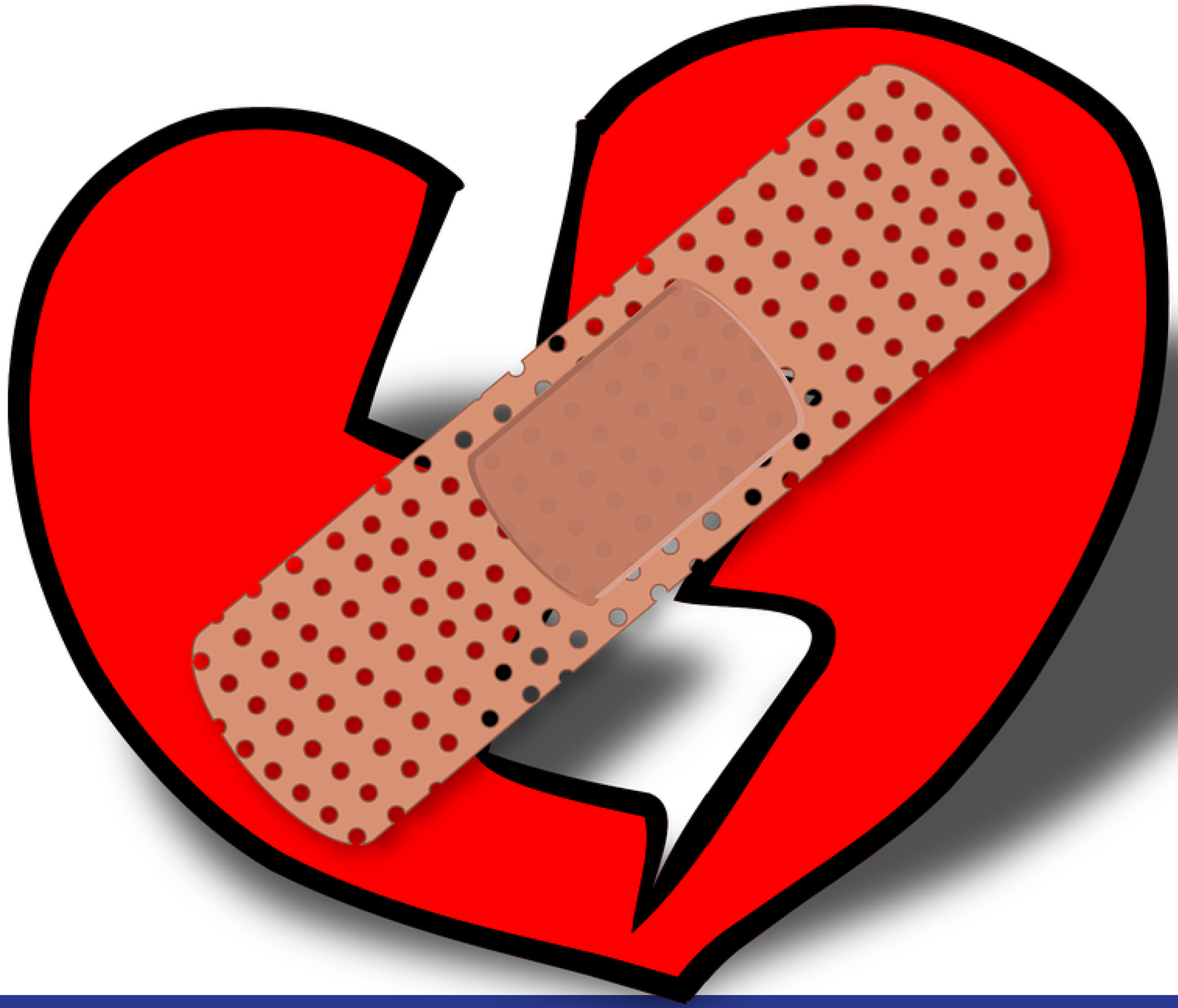
Management Refractory Shock

In Refractory shock send cortisol levels and start stress dose Hydrocortisone
Vasopressin may be use if patient not responding to vasopressor therapy

Outcomes

Depends on cause

The sooner you make the diagnosis and start therapy the better the outcome



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Recognition of Respiratory Failure and Shock in Children by Dr. Monica Kleinman

Summarize the clinical basics of fever

Describe the physiology of body temperature regulation and the pathogenesis of fever

Discuss the approach to evaluation and management of a fever in a child