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SYNOPSIS

Managing Shock in Paediatrics: A Practical Clinical Review

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Introduction

Shock is a pathophysiologic state characterised by a significant reduction in tissue perfusion and impaired oxygen delivery, resulting in inadequate delivery of oxygen and nutrients to meet tissue metabolic demands.[1] It usually results from a dysfunction in the circulatory system. This hypoxic state leads to anaerobic metabolism at the tissue level and production of lactic acid. As this progresses, there is disruption of cellular function leading to cellular failure and death.[1]

Shock is a common high-anxiety-provoking paediatric emergency with a prevalence of almost 50% of paediatric emergency room admissions. It is a leading cause of mortality in children worldwide, accounting for about 10 million deaths annually, with the majority of these deaths occurring in children under 5 in developing countries. [2-4]

Sepsis and hypovolemia (often from infectious gastroenteritis) are the most frequent causes, especially in children under five years. [3, 5] Children can maintain blood pressure for longer than adults in the presence of a compromised circulatory system because compensatory mechanisms such as tachycardia and increased systemic vascular resistance (vasoconstriction) are more effective in them. However, care must be taken, because once these compensatory mechanisms fail, they often deteriorate much more rapidly than in adults. Hypotension is an ominous, late-stage sign. Recognition must instead rely on subtle indicators, such as altered mental status, prolonged capillary refill (>2 seconds), and weak pulses. [1, 6]

Hypovolemic shock results from absolute or relative loss of blood, plasma, or other body fluids; distributive shock occurs when blood flow is abnormally distributed. Cardiogenic shock arises from primary heart pump failure, while obstructive shock results from physical obstruction to blood flow. [1, 6]

As shock progresses, the clinical features of different shock types tend to converge, making it more difficult to distinguish between aetiologies. Regardless of the cause or type, the final common pathway is inadequate tissue perfusion and insufficient oxygen supply to meet cellular metabolic demands, and progression from reversible compensated shock to irreversible multi-organ failure and death. Early recognition and prompt institution of appropriate treatment are crucial to preventing progression and improving outcomes in shock.

Pathophysiology and compensatory mechanisms

Oxygen delivery to tissues is determined by blood volume, cardiac output and arterial oxygen. Cardiac output is a product of heart rate and stroke volume. In children, stroke volume is relatively fixed due to non-compliant ventricles. Therefore, children are heart-rate dependent to maintain output.[1] Arterial oxygen content depends on haemoglobin concentration, arterial oxygen saturation, and the arterial partial pressure of oxygen, with most oxygen being carried on haemoglobin and a small portion delivered as dissolved O2. When there is an insult to the circulatory system, the initial compensatory mechanisms are directed at preserving tissue perfusion. Once the body recognises an insult, there is a rapid activation of the sympathetic nervous system. Catecholamines are produced, which increase heart rate and contractility and increase systemic vascular resistance to improve blood flow to the heart and brain (Table I). The RAAS and vasopressin are activated to promote fluid retention, and the respiratory system increases rate to counter the developing metabolic acidosis[1]. In children, these mechanisms are usually so effective that blood pressure often remains normal until the child reaches a critical point of collapse.

Compensated shock (Early Stage)

In compensated shock, vital organ functions are maintained; in this stage, blood pressure remains normal, but signs of poor peripheral perfusion are

present. Tachycardia is often the earliest sign of distress. Because of peripheral vasoconstriction from a high SVR, the skin may be cool, pale, or mottled, and capillary refill time is usually greater than 2 seconds, except in warm shock, where it may be flash or <1 second. There may be mild agitation or confusion, along with reduced urine output.

Decompensated/Hypotensive Shock (Late Stage)

This stage begins when compensatory mechanisms fail. In uncompensated shock, hypotension develops, and organ and cellular function deteriorate. Hypotension is a late and ominous finding in children. Depressed mental status (lethargy/coma), weak or absent central pulses, and severe oliguria/anuria (no urine output).

Multisystem Effects of Shock

- Shock is a generalised state of tissue hypoperfusion that affects multiple organ systems (Figure 1).
- Respiratory failure is common due to fatigue and possibly of lung function.
- Renal failure is usually pre-renal, but can progress to acute tubular necrosis and acute cortical necrosis.
- Hepatic dysfunction is usually key to prognostication, and hepatocellular damage must be halted. Coagulation abnormalities are usually present to some degree.
- Gastrointestinal shock can lead to mesenteric ischaemia, a devastating consequence. There is intense, prolonged splanchnic vasoconstriction; intestinal mucosal hypoxia; and acidosis. This eventually leads to transmural necrosis of the bowel, bacterial translocation, sepsis, and multisystem organ dysfunction.
- Endocrine - multiple.

Types of shock

1. Hypovolemic Shock

This is the most common type of shock in children globally. It results from a decrease in intravascular volume, which reduces preload and cardiac output.

Causes:

- Non-haemorrhagic: Dehydration from severe gastroenteritis (vomiting/diarrhoea), burns, or third space loss, where fluid leaks into the interstitial space, such as paralytic ileus or nephrotic syndrome.
- Haemorrhagic: Acute blood loss from trauma or gastrointestinal bleeding.
- Clinical Features: Tachycardia, cool and pale extremities, prolonged capillary refill (>2 seconds), dry mucous membranes, and decreased urine output. They are usually tachypnoeic without significant respiratory distress as the body compensates for metabolic acidosis.

2. Distributive Shock

This occurs due to abnormal vasodilation, which causes blood to pool in the peripheral vessels. This results in a relative hypovolemia because the blood volume is present but not reaching vital organs effectively.

Causes:

- Sepsis: The most frequent cause of distributive shock.
- Anaphylaxis
- Neurogenic Shock: Spinal cord or brain injury that disrupts sympathetic control of vascular tone.

Clinical Features:

- Warm Shock: Common in early sepsis or anaphylaxis. Features include warm, flushed skin, "flash" capillary refill (<1 second), and bounding peripheral pulses.
- Cold Shock: More common in infants and young children with sepsis, where they compensate with intense vasoconstriction, appearing more like hypovolemic shock.
- Neurogenic specific: Hypotension without the expected reflex tachycardia (bradycardia or normal heart rate).

3. Cardiogenic Shock

This is caused by primary pump failure, where the heart is unable to maintain sufficient cardiac output despite adequate volume.

- Causes: Viral myocarditis, cardiomyopathies, severe arrhythmias (e.g., supraventricular tachycardia), or congenital heart defects.
- Clinical Features: Unlike hypovolemic shock, these children often show significant respiratory distress (grunting, retractions) due to pulmonary oedema. Other signs include hepatomegaly (enlarged liver), a gallop heart rhythm, and weak pulses in all extremities.

4. Obstructive Shock

This results from a physical obstruction to blood flow into or out of the heart or great vessels.

Causes:

- Tension Pneumothorax
- Cardiac Tamponade
- Massive Pulmonary Embolism
- Ductal-dependent congenital heart lesions: In neonates, when the ductus arteriosus closes (e.g., coarctation of the aorta).

Clinical Features: Similar to cardiogenic shock with signs of heart failure (hepatomegaly, jugular venous distention), but may also include specific signs like unilateral absent breath sounds (pneumothorax) or muffled heart tones (tamponade)

Approach to the Management of Shock

A. Initial resuscitation

The first step in the resuscitation of a child in shock is to evaluate and stabilise the airway, breathing and circulation immediately. Immediately commence non-invasive monitors, such as a pulse oximeter and cardiorespiratory monitor. There must be early consideration for PICU admission or a high dependency unit with capacity for intensive monitoring.

Ensure that the airway is patent and maintainable. Provide high-flow supplemental oxygen (100%) via a non-rebreather mask. In cases of respiratory failure, intubation and mechanical ventilation may be necessary. However, it may be delayed (if the airway can be maintained and oxygenation supported without immediate intervention) until fluid resuscitation has begun, to avoid the negative, potentially life-threatening effects of positive-pressure ventilation on venous return and cardiac stability in hypovolemic patients. [7]

Once the airway and breathing have been stabilised, immediately focus on improving circulation and systemic oxygen delivery. Circulatory improvement is achieved via volume expansion and, if necessary, pharmacologic therapy with vasopressors and cardiac inotropic agents. Establish at least two large-bore IV lines. If IV access cannot be achieved within minutes, an intraosseous (IO) line is recommended as it is just as effective for rapid fluid and medication delivery[8, 9]

B. Fluid Resuscitation

Current clinical guidelines for paediatric shock now focus on a context-specific, personalised approach to fluid resuscitation where the primary goal is to restore tissue perfusion while strictly avoiding the high mortality risk associated with fluid overload.

Type of fluid: The Surviving Sepsis Campaign (SSC) 2020 and updated 2025 PALS guidelines recommend balanced or buffered crystalloids such as Ringer's Lactate and Plasma-Lyte over 0.9% Saline. Balanced solutions have been shown to reduce the risk of hyperchloremic acidosis, acute kidney injury (AKI), and the need for renal replacement therapy (RRT). In contrast, 0.9% Saline has been shown to reduce the length of hospital stay. Conversely, colloids such as starches or gelatins increase the risks of coagulopathy and death and should therefore be avoided.[8, 10]

Volume of boluses: Give repeated 10 -20ml /kg boluses, as necessary, up to a total of 40–60 ml/ kg in the first hour of treatment of hypovolemic or distributive shock. After each bolus, reassess heart rate, pulses, blood pressure, and CRT, as well as signs of fluid overload or cardiac failure (such as fine crepitations, increasing liver edge, or raised jugular venous pressure). If the signs of shock recede, continue maintenance fluids and replace fluid deficit at a slower rate. Consider vasoactive medication and respiratory support if repeated fluid boluses are required.[8, 10]

Although newer guidelines have suggested that in healthcare settings without immediate access to an intensive care, boluses should not be given unless hypotension is present, this may be deleterious in children as hypotension is a late and usually ominous sign in children.

C. Vasoactive agents

If features of shock persist after 40–60 mL/kg of fluid (or earlier if signs of fluid overload are present), vasoactive agents should be started. Vasopressors are drugs that act on vascular smooth muscle and cause vasoconstriction, leading to increased SVR and MAP. Examples include Adrenaline, Noradrenaline, Phenylephrine, and Vasopressin. Inotropes are drugs that increase cardiac contractility, such as Dobutamine and Milrinone. Adrenaline and Noradrenaline are now preferred first-line agents over Dopamine. Adrenaline for cold shock (low cardiac output/high systemic vascular resistance) and noradrenaline for warm shock (vasodilation/low systemic vascular resistance). [7, 11]

Vasoactive meds can be started via peripheral IV or Intraosseous (IO) access if a central line is not yet available to prevent delays.

Table I: Compensatory responses to the shock state

Aim	Response /compensation	
Maintain effective blood volume	Decreased venous capacitance	Increased sympathetic tone Release of adrenaline, vasopressin and angiotensin II
	Decreased renal losses	Reduced GFR, aldosterone release
	Fluid redistribution to IVS	Starling effect(ISS), osmotic effect (ICS)
Maximise cardiac performance	Increased HR and contractility	Increased sympathetic tone and adrenaline release
Preferential perfusion to vital organ	Extrinsic regulation of systemic arterial tone Autoregulation of vital organs	
Optimising conditions for oxygen unloading	Increased concentration of RBC 2-3 DPG Tissue acidosis Decreased tissue PO2	

Table II: Vasoactive agents and their effects

Medication	Primary Mode of Action (Receptors)	Physiological Effect	Primary Indication (Shock Type)
Adrenaline	α1, β1, β2	Increases HR, contractility, and SVR (at higher doses).	First-line for Cold Septic Shock and Anaphylaxis.
Noradrenaline	α1 > β1	Potent vasoconstriction; minimal effect on HR.	First-line for Warm (Distributive) Shock; Fluid-refractory Sepsis.
Dopamine	α, β, Dopaminergic	Dose-dependent (low: renal; mid: HR; high: SVR).	*Second-line* (largely replaced by Adr/Norad in current guidelines).
Milrinone	PDE-3 Inhibitor	Inodilator: Increases contractility while reducing SVR/PVR.	Cardiogenic shock (with normal BP); post-cardiac surgery.
Dobutamine	β1>β2	Increases contractility and HR; mild vasodilation.	Cardiogenic shock; myocardial dysfunction in sepsis.
Vasopressin	V1 receptors	Pure vasoconstriction (non-catecholamine pathway).	Refractory Distributive Shock (Catecholamine-resistant).

D. Special considerations in management

In anaphylactic shock, intramuscular adrenaline is indicated, 10 micrograms/kg. Nebulised adrenaline is given if stridor is present, 0.4 ml/kg of 1:1000 (maximum 5 ml). IM adrenaline can be repeated every 5 minutes as needed. In shock resistant to IM adrenaline and fluid, an infusion of IV adrenaline may be useful.

In cardiogenic shock, use smaller boluses of 5–10 mL/kg over 10–20 minutes with frequent reassessment for pulmonary oedema.

In haemorrhagic shock, give an initial crystalloid bolus of 20 mL/kg, followed quickly by blood products, preferably packed red blood cells.

In obstructive shock, the obstruction must be relieved as soon as possible.

In severe acute malnutrition, avoid rapid boluses; use 10–15 mL/kg/hour only if signs of severe shock are present.

In patients with raised intracranial pressure (ICP) and a mixed picture of shock, the priority is management of shock, as adequate brain perfusion depends on adequate cardiac output. [8, 12]

E. Critical Adjunctive Therapies

- Correct hypoglycaemia with intravenous glucose and address low calcium levels to support cardiac function.
- Corticosteroids: Use IV hydrocortisone only if the shock remains fluid-refractory and catecholamine-resistant (not responding to fluids and high-dose inotropes)[13]

F. Constant Reassessment and Monitoring

A successful shock treatment is described as achieving specific clinical targets within the first hour:

- Capillary refill time <2 seconds
- Normal blood pressure for age
- Normal mental status

- Urine output 1 mL/kg/hr

Arterial blood gas (ABG) monitoring: rising lactate levels and base deficits predict deterioration early and indicate CO2 retention as a marker of impending exhaustion, while decreasing lactate levels are a key marker of improving tissue perfusion.

Multimodal Monitoring: Point-of-Care Ultrasound (POCUS) is increasingly used for immediate assessment of cardiac contractility and volume status, and for detecting signs of fluid overload.[14]

G. Advanced Life Support & Organ Support

- Respiratory Support: Early non-invasive ventilation (NIV) is useful in reducing metabolic demand and should therefore be considered early. Still, invasive ventilation is usually reserved for fluid-refractory shock or respiratory failure.
- Extracorporeal therapies such as extracorporeal membrane oxygenation (ECMO) and continuous renal replacement therapy (CRRT) are used as bridge or rescue therapies for refractory shock and severe fluid overload.[13, 15]

Conclusion

The effective management of paediatric shock hinges on early recognition and prompt institution of physiology-directed therapy. Compensatory mechanisms in children can mask features of shock, so attention must be paid to prioritise early markers of occult hypoperfusion, such as delayed CRT, altered mental status, and rising lactate levels. Current evidence suggests that the use of individualised early fluid resuscitation with balanced fluids, early use of vasoactive medications and integrating advanced tools like POCUS can significantly reduce mortality and ensure better long-term outcomes for critically ill children.

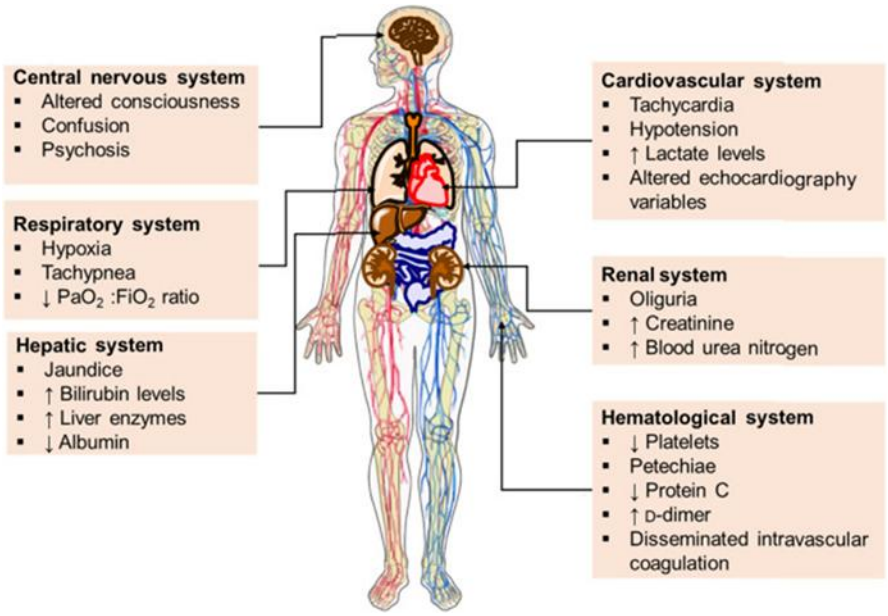

Figure 1: Multisystemic Effects of Shock[16]

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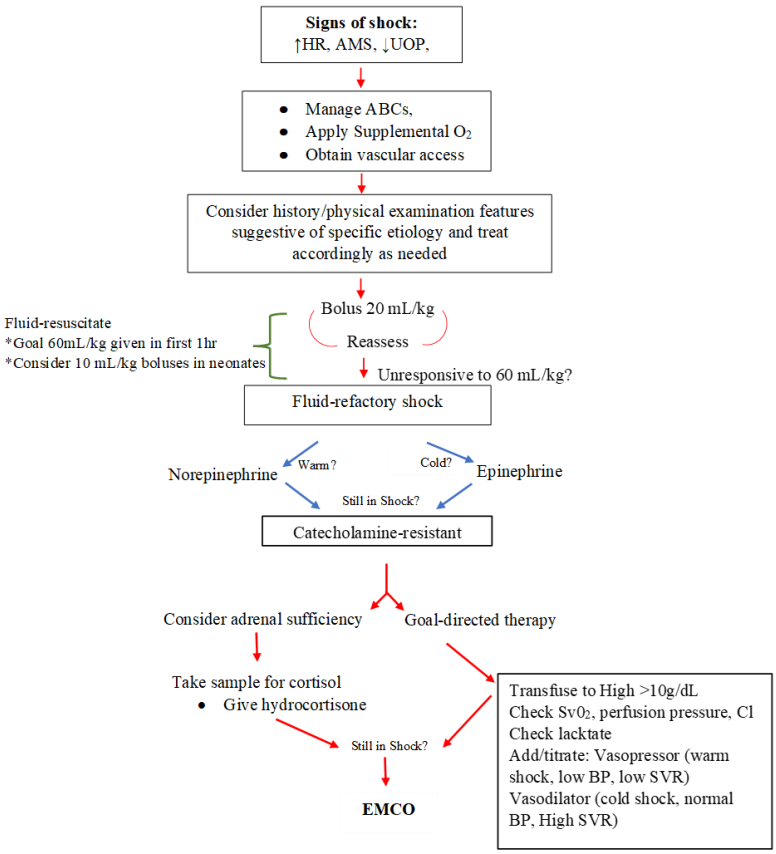

Figure 2: Management Algorithm of Shock [17]