Approach to a neonate with shock

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Introduction:

- Neonatal shock is common clinico-pathological condition during management of sick newborns. Shock is a dynamic and unstable pathophysiologic state characterized by inadequate tissue perfusion.
- Although the effects of inadequate perfusion are reversible initially, prolonged hypoperfusion and tissue hypoxia can disrupt critical biochemical processes, which if not addressed result in cell death, end-organ failure, and, possibly, death.

Definition:

Shock:

- Shock, or circulatory failure, is defined as a physiologic state characterized by tissue hypoxia due to reduced oxygen delivery and/or increased oxygen consumption or inadequate oxygen utilization.Physical manifestations include tissue hypoperfusion (cold extremities, acrocyanosis, and poor capillary refill), hypotension, and metabolic acidosis. Shock is frequently reversible at first, but it must be recognised and treated as soon as possible to avoid irreversible organ dysfunction.

Hypotension:

- Hypotension is commonly defined in older infants and children by a numerical threshold, such as blood pressure (BP) less than the 5th percentile for age. However, using a numerical definition to classify a neonate's blood pressure as "normal" or "abnormally low" is difficult because BP values vary greatly depending on birth weight, gestational age, and postnatal age.As a result, low blood pressure should not be the only criterion for therapeutic action.

Incidence:

- Premature and very low birth weight (VLBW) neonates are the most vulnerable to shock. Within 24 hours of being admitted to the neonatal intensive care unit (NICU),

approximately 20% of VLBW neonates become hypotensive. However, the exact incidence of shock is unknown. Neonatal sepsis and septic shock are the most common causes of septic shock in the NICU; neonatal sepsis induces septic shock in approximately 1% to 5% of cases, with a death rate of around 71%.

Etiology:

Shock can be classified based on the underlying pathogenesis:

1. Hypovolemic,
2. Distributive,
3. Cardiogenic,
4. Obstructive shock,
5. Multifactorial shock

The etiological classification and pathophysiology is discussed in tabulated form in Table 1.

Table 1: Etiological classification and pathophysiology

Type of shock	**Pathophysiology**
Hypovolemic	
Haemorrhagic	▪ Fetomaternal haemorrhage ▪ Severe bleeding (eg, subgaleal hemorrhage, umbilical cord rupture, internal bleeding) ▪ Twin-twin transfusion
Non-Haemorrhagic	▪ Third spacing from acute intestinal injury (eg, volvulus, necrotizing enterocolitis) ▪ Gastrointestinal fluid loss from congenital chloridorrhea ▪ Polyuria due to congenital diabetes insipidus
Distributive	
Sepsis	

Non-Sepsis	▪ Adrenal insufficiency ▪ Hydrops fetalis ▪ Neonatal toxic shock syndrome
Cardiogenic	
Cardiomyopathy	▪ Myocardial ischemia/hypoxemia ▪ Myocarditis ▪ Congenital cardiomyopathy
Arrhythmia	▪ Congenital complete heart block ▪ Tachyarrhythmia (eg, SVT, VT)
CHD	▪ Hypoplastic left heart syndrome ▪ Critical aortic stenosis ▪ Critical coarctation of the aorta ▪ Interrupted aortic arch ▪ Obstructed total anomalous pulmonary venous connection
Obstructive	
Mechanical	▪ Tension pneumothorax ▪ Pericardial tamponade ▪ Constrictive pericarditis
Pulmonary	▪ Severe pulmonary hypertension ▪ Pulmonary embolus
Multifactorial	▪ NEC ▪ Sepsis ▪ Hydrops Fetalis ▪ Pulmonary hypertension

Clinical Manifestations:

Clinical parameters	Interpretation
Vital signs abnormalities	
Abnormal HR	**Tachycardia (HR>180 bpm)** – common but nonspecific **Variable HR** – early sign of shock **Bradycardia (HR<90 bpm)** – terminal finding
Hypotension	**BP <5th percentile for the gestational age and postnatal age** – warrants additional investigations **Low BP+clinical signs of poor perfusion** – Treatment intervention
Abnormal body temperature	**Fever** – presentation of sepsis **Hypothermia** – probability of shock due to underlying sepsis **Core -periphery temperature difference** – presentation of sepsis
Decreased peripheral perfusion	
Cold extremities, acrocyanosis and pallor	Initial signs of decreasing cardiac output
Delayed capillary refill time (CRT)	**>4 seconds -** Isolated, predictive value poor. **>4 seconds with other findings indicative of poor peripheral perfusion** (low BP, weak pulses, cool extremities, and abnormal neurologic signs) **-**suggestive of neonatal shock
Neurologic findings	Lethargy, irritability, poor feeding, and poor tone **– Initial phase of shock** Stupor / Coma **– Advanced phase of shock**
Respiratory findings	**Tachypnoea (RR>60/min) –** Compensatory response to metabolic acidosis **Respiratory distress** – Primary pulmonary disease or cardiorespiratory compromise **Periodic breathing** – Decreased cerebral perfusion or severe metabolic acidosis

	Hypoxemia – Cardiac dysfunction or obstructed blood flow
Other findings	
Renal	**Low urine output** – Low systemic blood flow
Gastrointestinal (GI)	**Poor feeding** – Lethargy **Vomiting** - Decreased GI motility

Table. 2. Clinical manifestations of neonatal shock

Phase	**Compensated**	**Decompensated**	**Irreversible**
Intravascular volume loss	Upto 25%	25-40%	>40%
Heart rate (HR)	Tachycardia	Marked tachycardia	Severe tachycardia, Bradycardia
Peripheral pulses (PP)	Bounding	Feeble	Imperceptible
Blood pressure (BP)	Normal	Hypotension	Severe hypotension
Pulse pressure (PP)	Normal/wide	Low	Remarkably low
Core-peripheral temperature difference	Increased $>2^{O}$	Increased $>5^{O}$	
Urine output (U/O)	Normal/reduced	Oliguria	Anuria
Mentation	Irritable	Lethargic	Coma

Fig. 3. Clinical features associated with 3 phases of shock

Parameters	**Cardiogenic**	**Hypovolemic**	**Septic (Early/late)**
Arterial BP	Low	Low	Low
Central venous pressure (CVP)	High	Low	Normal
Pulse pressure (PP)	Decreased	Decreased	Normal/Decreased
Cardiac output (CO)	Low	Low	High/normal/low
Core to periphery temperature difference	Increased	Increased	Normal/increased

Table. 4 Signs of hypotension and hypoperfusion in different types of shock

Diagnosis of neonatal shock:

- In neonates, the signs and symptoms of shock vary.
- The diagnosis of shock is clinically based on a constellation of clinical, biochemical, and hemodynamic features. These include findings mentioned in Table 1,2 & 3.
- In clinical practise, the reference range blood pressure limitations are defined as blood pressure readings between the 5th (or 10th) and 95th (or 90th) percentiles that are based on gestational age and postnatal age.
- Even if born at 24 to 26 weeks' gestation, most preterm infants' mean blood pressure would be 30 mm Hg by the third day of life.
- As a general rule, the lower limit of normal mean blood pressure in mm Hg on the day of birth is approximately equal to the gestational age in weeks.

Diagnostoc Evaluation:

History:

The newborn history, including review of maternal health issues, antenatal screening, and pregnancy and delivery complications, often can identify the underlying cause of shock.

Table 5. Summary of perinatal history and probable type of shock

History	Probable underlying type of shock
Significant blood loss from placental anomalies, maternal bleeding, or umbilical cord abnormalities. Internal bleeding due to traumatic vacuum-assisted delivery (eg, subgaleal bleed).	Hypovolemic shock
Prolonged rupture of membrane Maternal chorioamnionitis Maternal fever during labor Bacteriuria during the pregnancy Previous delivery of an infant affected by GBS disease Maternal history of herpes genital lesions may be indicative of	Septic shock

shock due to disseminated herpes simplex virus (HSV) infection History of infection in household	
Maternal history of systemic lupus erythematosus or Sjögren syndrome resulting in neonatal heart block. Antenatal asphyxia. Congenital heart disease (CHD) detected by prenatal ultrasound or newborn screening.	Cardiogenic shock
Hydrops fetalis	Distributive shock / multifactorial

Physical findings in neonatal shock:

- Clinical findings have been summarized in table 2, 3 & 4.
- Certain findings may point towards specific etiology.

Table. 6. Clinical findings and specific etiology of shock

Clinical findings	Probable etiology
Pathologic murmur or gallop rhythm	Cardiac cause
Weak or absent lower extremity pulses (particularly in comparison to upper extremity pulses)	Cardiogenic shock due to coarctation of aorta
Chest asymmetry and absent breath sounds on one side	Tension pneumothorax
Abdominal distention	NEC
Disorder of sexual differentiation (DSD)	Adrenal insufficiency
Rash	Congenital infection, sepsis. Neonatal lupus

Laboratory and Imaging tests:

- The following tests may help identify the cause, assess the severity, and guide the initial treatment.

Table. 7. Laboratory tests and interpretation

Lab test	Interpretation
Arterial blood gas (ABG)	Metabolic acidosis – degree of hypoxia or hypoperfusion Respiratory acidosis – Primary pulmonary disease e.g. pneumonia Hypoxemia – depends upon the degree of respiratory compromise and underlying etiology
Serum lactate	lactate levels increase (>4 mmol/L) as the severity of shock increases
CBC	Elevated (>30,000/mm3) and depressed (4000/mm3) total white blood cell (depending upon the age of neonate) counts are associated with systemic bacterial infection Thrombocytopenia – sepsis
Septic screen	I/T ratio >0.2 CRP >2mg/dl PCT > 2ng/ml IL8 >>70 pg/ml PCR 16SrRNA? s TERM-1 >60ng/ml CD64 and combination tests
Blood chemistries	Hypo and hyperglycemia, hyperkalemia, low bicarbonate, deranged renal and liver functions –findings vary based on type and severity of shock
Additional tests	Blood, urine & CSF culture, viral testing, cross match.
Lung ultrasonography	Can be useful for neonates with respiratory distress
Chest radiograph	Chest radiography can be useful for neonates with respiratory distress or an abnormal cardiopulmonary examination

Table. 8. List of parameters used for assessment of shock:

Conventional parameters (commonly used in standard practice)	Capillary refill time Urine output Heart rate Blood pressure Presence of lactic acidosis Central venous pressure (approximates right atrial pressure and can give valuable information regarding the preloading conditions) Mixed venous saturation ScvO2 (Normal 70-75% - Considered as the balance between oxygen demand and delivery and has been used as a determinant for tissue hypoxia) Arterio venous oxygen difference (Normal is 5 ml/100 ml of blood or 25% - excellent estimate of tissue oxygen delivery)
New parameters (now being used in clinical practice)	Functional echocardiography Near infrared spectroscopy
Novel parameters (research tools at this time, not being used in clinical practice)	Electrical cardiometry Visible light spectroscopy Perfusion Index Functional cardiac MRI

Table. 9. Noninvasive bedside tests for assessment of shock

	Functional ECHO (fECHO)	**Other**
Preload	SVC flow IVC pulsatility End diastolic LV and RV volume (filling)	Electronic velocimeter (ICON) stroke volume variation (SVV)
Cardiac function	Systolic function Diastolic function Pulmonary pressures	ICON – SV, STR, CI
Afterload (systemic vascular resistance)		Systolic and diastolic blood pressure ICON
Capillary perfusion		SpO2 Pulsatiliti index (PI) (0.02-20%)
Endorgan perfusion		NIRS – rSO2, FTOE

Treatment of shock:

- It is important to recognize and treat the shock at the earliest.
- VTIPPSS is the mnemonic for the treatment of shock to manage hypoxia, hypoglycemia, hypocalcemia, hypothermia, anemia, electrolyte imbalance, acidosis, and coagulation dysfunction. It is defined as follows:

  V—Ventilation: Oxygen and ventilatory support to support breathing is the cornerstone.

  T—Thermoregulation

  I—Infusion: Infusion of isotonic crystalloid fluid, plasma, and blood is the mainstay of treatment.

  P—Pump/cardiovascular support (inotropes)

  P—Pharmacotherapy (antibiotics/steroids)

  S—Specific therapy

  S—Supportive care

Summary:

- Shock is a dynamic and unstable pathophysiologic state characterized by inadequate tissue perfusion due to reduced oxygen delivery and/or increased oxygen consumption or inadequate oxygen utilization. If untreated it leads to tissue/cellular damage that results in end-organ failure and, in some cases, death.
- The causes of neonatal shock are classified into four pathophysiologic mechanisms. However, neonatal shock may be the result of more than one of these processes (multifactorial shock).
- Regardless of the etiology, neonates with shock typically present with signs of poor perfusion (cool extremities, acrocyanosis, pallor), tachycardia, and metabolic acidosis. Late signs of shock include bradycardia and hypotension.
- Stabilization of the patient's hemodynamic status takes precedence over the diagnostic evaluation, and resuscitation should not be delayed. However, a focused diagnostic evaluation is conducted in concert with resuscitative efforts

Suggested reading:

1. Singh Y, Katheria AC, Vora F. Advances in Diagnosis and Management of Hemodynamic Instability in Neonatal Shock. Front Pediatr. 2018;6:2. Published 2018.
2. Schwarz CE, Dempsey EM. Management of Neonatal Hypotension and Shock. Semin Fetal Neonatal Med. 2020 Oct;25(5):101121.
3. de Boode WP, van der Lee R, Horsberg Eriksen B, Nestaas E, Dempsey E, Singh Y, Austin T, El-Khuffash A; European Special Interest Group 'Neonatologist Performed Echocardiography' (NPE). The role of Neonatologist Performed Echocardiography in the assessment and management of neonatal shock. Pediatr Res. 2018 Jul;84(Suppl 1):57-67. doi: 10.1038/s41390-018-0081-1.