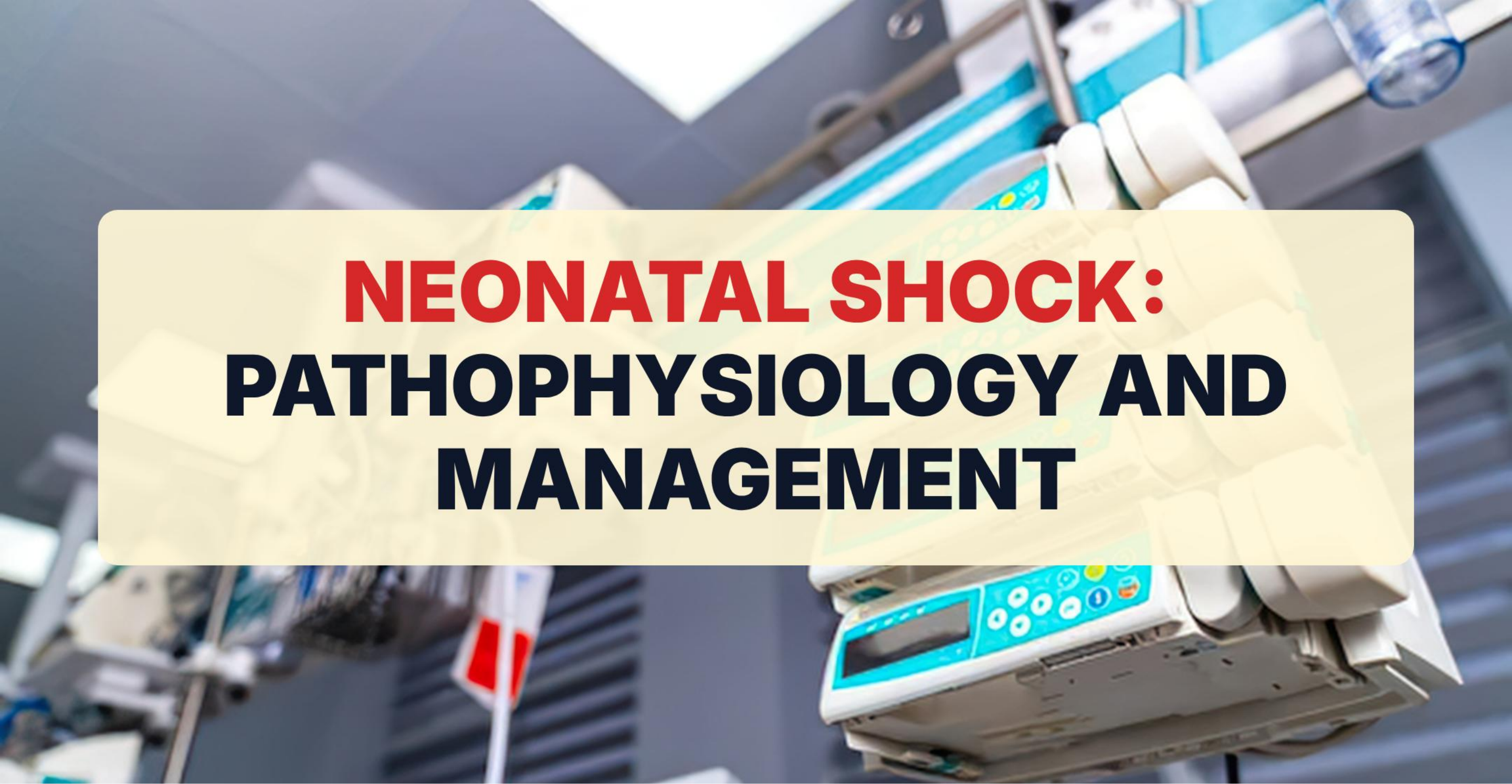




NEONATAL SHOCK: PATHOPHYSIOLOGY AND MANAGEMENT

For Neonatologists

A complete, up-to-date guide to early recognition, advanced pathophysiology, differentiation of hemodynamic phenotypes and targeted management in the NICU.

SOURCES & REFERENCES

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BASIC PATHOPHYSIOLOGY

The Concept of Shock and the DO_2/VO_2 Balance

Acute circulatory failure in which oxygen delivery (DO_2) does not meet tissue metabolic demand/consumption (VO_2).

Immediate consequence: anaerobic metabolism → lactate accumulation → lactic metabolic acidosis → progressive end-organ injury.

FUNDAMENTAL HEMODYNAMIC EQUATIONS:

- $\text{DO}_2 = \text{CO} \times \text{CaO}_2$ (O_2 delivery = cardiac output $\times$ arterial O_2 content)

- $\text{CO} = \text{SV} \times \text{HR}$ (cardiac output = stroke volume $\times$ heart rate)
- $\text{CaO}_2 = (1.34 \times \text{Hb} \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$

CRITICAL DO_2 :

Below critical DO_2 , O_2 consumption becomes directly dependent on delivery. **Hemoglobin (Hb)** and **saturation (SaO_2)** are therapeutic targets as crucial as cardiac output.

Therapeutic goal: restore effective O_2 delivery to the mitochondria, NOT just normalize blood pressure numbers!

What Makes the Neonatal Heart Different

The newborn myocardium has unique properties that shape its hemodynamic response.

1. CO DEPENDS ON HR

- Stroke volume (SV) is small and fixed.
- HR is the main compensatory tool (160-180 bpm).

2. LIMITED RESERVE

- Disorganized myofibrils, fewer sarcomeres.
- Immature sarcoplasmic reticulum: dependent on extracellular Ca^{2+} .

3. AFTERLOAD INTOLERANCE

- Reduced ventricular compliance.
- A rise in afterload can REDUCE cardiac output.

4. ↓ INOTROPIC RESPONSE

- Few β_1 receptors in the myocardium.
- Abundant α_1 receptors in the peripheral vasculature.

CLINICAL ALERT: BLUNTED BOWDITCH EFFECT

Extreme tachycardia (> 180 bpm) shortens diastole too much, reduces ventricular filling and causes **subendocardial ischemia**. The compensatory mechanism becomes the aggressor!

ADULT VS NEONATAL ADAPTATION

Circulatory Transition and Shunts

The physiological changes at birth and the presence of shunts drastically change blood flow.

KEY EVENTS OF THE CIRCULATORY TRANSITION:

1. **Placenta removal:** immediate rise in systemic vascular resistance (SVR).
2. **Lung aeration:** sharp fall in pulmonary vascular resistance (PVR).
3. **Closure of fetal shunts:** functional closure of the foramen ovale (FO) and ductus arteriosus (PDA).

DYNAMICS OF BIDIRECTIONAL SHUNTS:

- The direction of ductal flow depends directly on the **PVR/SVR ratio**.
- The right ventricle (RV) is not yet remodeled and has **low compliance**.
- Positive pressure (mechanical ventilation) acutely changes both preload and afterload.

PRACTICAL IMPLICATION IN THE NICU:

A large left-to-right shunt through the PDA causes **systemic ductal steal**, reducing mesenteric and renal perfusion. A right-to-left shunt occurs when PVR exceeds SVR (pulmonary hypertension).

ETIOLOGICAL CLASSIFICATION

Etiological Phenotypes of Neonatal Shock

Different pathophysiological mechanisms call for different therapeutic strategies.

Type of shock	Main mechanism & common etiology
Distributive	Abnormal vascular tone. Sepsis (most common), adrenal insufficiency, neurogenic.
Hypovolemic	↓ Preload from volume loss. Fetal/maternal hemorrhage, IVH, third-space losses.
Cardiogenic	Myocardial dysfunction (LV/RV). HIE, transitional maladaptation, myocarditis, arrhythmias.
Obstructive	Mechanical obstruction to filling/ejection. Tension pneumothorax, tamponade, duct-dependent lesions.
Dissociative	DO ₂ preserved, but O ₂ carrying/utilization capacity impaired. Severe anemia, methemoglobinemia.
Mixed	Several mechanisms coexist (e.g., sepsis + myocardial dysfunction + PPHN).

SEPTIC SHOCK

Septic Shock: Warm vs. Cold

The clinical presentation in neonates is heterogeneous and may shift between phenotypes.

Feature	WARM SHOCK (↓ SVR)	COLD SHOCK (↑↑ SVR)
Peripheral signs	Warm extremities, rapid ('flash') CRT, bounding pulses, low diastolic BP.	Cold extremities, prolonged CRT (> 3 s), weak pulses, late hypotension.
TnECHO / POCUS	Hyperdynamic; underfilled ventricles; misleadingly high EF/FS.	LV or biventricular systolic dysfunction; associated reactive PH.
Therapeutic target	↑ SVR: vasopressor alone (norepinephrine or vasopressin).	Inotrope / inodilator: epinephrine or dobutamine/milrinone.

KEY POINT: the hemodynamic profile in septic shock is dynamic. Serial reassessment with targeted neonatal echocardiography (TnECHO) is essential to adjust vasoactive drugs.

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Continuous medication

+

Adrenaline (Epinephrine)

▼

IV continuous

1mg/mL

+

Weight (kg)

+

Desired dose (mcg/kg/min)

+

Intended drip (mL/h)

Additional information

Bibliographical references

>

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Continuous medication

+

Dobutamine

▼

IV - central access

IV - peripheral access

12.5mg/mL

+

Weight (kg)

+

Desired dose (mcg/kg/min)

+

Intended drip (mL/h)

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In critical moments, always
have the **right tool** at hand.

≡

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Continuous medication

+

Milrinone

▼

IV continuous

1mg/mL

0.2mg/mL

Loading

Maintenance

+

Weight (kg)

+

Desired dose (mcg/kg)

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≡

neofast

Continuous medication

+

Milrinone

▼

IV continuous

1mg/mL

0.2mg/mL

Loading

Maintenance

+

Weight (kg)

2

+

Desired dose (mcg/kg/min)

0.3

Suggested maintenance dose: 0.3 to 0.75 mcg/kg/min

+

Intended drip (mL/h)

0.2

Milrinone

IV continuous - 1mg/mL

Dose	0.86mL
Dilute with NS , D5W	3.94mL
Final concentration	180mcg/mL
Total in 24 hours	4.8mL
Interval	Continuous by 35h

Fill in to get the results.

Phases of Shock and Cellular Injury

The progression from compensated to irreversible shock and the impact of severe acidosis.

1. COMPENSATED PHASE ('OCCULT SHOCK'):

- Peripheral and splanchnic α_1 vasoconstriction preserves cerebral and coronary flow.
- Compensatory tachycardia; blood pressure kept within normal values.

2. DECOMPENSATED PHASE:

- Failure of compensatory mechanisms → hypotension and oliguria.
- Rising lactate, lactic acidosis and tissue microcirculatory dysfunction.

3. IRREVERSIBLE PHASE:

- Severe cellular and mitochondrial injury → multiple organ failure.
- Complete refractoriness to catecholamines.

IMPACT OF SEVERE METABOLIC ACIDOSIS (PH < 7.10):

- Excess H^+ acts as a potent direct **myocardial depressant**.
- Causes **generalized vasoplegia** and refractoriness to endogenous and exogenous catecholamines.

CLINICAL RECOGNITION

Clinical Picture and Warning Signs

Systematic signs of poor tissue perfusion by organ system.

PERIPHERAL PERFUSION

- CRT > 3 seconds.
- Cold extremities and mottling.
- Central-peripheral temperature gap > 2 °C.
- Weak pulses (or bounding in PDA/warm shock).

CARDIOVASCULAR

- Persistent tachycardia (> 10-15% above baseline).
- Hypotension (late sign!).
- Rule: MAP < gestational age (weeks).
- **Bradycardia = pre-arrest!**

NEURO / RENAL

- Lethargy, hypotonia, weak cry.
- Recurrent apnea.
- Urine output < 1 mL/kg/h (of limited use in the first 48 h).

METABOLIC

- High and rising lactate.
- Base excess (BE) < -7 mmol/L.
- Hypo/hyperglycemia and temperature instability.

DIAGNOSTIC ACCURACY

Accuracy of Clinical Parameters and Tests

Limitations of traditional parameters used alone in the newborn.

Parameter	Diagnostic performance	Limitation / pitfall
CRT $\geq$ 3 s	Sensitivity 55% / specificity 80%	7 in 10 neonates with low SVC flow have a normal CRT!
Blood pressure	Most used method in the NICU	Weak correlation with systemic blood flow. Normotension $\neq$ adequate perfusion!
Lactate	> 5 mmol/L on day 1 (specific)	Clearance (trend) matters more than a single value.
Troponin	Marker of myocardial injury	Serial measurement (admission $\rightarrow$ 24-48 h). Guides coronary protection.
NT-proBNP	Wall stretch (PDA)	Sensitivity 90% / specificity 89% for a hemodynamically significant PDA.

ADVANCED MONITORING

Monitoring Tools in the NICU

Invasive and non-invasive methods of hemodynamic assessment compared.

Method	What it tells you	Advantage / limitation
Invasive BP	Continuous BP; access for blood gases	Gold standard. Risk of thrombosis and damped waveform.
NIRS (rSO ₂)	Tissue oxygenation (brain, kidney, splanchnic)	Continuous (ideal range: 55-85%). Mean and trend.
ScvO ₂	Global O ₂ extraction (DO ₂ /VO ₂ balance)	Target ≥ 70%. Umbilical catheter position changes the value.
TnECHO / POCUS	Preload, contractility, SVR, shunts, PH	Defines the hemodynamic phenotype. Requires training.

BEDSIDE POCUS

POCUS in Shock: the SAFE-R Protocol

A systematic point-of-care ultrasound approach in the neonatal emergency.

- 1. HEART (subcostal):** rule out pericardial effusion and cardiac tamponade (indication for emergency pericardiocentesis).
- 2. LUNG (chest):** look for tension pneumothorax, pleural effusion and severe pulmonary edema (guides drainage/ventilation).
- 3. AORTA (abdominal):** assess pulsatility. Absent pulsatility suggests duct-dependent heart disease (**start PGE1 immediately!**).
- 4. ABDOMEN:** look for free fluid (hemoperitoneum, ascites, cause of hypovolemic shock).
- 5. HEAD (fontanelle):** identify acute intraventricular hemorrhage (IVH) or post-hemorrhagic hydrocephalus.

INFERIOR VENA CAVA (IVC) ASSESSMENT:

Assess size and respiratory collapsibility to **differentiate hypovolemia** from **hypervolemia**/congestion.

TnECHO Parameters in Neonatal Shock

Echocardiographic domains to map the hemodynamic state.

Domain	Parameters assessed	Clinical interpretation
Systemic flow	LVOT VTI × area × HR; SVC flow	Without shunts, LV output reflects systemic flow.
LV function	EF (Simpson), FS (< 28%), GLS, TDI	'Normal' EF/FS is misleading when SVR is low!
Pulmonary pressure	TR jet (RVSP); septal flattening	Right-to-left ductal shunt means PVR > SVR.
Shunts & volume status	PDA (size/direction), PFO, IVC	Maps ductal steal and fluid responsiveness.

TREATMENT: VOLUME

Fluid Management: Indications and Risks

Volume expansion should be treated as a carefully considered drug prescription.

'FLUID IS A DRUG'

- **Clear indication:** true hypovolemia (hemorrhage, losses) or sepsis with low preload confirmed on TnECHO (small LV, reduced VTI, collapsible IVC).
- **Recommended solution:** isotonic crystalloid (0.9% saline) at **10 mL/kg** per bolus.
- **Infusion time:** term infants over 15-30 min; preterm infants over 30-60 min (maximum 10-20 mL/kg in total).

RISKS OF FLUID OVERLOAD IN THE NEONATE:

1. **Immediate pulmonary congestion:** due to low diastolic myocardial compliance.
2. **Increased ductal shunt (PDA):** worsens systemic steal.
3. **Risk of IVH in extremely preterm infants:** rapid boluses cause abrupt swings in cerebral perfusion pressure.
4. **Positive fluid balance:** associated with higher morbidity and mortality in the NICU.

PHARMACOTHERAPY

Vasoactive Drugs and Corticosteroids

Choice of vasoactive drug guided by the newborn's hemodynamic phenotype.

Drug & dose	Main mechanism	Predominant effect & cautions
Dobutamine 5-20 mcg/kg/min	Inotrope / inodilator ($\beta_1 > \beta_2$)	++++ inotropy, ++ vasodilation. Caution: tachycardia and $\uparrow$ O ₂ consumption.
Epinephrine 0.05-1 mcg/kg/min	Inotrope / vasopressor (β_1/β_2 at low doses; α_1 at high doses)	Potent inotrope. High doses cause vasoconstriction. Raises lactate/glucose.
Norepinephrine 0.05-1 mcg/kg/min	Potent vasopressor ($\alpha_1 \gg \beta_1$)	++++ vasoconstriction. Requires corrected volume status. Ideal in warm shock.
Milrinone 0.25-0.75 mcg/kg/min	Inodilator (PDE3 inhibitor)	++++ inotropy/lusitropy. Lowers PVR/SVR. Watch for hypotension.

PHARMACOTHERAPY

Corticosteroids

CORTICOSTEROIDS IN REFRACTORY SHOCK:

Hydrocortisone (1 mg/kg every 6-8 h): indicated in shock refractory to fluids + escalating vasoactive doses due to *relative adrenal insufficiency of prematurity*.

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Medicines

Hydrocortisone

IV bolus or IM

Intermittent IV

100mg Powder

250mg Powder

500mg Powder

Without BSA

BSA (height)

BSA (without height)

Weight (kg)

2

Desired dose (mg/kg/dose)

1

Suggested dose: 0.25 to 0.5 mg/kg/dose

Additional information

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Medicines

Hydrocortisone

Intermittent IV - 100mg Powder

Dose	2mg
Interval	12/12h

Reconstitution

Reconstitute with SWFI or own diluent	2mL
Concentration after reconstitution	50mg/mL

Dilution

Remove from vial	1mL
Dilute the dose in mL with NS , D5W or D10W	49mL
Final concentration after dilution	1mg/mL
Dose after dilution	2mL
Infusion rate	20-30 min

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FINAL SUMMARY

Key Points

- 1. Neonatal myocardium:** depends on HR, needs Ca^{2+} and tolerates afterload poorly.
- 2. Normal BP $\neq$ adequate perfusion:** monitor microcirculation signs and tissue O_2 delivery.
- 3. Lactate and BE:** value the trend over time (clearance), not a single value.
- 4. Hemodynamic phenotyping:** management should be guided by TnECHO / POCUS.
- 5. Fluid is a drug:** 10 mL/kg boluses with careful reassessment (caution in preterm infants).
- 6. Choice of vasoactive drug:** pick it according to the pathophysiology (SVR vs. contractility).
- 7. Refractory shock:** hydrocortisone, TSH/free T4, and re-investigate PDA, PPHN and heart disease.
- 8. Beware of acidosis:** do not use sodium bicarbonate routinely, and do not deplete K^+ during the acidotic phase.

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**Faster
shifts**



Check it out!

**Easier
shifts**

