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# Textbook of Clinical Neonatology

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# 5.51 Neonatal Shock

*Vinay H Joshi, Preetha V Joshi*

*Case study:*

*Case 1: 28 weeks baby, born with history of preterm premature rupture of membrane of 18 hours, with prenatal steroids to mother, birth weight of 810 grams, develops RDS, requiring surfactant therapy. Currently on ventilator, day 4, started having episodes of tachycardia and desaturation. The heart rate (HR) is 174 beats/min, blood pressure (BP) 38/20 mm Hg, and mean arterial pressure (MAP) 26. There is mild pallor, capillary refill time is 3–4 seconds and pulses well felt. The heart sounds are normal, and there is no murmur. Breath sounds are normal. AF is level. Urine output is adequate. Is it shock?*

*Case 2: A 2-day-old, male, term baby with birth weight 3.5 kg, is delivered by emergency lower segment cesarean section (LSCS) for meconium stained liquor. The baby is admitted for respiratory distress and referred on day 3 for worsening. On admission, the infant is febrile, HR 190 beats/min, CRT > 5 sec and pulses weak, respiratory rate (RR) 78 breaths/min, BP 42/28 mm Hg, mean BP 34 mm Hg, oxygen saturation ($SpO_2$) 90% both limb. What next for perfusion/shock?*

## INTRODUCTION

Shock is a state of acute circulatory failure resulting into decreased tissue and organ perfusion leading to depletion of oxygen and substrate in the cells. When this happens, anaerobic metabolism sets in, leading to accumulation of toxic waste products eventually causing cell death.

## PATHOPHYSIOLOGY

Maintenance of adequate tissue perfusion depends on a combination of three major factors:

1. Cardiac output
2. Integrity and maintenance of vasomotor tone of arterial, venous, and capillary beds
3. The ability of the blood to perform its necessary delivery of metabolic substrates and removal of metabolic wastes

Cardiac output is the product of HR and stroke volume. Neonatal cardiac output depends more on HR than stroke volume. Very high (>180/min) and very low (< 80/min) HRs are likely to compromise cardiac output if prolonged. At higher rates, ventricular filling time and end-diastolic volume are diminished, and myocardial oxygen consumption is increased. Because myocardial perfusion occurs during diastole, further increases in HR may produce undesirable cardiac ischemia, leading to ventricular dysfunction.

Stroke volume, the other major determinant of cardiac output, is influenced by preload, afterload, and myocardial contractility.

## CARDIAC OUTPUT

- *Preload*: It is determined by the amount of blood filling the ventricles during diastole; increases in preload increase stroke volume up to a maximum value, beyond which stroke volume falls according to the Frank–Starling law **(Fig. 1)**.

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- *Afterload*: Afterload is the force that the myocardium generates during ejection against systemic and pulmonary vascular resistances (for the left and right ventricles, respectively); reductions in afterload increase stroke volume if other variables remain constant.
- *Contractility*: Contractility is a semiquantitative measure of ventricular function, and an increase in contractility produces an increase in stroke volume, if preload and afterload are unchanged.

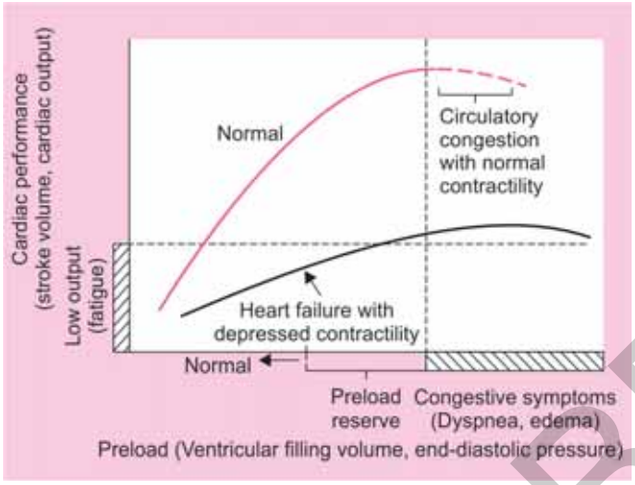


**Fig. 1:** Frank–Starling law.

Clinically significant alterations in preload, afterload, and contractility may be achieved by the use of effective fluid resuscitation and/or vasoactive medications.

The ability of the blood to impart delivery of oxygen and nutrients and to remove metabolic excretory products is largely determined by adequate lung ventilation and perfusion, oxygen-carrying capacity, and oxygen extraction by the tissues.

## ROLE OF BLOOD PRESSURE IN SHOCK

Usually, mean BP, rather than systolic pressure, is used to judge the normality of data obtained from the indwelling arterial line. Based on these data, the statistically defined lower limits of mean BP during the first day of life are approximately numerically similar to the gestational age reference range of the infant. However, most preterm infants, even at 24–26 weeks' gestation, have a mean BP of 30 mm Hg or greater by the third day of life.

Oxygen delivery to the tissues is influenced by cardiac output and blood flow more so than BP; hence, values of BP that are statistically abnormal are not necessarily pathologic. Similarly, hypotension is not synonymous with shock but will be a strong determinant of morbidity in later stages of shock.

*Phases of shock*: Shock is a progressive disorder but can generally be divided into three phases: (1) Compensated, (2) Uncompensated, and (3) Irreversible **(Tables 1 and 2)**.

**TABLE 1:** Types of shock.

| | *Compensated* | *Decompensated/hypotensive* |
|---|---|---|
| Pulse | Tachycardia | Marked tachycardia; can arrest |
| Skin | White, cool, moist | White, "waxy", cold, marked diaphoresis |
| BP | Normal | Hypotension |
| Level of counciousness | Unaltered | Altered, ranging from drowsy to coma |

**TABLE 2:** Etiological classification of shock.

| *Sl. No* | *Types* | *Causes* |
|---|---|---|
| 1. | Cardiogenic | Perinatal asphyxia, congenital heart disease, cardiomyopathy (IDM), heart failure, arrhythmias, or myocardial ischemia, bacterial toxins |
| 2. | Hypovolemic | Acute blood loss—antepartum hemorrhage, umbilical cord accidents, traumatic birth, twin-to-twin transfusion, perioperative, IVH |
| 3. | Distributive | Sepsis, vasodilators, myocardial depression, or endothelial injury |
| 4. | Obstructive | *Inflow obstructions*<br>• *TAPVR*:<br>– Increased intrathoracic pressure caused by high airway pressures or air-leak syndromes (e.g., pneumothorax).<br>• *Outflow obstructions*:<br>– Pulmonary stenosis or atresia/aortic stenosis or atresia<br>– Hypertrophic subaortic stenosis (IDM)<br>– Coarctation of the aorta |
| 5. | Dissociative | Profound anemia or methemoglobinemia |

(IDM: infant of diabetic mother; IVH: intraventricular hemorrhage; TAPVR: total anomalous pulmonary venous return)

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## CLINICAL FEATURES

Clinical manifestations of shock and hypotension include prolonged capillary refill time, tachycardia, mottling of the skin, cool extremities, poorly felt peripheral pulses, and decreased urine output. But none of these alone can be used to diagnose shock. **Table 3** discusses the significance of each parameter.

## DIAGNOSIS AND MANAGEMENT

The following depicts certain clues which can help in diagnosing the type of shock and further management **(Table 4)**.

Essential investigations include complete blood count (CBC), C-reactive protein (CRP), blood culture, blood gas, and markers of organ perfusion such as liver function test (LFT), blood urea nitrogen (BUN), creatinine, coagulation profile based on clinical status. Indicators of hemodynamic improvement include vital, urine output, blood gases, and 2D ECHO parameters.

Echocardiography and Doppler flow velocimetry may provide semiquantitative and semiqualitative noninvasive analysis of myocardial function along with the cardiac anatomy **(Table 5)**. Superior vena cava (SVC) flow in newborn infants has been reported to be a novel marker of systemic blood flow. Low SVC flow (<40 mL/kg/min) has been used to diagnose hypotension and to predict long-term outcome. SVC flow assessment has an advantage of being independent of the normal neonatal shunts [patent foramen ovale and patent ductus arteriosus (PFO and PDA)] in a transitional circulation.

## TREATMENT APPROACH

Initial stabilization and therapy:

- Secure a patent airway and provide supplemental oxygen
- Initiate positive-pressure ventilation as soon as required
- Achieve intravascular or intraosseous access, and infuse 10 mL/kg of crystalloid (to repeat the same

**TABLE 3:** Clinical markers for poor perfusion.

| *Parameters* | *Comments* |
|---|---|
| Color | • Suggests the adequacy of peripheral perfusion and/or oxygenation<br>• Influenced by many factors such as oxygenation, Hb, skin temperature, skin thickness, peripheral perfusion, race, GA, ambient temperature, and light |
| Capillary refill time (CRT) | • The accepted upper normal time is <3 sec<br>• Interobserver and site variations are known<br>• CRT is more reliable when measured on the chest but not the forehead, palm, or heel |
| Central—peripheral temperature difference (CPTd) | • Under normal conditions, CPTd will be <1°C during the 1st postnatal days in ELBW infants<br>• In shock, secondary to poor peripheral perfusion, vasoconstriction will decrease skin temperature and thus lead to an increased CPTd.<br>• CPTd depends largely on body temperature, environmental temperature, and the use of vasoactive drugs |
| Blood pressure | • Hypotension is a late sign of shock<br>• BP is not linearly related to systemic blood flow, since BP is not only determined by CO, but also by SVR |
| Heart rate | • Tachycardia leads to decreased cardiac output and contractility due insufficient filling time<br>• HR can be influenced by many factors, such as body temperature, stress, pain, medication, etc. |
| Urine output (UO) | • Denotes adequacy of renal perfusion<br>• UO is a poor marker of circulatory failure in the absence of a direct relationship with systemic blood flow |

**TABLE 4:** Certain clues for help in diagnosing the type of shock and further management.

| *Parameters* | *Cardiogenic* | *Hypovolemic* | *Early septic (warm)* | *Late septic (cold)* |
|---|---|---|---|---|
| Blood pressure | Low | Low | Low | Low |
| Central venous pressure (CVP) | High | Low | Normal | High |
| Pulse pressure | Decreased | Decreased | Normal | Decreased |
| Systemic vascular resistance | High | High | Low | High |
| Cardiac output | Low | Low | High/Normal | Low |
| Core to peripheral skin temperature difference | Increased | Increased | Normal/decreased | Increased |

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**TABLE 5:** Echo-aided diagnosis.

| *Clinical diagnosis* | *2-D Echo characteristics* |
|---|---|
| Hypotension of prematurity | Structurally and functionally normal heart, +physiological PFO and PDA, reduced SVC flows |
| Persistent pulmonary hypertension of newborn (PPHN) | Structurally normal heart, presence of increased TR and increased PA pressures, systemic to suprasystemic RV pressures, RV dilated and flattening of IVS/bulging of IVS to the left, right-to-left/bidirectional shunt at PFO and PDA level, +/– RV dysfunction |
| Hemodynamically significant PDA (HsPDA) | Structurally and functionally normal heart, large PDA (≥1.8 mm), volume overload of LA and LV, increased LA/aortic root ratio (>1.2), diastolic steal at the postductal descending aorta |
| Septic shock | Structurally normal heart, depressed cardiac function [ejection fraction (EF), shortening fraction (SF)], reduced aortic blood flow, reduced IVC diameter, collapsible IVC |
| Cardiogenic shock | Structurally normal or abnormal heart, depressed cardiac function (reduced EF, SF), paradoxical movement of IVS, distended SVC |
| Perinatal asphyxia | Depressed cardiac contractility, symptoms of PPHN, presence of TR/MR, asphyxial cardiomyopathy |
| Obstructive shock | TAPVC, coarctation of aorta, interrupted aortic arch, critical aortic stenosis, HLHS, pericardial effusion |
| Hypovolemic shock | Structurally and functionally normal heart, underfilled ventricles, reduced IVC diameter, collapsible IVC |

(HLHS: hypoplastic left heart syndrome; IVC: inferior vena cava; IVS: interventricular septum; LA: left atrium; LV: left ventricle; PFO: patent foramen ovale; PDA: patent ductus arteriosus; PA: pulmonary artery; RV: right ventricle; TAPVC: total anomalous pulmonary venous connection; TR: tricuspid regurgitation)

volume if needed) or blood in case of hemorrhagic shock
- First dose of broad spectrum antibiotic to be given as early as possible
- Correction of negative inotropic factors such as hypoxia, acidosis, hypoglycemia, and other metabolic derangements will improve cardiac output.

## Factors Affecting Oxygen Consumption and Delivery (Flowchart 1)

There are three crucial steps in management of neonatal shock:

1. The first step is to fill the vasculature by way of volume expanders.
2. The second is to tighten the vasculature with the use of catecholamines.
3. The final step is to compensate for the immature vasculature with steroids.

## Volume Expanders

Fluid boluses are used as much as possible only when there is physiological evidence of external hemorrhage or fluid loss. An infusion of 10–20 mL/kg isotonic saline solution or Ringer's lactate over 30–60 min is used to treat suspected hypovolemia, if hematocrit is more than 40% and cardiogenic shock is unlikely. In an extreme preterm baby with distributive shock, fluid boluses may lead to opening up of a PDA or in a setup of cardiogenic shock in addition to causing volume overload. Whenever required, crystalloids are preferred over colloids in management of shock.

## Choice of Vasoactive therapy

In this important area of management, there is no clinical outcome-based evidence to decide which inotrope to give which baby to improve short and long-term outcomes of neonatal shock. So the management mentioned in this chapter is more of a recommendation based on best available evidence in neonatal and pediatric literature.

We consider an inotrope when the initial fluid resuscitation is complete and the circulatory function as assessed clinically, biochemically, and echocardiographically (**Table 5**), still remains compromised.

It would be ideal to divide this decision of the right inotrope, etiologically, but we really have no evidence to help us do the same. A lot of centers use echocardiographical parameters to help guide these decisions and monitor the effects of inotropes, in addition to monitoring the clinical and biochemical parameters of circulatory dysfunction.

There are a number of vasoactive drugs used and studied in the neonatal population:

### *Vasopressors*

- *Dopamine*:
  - *Mechanisms of action*: Immediate precursor of epinephrine and norepinephrine. Along with actions on α and β-adrenergic receptors, has effects on $D_1$- and $D_2$-dopaminergic receptors located in the vascular beds of the kidneys, mesentery, and coronary arteries.
  - *Dose*: Infusion rates of 5–10 μg/kg/min increase the stroke volume and cardiac output. Higher infusion rates increase vascular resistance.

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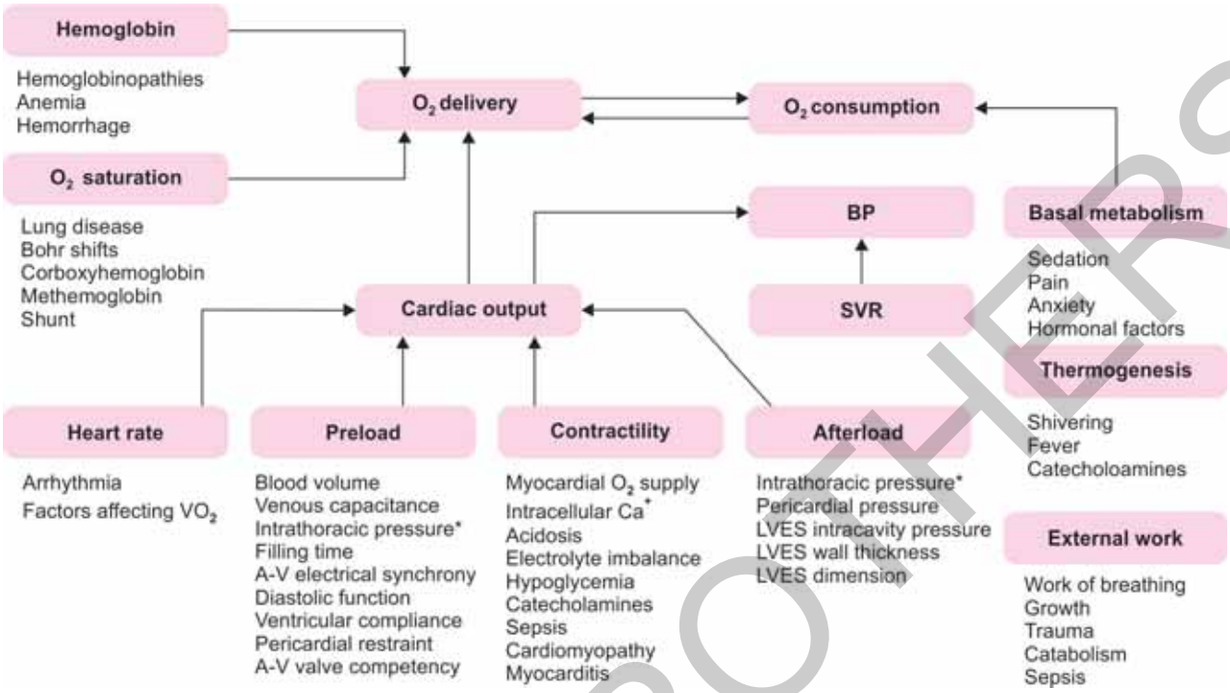
**Flowchart 1:** Factors affecting oxygen consumption and delivery.




- *Indications*: First-line inotrope in most forms of neonatal shock especially septic shock. Also used in cardiogenic shock in congenital heart disease and PDA in preterms.
- *Adverse effects*: Tachycardia and vasoconstriction

▪ *Adrenalin (epinephrine)*:
- *Mechanisms of action*: Endogenously secreted as a hormone by the adrenal medulla synthesized from norepinephrine. $B_1$ agonism increases myocardial contractility, increases myocardial oxygen demand (out of proportion to the inotropic effect), thereby decreasing myocardial efficiency at higher doses. Increases HR and systolic BP. $B_2$ agonism reduces systemic vascular resistance (SVR), and hence, diastolic BP. α-agonism at higher infusion rates, SVR increases, increased pulmonary vascular resistance, hepatic and splanchnic blood flow increases, and renal blood flow reduces.
- *Dose*: 0.05–0.3 µg/kg/min, to be administered through a central line
- *Indications*: Gaining importance in shock and replacing dopamine as the first line of choice especially in septic shock in children and severe PPHN
- *Adverse effects*: Dysrhythmias—atrial and ventricular extrasystoles, extreme tachycardia and hypertension, increases oxygen consumption, severe vasospasm and tissue injury due to $\alpha_1$-agonist (occurs from infiltration into local tissues)

▪ *Norepinephrine (noradrenalin)*:
- *Mechanism of action*: Synthesized from dopamine. It is the principal neurotransmitter of the sympathetic nervous system. It has moderately potent α- and $\beta_1$-agonist. Actions of norepinephrine elevate SVR. Reduced blood flows in the renal, splanchnic, and hepatic vasculature. There is enhanced coronary blood flow due to increased afterload (SVR) which is further augmented by agonism of α-adrenergic receptors of coronary arteries.
- *Dose*: 0.05–0.3 µg/kg/min
- Infusion rate is titrated to the lowest infusion rate that improves perfusion as judged by skin color and temperature, mental status, urine output, and reduction in plasma lactate level.
- *Indications*: It can be used second line after dopamine or/and adrenaline. It is useful in the context of shock with tachycardia as it does not produce significant tachycardia and may lower HR through reflex mechanisms.
- *Adverse effect*: Inappropriate infusion rates may cause compromised organ blood flow. Extravasation leads to skin necrosis.

▪ *Vasopressin*:
- *Mechanism of action*: Vasopressin exerts its hemodynamic effects via the $V_{1a}$ receptor.

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It is an endogenous hormone produced by the hypothalamus, and stored in the posterior pituitary gland for release in response to the following stimuli.

- *Dose*: 0.0001–0.0003 μ/kg/min
- *Indications*: Vasodilatory septic shock—increases mean arterial pressure and SVR without increase in the cardiac index, may be useful in reducing the infusion rates of catecholamines.
- In catecholamine-resistant vasodilatory shock, vasopressin used in combination with norepinephrine causes reduction in the HR, requirement of lesser rates of norepinephrine. Reduces rates of dysrhythmias, and better gastric perfusion, than when norepinephrine is administered alone.
- *Adverse effects*: Elevation of liver enzymes and total bilirubin, decrease in platelet count cardiac arrest in presence of severe refractory shock, ischemic skin lesions of the distal limbs, trunk, or tongue in patients with preexisting peripheral arterial occlusive disease with presence of septic shock, skin necrosis due to extravasation of vasopressin administered through a peripheral venous catheter.

### *Inotropes*

These are drugs with predominant inotropic action.

- *Dobutamine*:
  - *Mechanism of action*: It is a synthetic catecholamine; its molecular structure resembles dopamine. It has mainly inotropic and minor chronotropic and vasopressor activity. It also reduces SVR.
  - *Dose*: 5–20 μ/kg/min
  - *Indications*: Septic shock, sometimes used as an adjunct to dopamine. This is now increasingly been replaced by adrenaline. Also indicate for cardiogenic shock
  - *Adverse effects*: Tachycardia and dysrhythmias
- *Milrinone*:
  - *Mechanism of action*: It acts by selective inhibition of the enzyme phosphodiesterase (PDE) III, thereby increasing levels of cyclic AMP in cardiac and smooth muscle myocytes. It is a nonsympathomimetic inotropic agent, and does not interact with adrenergic receptors. It has positive inotropic and lusitropic effects on the myocardium, and also causes relaxation of vascular smooth muscle.
  - *Dose*: 0.5 μ/kg/min. A loading dose is usually not given.
  - *Indications*: Cardiogenic shock, myocarditis, cold shock once the BP improves and postoperative low cardiac output syndrome (LCOS). It might be also useful in the setting of pulmonary hypertension.
  - *Adverse effects*: Clinically significant thrombocytopenia is rare with milrinone (compared to amrinone).

Currently available inotropes are dobutamine, milrinone, and levosimendan. Data on the use of levosimendan is scarce outside the purview of postoperative neonatal LCOS **(Table 6)**.

**TABLE 6:** Clinical scenarios requiring inotrope and vasopressors.

| *Case* | *Causes* | *Choice of drug* |
|---|---|---|
| Preterm circulatory dysfunction | • Transition from the low resistance placental flow to the high resistance systemic blood vessels<br>• The preterm myocardium has lesser contractile elements and therefore cannot hold out against the increased afterload<br>• Presence of large shunts like the PDA<br>• Need for positive pressure ventilation and the ensuing cardiorespiratory interactions<br>• Loss of autoregulatory function<br>• Relative adrenocortical insufficiency | Dopamine, dobutamine (based on SVC flow), and adrenalin |
| Hypovolemia | Intrapartum hemorrhage | Packed red cell transfusion |
| | Sepsis as a result of third space losses | Fluids, dopamine, adrenaline, noradrenaline, and vasopressin |
| Patent ductus arteriosus (Clinically significant) | Left ventricular dysfunction | Dopamine, milrinone, and adrenaline |
| Severe PPHN | Right ventricular dysfunction | Adrenaline, dopamine, milrinone (once the BP stabilizes), and vasopressin (emerging evidence) |
| *Septic shock*: Follow the protocol for term **(Flowchart 2)** and preterm **(Flowchart 3)** babies | | |

(SVC: superior vena cava; PDA: patent ductus arteriosus)

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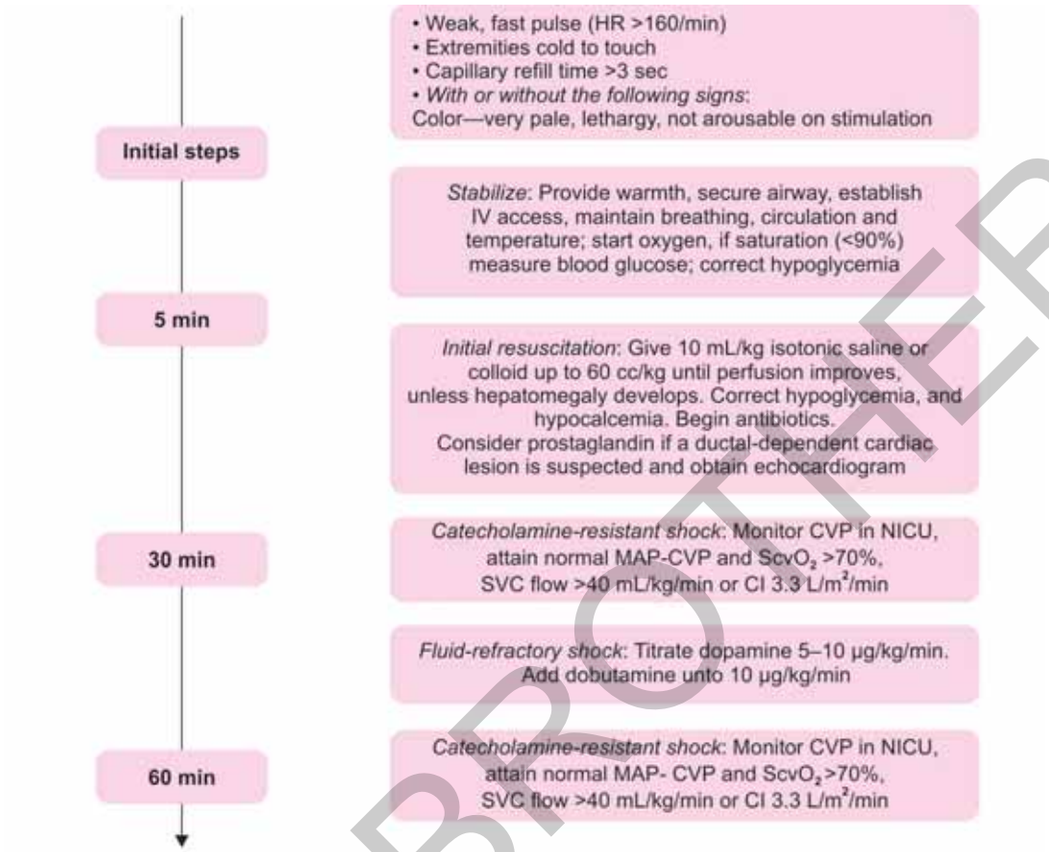
**Flowchart 2:** Management of term septic shock.




| *Cold shock with normal BP and evidence of poor LV function* | *Cold shock with low BP and evidence of RV dysfunction* | *Warm shock with low BP* |
|---|---|---|
| If $ScvO_2$ <70%, SVC flow <40 mL/kg/min or CI <3.3 L/$m^2$/min, add vasodilator (nitrovasodilators, milrinone with volume loading) | If PPHN with $SvcO_2$ <70% SVC flow <40 mL/kg/min or CI <3.3 L/$m^2$/min, add inhaled nitric oxide, consider milrinone, consider inhaled iloprost or IV adenosine | Add volume and norepinephrine, Consider vasopressin, terlipressin, or angiotensin. Use inotrope to keep $ScvO_2$ >70%, SVC flow >40 mL/kg/min and CI 3.3 L/$m^2$/min |

*Refractory shock*: Rule out and correct pericardial effusion, pneumothorax. Use hydrocortisone for absolute adrenal insufficiency and T3 for hypothyroidism. Consider closing PDA if hemodynamically significant.

### Steroids in Shock

Use of steroids should be limited in catecholamine-resistant shock states. Hydrocortisone, the synthetic version of cortisol, compensates for the neonate's relative state of adrenal insufficiency. Hydrocortisone dosage for refractory hypotension is 1–2 mg/kg/dose. If efficacy is noted, the dose can be repeated Q8–12h for 2–3 days, especially if low serum cortisol levels are documented before hydrocortisone treatment.

While these findings look promising, a recent Cochrane review was unable to comment on the safety and efficacy of steroid administration in the hypotensive neonate because of a lack of associated research.

### Supportive Therapy

Maintain normothermia and euglycemia. Avoid dyselectrolytemia early total parenteral nutrition and early enteral feeding when BP and perfusion is optimized **(Box 1)**.

## OUTCOMES

The outcome of shock with multiorgan dysfunction in the newborns is poor. One of the studies showed only 28% of the extremely low birth weight (ELBW) babies with septic shock survived free of disability at 18 months of age. The various factors contributing to the mortality and/or severe disability are refractory shock, acute renal failure,

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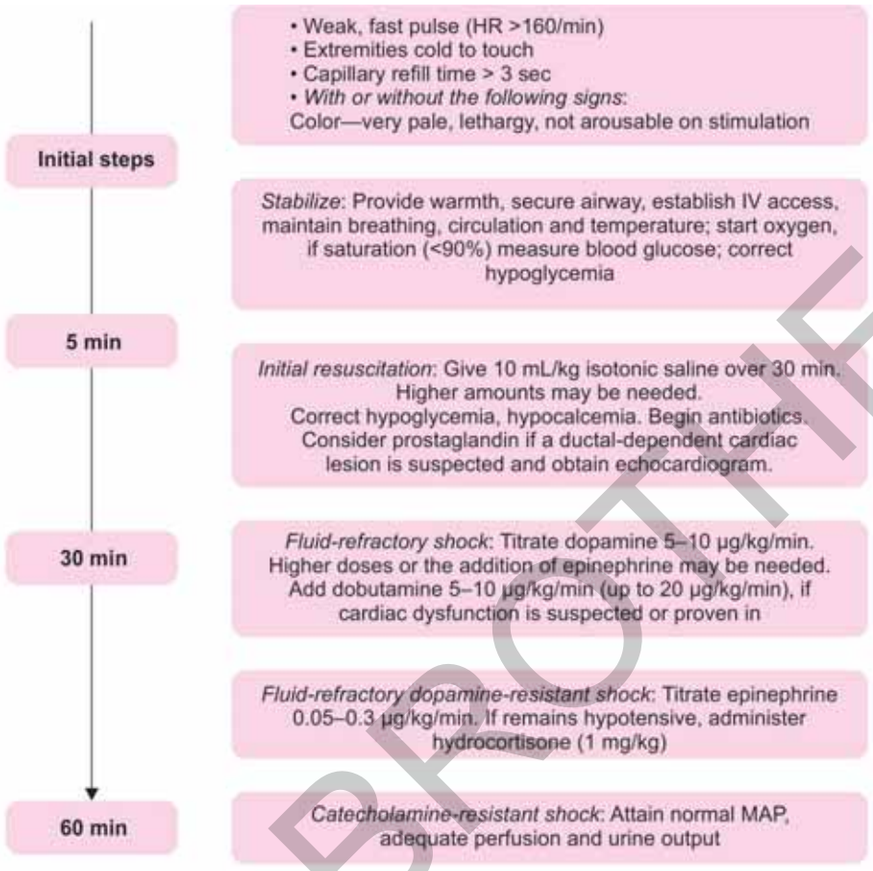
**Flowchart 3:** Management of preterm septic shock.




| *Cold shock with normal BP and evidence of poor LV function* | *Cold shock with low BP and evidence of RV dysfunction* | *Warm shock with low BP* |
|---|---|---|
| Consider milrinone (if normal renal function) | If PPHN, add inhaled nitric oxide, consider milrinone (if normal renal function) | Consider vasopressin or terlipressin in conjunction with inotropes |

*Refractory shock*: Rule out and correct pericardial effusion, pneumothorax, consider IVIg and closing PDA if hemodynamically significant. Consider inhaled nitric oxide with persistent hypoxemia and PPHN physiology.

**BOX 1:** Therapeutic targets in the management of neonatal shock.

- Normal HR
- Capillary refill time < 2 sec
- Warm extremities
- Normal sensorium, alertness
- Normal CVP 8–12 cm and ventilated 12–15 cm
- Normal blood pressure for gestational age
- Urine output >1 mL/kg/h
- Serum lactate (<1.2 mmol/L)
- Normal base deficit
- $ScvO_2$ (central venous $O_2$ saturation) ≥ 70

(CVP: central venous pressure; HR: heart rate; $ScvO_2$: central venous oxygen saturation)

neutropenia, deranged coagulation profile, and metabolic acidosis. Severe necrotizing enterocolitis (NEC) requiring surgical intervention, complicating shock, was associated with adverse neurological outcome.

However, early detection, close monitoring, and prompt management have managed to change these outcomes with good neurodevelopmental outcomes.

## KEY MESSAGES

- A high index of suspicion is needed to diagnose shock.
- None of the clinical markers in isolation are diagnostic of shock. More the markers abnormal more are the chances of compensated shock.

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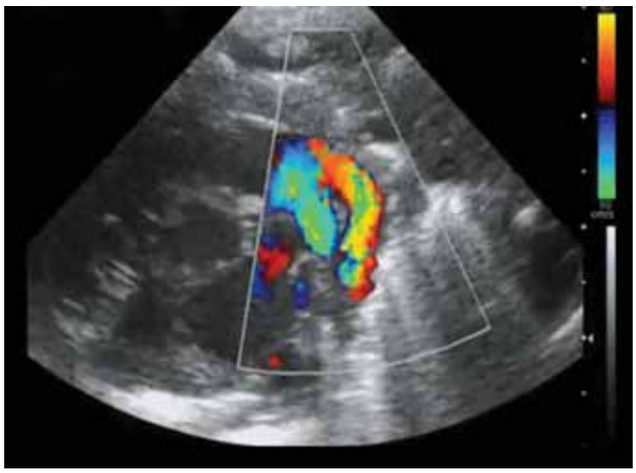

**Fig. 2:** 2D ECHO: Structurally normal heart; large PDA (3 mm); LA/LV dilated.

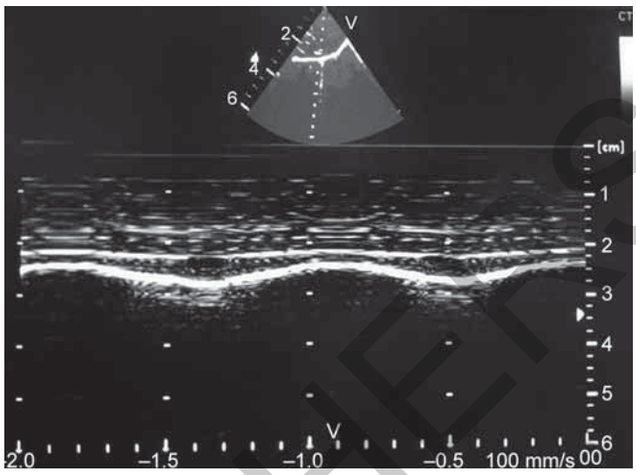


**Fig. 3:** Collapsed inferior vena cava.

- Blood pressure falls late in the course of shock. It may take hours for a compensated shock to develop into hypotensive shock but it may take minutes for a hypotensive shock to end in cardiopulmonary arrest.
- The three key domains of shock management are ensuring adequate intravascular volume, titrating inotropes, and treating the underlying cause.
- Meticulous, ongoing clinical monitoring is essential for improving outcomes.
- Point-of-care ultrasound for heart in clinical corelation helps titrate shock therapy.

## CASE RESOLUTION

*Case 1*: 2-D ECHO study was done **(Fig. 2)**.

*Actions*:

- Fluid optimization (restriction)
- Optimization of ventilation (HFOV)
- IV LASIX
- IV indomethacin (3-day course) started with ECHO monitoring

*Case 2*:

- Septic screen positive on admission. Antibiotics were started after taking blood culture
- Functional ECHO study was done.

Inferior vena cava collapse sign of decreased preload; NS bolus was given **(Fig. 3)**.

*Poor contractility*: IV adrenaline infusion was started at 0.05 µ/kg/min **(Fig. 4)**.

*For afterload reduction:* Milrinone was started after BP stabilized.

Subsequently, there was improved BP, vitals over 2 days. Blood culture showed gram negative organism

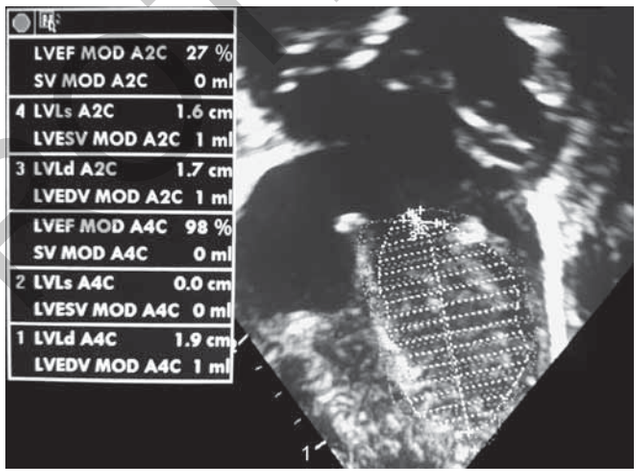


**Fig. 4:** Poor cardiac contractility.

sensitive to ongoing antibiotics. Inotropes were weaned and baby achieveed full feeds by day 6.

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# 5.52 Cardiac Ultrasound in Neonates

*Rema Nagpal, Pradeep Suryawanshi*

## INTRODUCTION

Bedside or "point-of-care" ultrasonography (POCUS) in neonatology is a bedside, real time, focused, serially performed imaging modality, which can be used to understand the underlying physiology of the disease process, target treatment, and monitor responses. It has widely gained acceptance in India as a noninvasive and affordable imaging modality, and is increasingly being used by neonatal physicians in their day-to-day clinical management in the neonatal intensive care units (NICUs).

Point-of-care ultrasonography now has a burgeoning number of uses besides cardiac US, which include lung ultrasound to determine the causes of respiratory distress, abdominal ultrasound, particularly for bowel wall viability, role in central line placements, endotracheal tube localization, and lumbar punctures. Besides, neonatal sonography also aids in on-going day-to-day management of various disease processes such as sepsis, and shock.

Experts suggest that good physical examination skills are valuable, cost effective and an essential component of patient care, but US has been found to be superior to physical examination. It however, remains an additional tool only, and cannot replace a systematic physical examination.

## ULTRASOUND PROBE SELECTION

Transducer characteristics, such as frequency and shape, determine ultrasound image quality. Selection of the transducer "frequency" determines image resolution. Superficial structures require higher frequency probes which have better resolution, but poor penetration. Deeper structures require a lower frequency probe, which has better penetration. Modern transducers are broadband, and can operate in a range of frequencies.

The footprint of the probe should be small enough to fit into the acoustic window. Transducer use is determined by the size of the acoustic window. Probes such as linear

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# Textbook of Clinical Neonatology

## *Under the aegis of IAP-NNF President's Action Plan, 2020*

**Objective**

The book intends to help understand theory and practice of newborn care by the primary care providers. It hopes to instill, stimulate and facilitate the science and art of newborn care with a humane and compassionate approach. We sincerely look forward:

- To appraise the reader to a diverse range of neonatal topics of clinical or practical importance
- To provide point of care, rapid and easy access learning material to clinical disorders and problem situations in caring newborns
- To provide lucid, comprehensive view with clinical pearls and pitfalls for whole spectrum of neonatal care.

**Highlights**

- Clinical approach to relevant practical topics
- Concise, precise and comprehensive guide to neonatal medicine
- Standardized format with case studies, diagnostic and therapeutic options and use of figures, diagrams, tables, flowcharts, pictures and illustrations translating into high-yield visual impact
- Clinical pearls and pitfalls on every topic
- Contains seven distinct Sections addressing; On Call Problems, Perinatal Issues, Care of the Normal Newborn, Infant Born to High-risk Mother, Care of the Sick Newborn, miscellaneous but clinically relevant topics, and Rapid Reference
- Eminent National Reviewer Board
- Keeps the learner engaged and alert by making reading easier and enjoyable
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