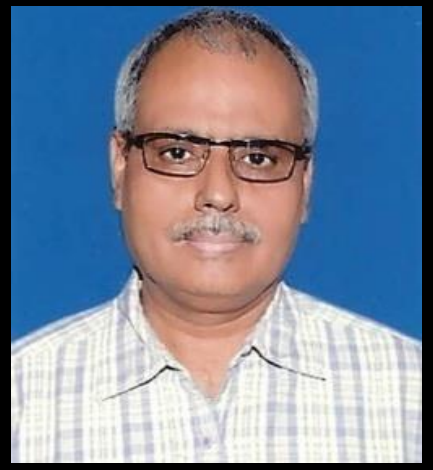
NEONATAL SHOCK



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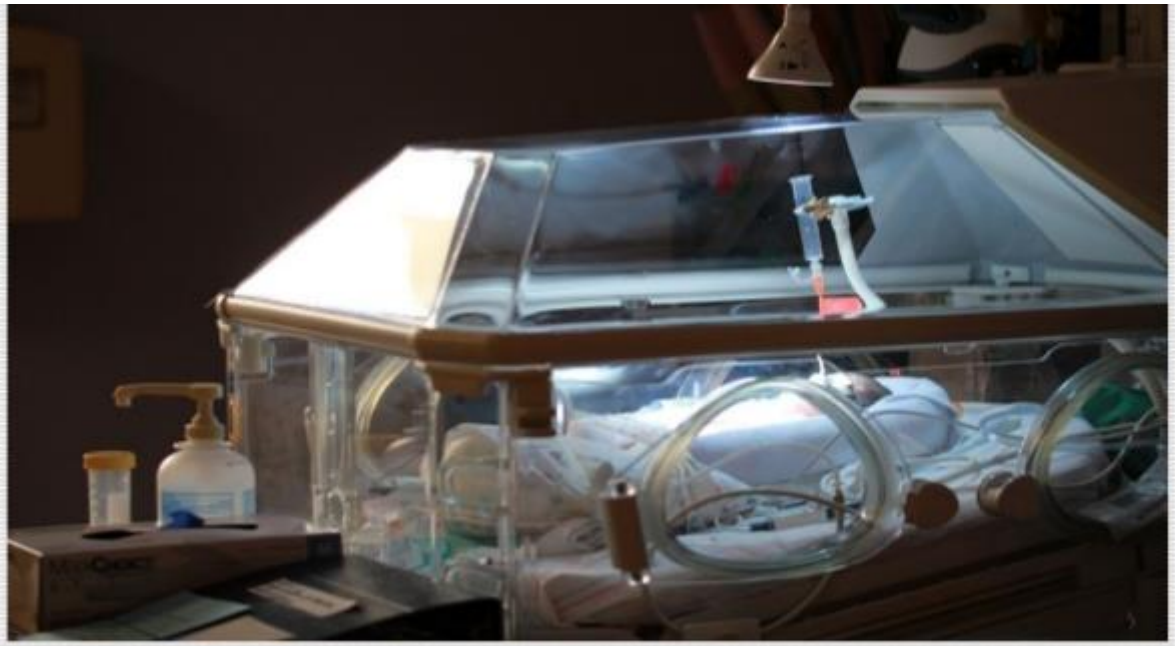

- Acute circulatory dysfunction resulting in inadequate supply of oxygen to the tissues to meet the metabolic demand.

- Cause of neonatal mortality and morbidity.

- Prognosis depends on the duration and severity of shock and the resultant extent of vital organ damage.

ETIOLOGY

- **HYPOVOLEMIC SHOCK**
  - Reduction in circulating blood volume resulting in decrease preload .
- Placental hemorrhage.
- Fetal to maternal hemorrhage.
- Twin to twin transfusion.
- Intracranial hemorrhage.
- Blood loss due to DIC

- **CARDIOGENIC SHOCK**
  - Myocardial pump failure.
- Asphyxia , sepsis, congenital cardiac defect.
- Large PDA in premature infant.
- Bacterial or viral myocarditis.
- Fetal and neonatal arrhythmia compromising cardiac output.
- Metabolic abnormalities – hypoglycemia or cardiomyopathy seen in infant of diabetic mother.

- **DISTRIBUTIVE SHOCK**
  - Impaired peripheral vasomotor tone.
- Sepsis.
- Anaphylaxis.
- Neurological injury – HIE.

- **OBSTRUCTIVE SHOCK**
  - Increase after load.
- Obstruction in inflow tract – TAPVC, Tricuspid atresia, mitral atresia, pneumothorax.
- Obstruction in outflow tract – pulmonary stenosis, coarctation of aorta, aortic stenosis.

- **SEPTIC SHOCK.**
- Combination of hypovolemic , distributive and cardiogenic shock.
- Fulminant sepsis.

PATHOPHYSIOLOGY

- Shock has three distinct but overlapping phases with unique pathophysiology change-

- **COMPENSATED PHASE.**
- Last for hours.
- Redistribution of blood flow to vital organs(heart, brain, adrenal) .
- Away from the non vital organs skin, gut, kidney.
- Blood pressure is preserved.
- Clinical finding-
- Cold, pale skin with delayed CRT.

- Tachycardia - maintain cardiac output.
- Weak peripheral pulse, narrow pulse pressure.
- Ileus.
- Oliguria.

- **DECOMPENSATED PHASE.**
- If compensated phase is not recognized and treated , intrinsic neuro hormonal mechanisms would fail and decompensated phase ensue.

- Hypotension.
- Change in consciousness.
- Risk of cardiac arrest.
- In premature infant – intraventricular hemorrhage.
  cerebral hemorrhage.
  periventricular leukomalacia
- **IRRIVERSIBLE PHASE.**
- Tissue hypoxia.
- Multiorgan failure.

- **CLINICAL FEATURE.**
- Sign of poor perfusion.
- Cool extremities, acrocyanosis, pallor.
- Prolonged capillary refill time.
- CRT>3 sec.
- Change in heart rate.
- Tachycardia – early sign of neonatal shock.
- bradycardia- pre terminal finding.

- **Neurological sign**
- Lethargy, irritability.
- Generalized Hypotonia.

- **Oliguria**.
- Urine output <1ml/kg/hr.

- **Hypotension** – late sign of shock.
- Mean BP in mm of Hg < gestational age in week.

- **INVESTIGATIONS**
- CBC.
- Coagulation parameter –PT , aPTT
- Electrolyte and blood sugar.
- KFT – blood urea, s .creatinine.
- Chest X ray.
- Echocardiography.
- Cranial USG.
- ABG.
- Serum lactate.
- Culture.

- **Functional echocardiography.**
- Used to assess cardiac function.
- SVC flow – surrogate marker for systemic cardiac output.
- SVC flow <50ml/kg/min and RVO <150ml/kg/min - lower limit in neonate for initiation of therapy.
- Preload – IVC collapsibility (in spontaneously breathing babies).

  IVC variability (in ventilated babies).
- Cardiac contractility- fractional shortening.

TREATMENT

- Treatment for shock involves addressing the underlying etiology and managing its cardiovascular systemic effects.
- **Fluid therapy –**
- 10-20ml/kg fluid (NS) – Hypovolemic shock.
- Fluid bolus over 10-20min in septic shock but in case of cardiogenic shock slow bolus over 1hr.
- **Anemia** – PRBC transfusion.
- **DIC-** FFP.

- Central venous pressure measurement may help in management.
- Measured – catheter with its tip in the right atrium or in the intrathoracic superior vena cava.
- Maintain CVP at 5-8 mmHg – improved cardiac output.
- If CVP exceeds 5-8mmHg – additional volume replacement will not be helpful.

- **Supportive management.**
- Maintain airway and breathing.
- Umbilical venous catheterization.
- correction of metabolic derangements like hypoglycemia, hypocalcemia, hypokalemia.
- Antibiotics.
- Blood product transfusion.

THERAPEUTIC ENDPOINTS

- Normal pulses.
- CRT<2sec.
- Warm extremities.
- Normal mental status.
- Normal BP.
- Urine output >1ml/kg/hr.
- Decrease serum lactate.

- **Fluid refractory shock ?**
- Infuse dopamine 10mcg/kg/min and /or
- Dobutamine 10-20 mcg/kg/min.(cardiogenic shock).

- **Dobutamine.**
- Cardioselective inotrope.
- Increase cardiac output with little effect on heart rate.
- Decrease SVR.
- Caution – may cause hypotension

- **Dopamine**
- Dose dependent stimulation.
- Lower dose(2-5mcg/kg/min) – renal and splanchnic vessel dilation through dopaminergic receptor.
- Moderate dose (5-10mg/kg/min) – augmented cardiac contractility.
- Higher dose(10-20mcg/kg/min) – elevate peripheral vascular resistance.
- Caution – PPHN.

- **Fluid refractory-dopamine resistant shock?**
- **Start epinephrine.**
- Dose – 0.05-0.3mcg/kg/min.
- Inotropic and chronotropic.
- At these dose , it has greater beta2 adrenergic effects in peripheral vasculature.
- **Norepinephrine**
- Dose – 0.1- 0.3mcg/kg/min.
- Potent nonselective alpha agonist with some effect at beta1 receptor.
- Dopamine resistant shock.
- Preferred agent in shock associated with SVR.

- **Catecholamine resistant shock?**
- **Milrinone**
- Phosphodiesterase 3 inhibitor.
- It enhances myocardial contractility without raising myocardial oxygen consumption or increasing afterload.
- Decreases vascular tone in the systemic and pulmonary vascular beds.
- Dose o.5-1 mcg/kg/min.
- Useful for treating low cardiac output state after corrective surgery for congenital defects.
- Adverse effect – hypotension , thrombocytopenia.

- **Vasopressin .**
- Catecholamine resistant shock.
- Inhibitory action on NO induced increased in the second messanger cGMP, a potent vasodilatory signal that predominates in the sepsis .
- Dose – 0.0002- 0.006mcg/kg/min.
- It is not routinely used to treat shock in infants but may be a therapeutic option .
- Caution in presence of myocardial dysfunction.

- **Hydrocortisone**
- Upregulation of adrenergic , angiotensin 2 receptors .
- Inhibition of expression of inducible nitric oxide synthetase.
- Inhibition of catecholamine metabolism.
- Increase intracellular calcium concentration.
- Dose – 1-2mg/kg/dose every 8hrly for 2-3days.

- **REFRACTORY SHOCK**
- Evaluate for pneumothorax and pericardial effusion.
- Hypothroidism – T3.
- In case of suspected duct dependent lesion – PGE1 infusion .

TYPICAL CLINICAL SCENARIOS

- **VLBW neonate in the immediate postnatal period**
- **Physiology-** poor vasomotor tone.
  immature myocardium.
  dysregulation NO production.
- **Recommended therapy.**
- Judicious use of volume if hypovolemia is suspected.
- Large volume infusion – increased risk of BPD and intraventicular hemorrhage.
- Dopamine infusion.
- Hydrocortisone – dopamine resistant.

- **Perinatal depression in preterm or full term neonate**
- **phyiology** –
- release of endogenous catecholamine leading to normal or increase SVR .
- Myocardial dysfunction.
- Pulmonary hypertension.
- Clinically- pallor , mottled appearance, poor perfusion.
- The baby is likely to be Euvolemic.

- **Recommended therapy.**
- Dopamine with or without dobutamine.
- Milrinone –
  - decrease afterload.
  - effect without risk of myocardial injury.

- **Septic shock.**
- **Physiology**
  - Hypovolemia.
  - Myocardial dysfunction.
  - Peripheral vasodilation.
  - Increase pulmonary pressure.

- **Therapy**
  - Volume resuscitation with crystalloid.
  - Dose 10-30ml/kg and should be repeated as needed.
  - Dopamine .
  - Epinephrine

- Consider ECMO in infant >34weeks gestation if they donot respond to these intervebtions.

- **Preterm neonate with PDA**

- **Physiology**
  - Ductal steal compromising vital organ perfusion.
  - Increased in left to right shunt with increased risk of pulmonary hemorrhage.

- **Therapy**
- Avoid high dose dopamine >10mcg/kg/min.
  - Increase left to right shunting
  - Reduce vital organ perfusion.

- Dobutamine
  - Enhance cardiac inotropy.

- **Target ventilation management**
  - Increase PVR by increasing PEEP.
  - Maintaining permissive hypercarbia.
  - Avoiding hyperoxygenation.

- **CONCLUSION:**
- The impact of neonatal shock on morbidity and mortality rate is high, but some promising strategies have been evaluated and developed over the years which has improve the current management trends and increase survival rates.


THANK YOU
WELLS FARGO
JDRF 2005
We're on Track For a Cure