INTRODUCTION

Asphyxia neonatorum *is the leading cause of neonatal mortality and morbidity. It is also an important cause of developmental delay and neurological problems both in term and preterm infant.*

It is ***respiratory failure*** *in the new-born, a condition caused by the inadequate intake of oxygen before, during, or just after birth.*

DEFINITION

Birth asphyxia *is defined as a reduction of* ***oxygen*** *delivery and an accumulation of* ***carbon dioxide*** *owing to cessation of blood supply to the fetus around the time of birth.*

Or

Birth asphyxia *is the nonestablishment of satisfactory* ***pulmonary respiration*** *at birth. It is failure of initiation and maintenance of spontaneous respiration with* ***hypoventilation, anaerobic glycolysis*** *and* ***lactic acidosis***

- *It is characterized by progressive **hypoxia, hypercapnia, hypoperfusion** and **metabolic acidosis**.*
- *It may result in **multiorgan system dysfunction** including **hypoxic ischemic encephalopathy** and **long term neuromotor sequelae.***
- *National Neonatology Forum of India has suggested that **birth asphyxia** should be diagnosed when the baby has **gasping** and*

PERINATAL ASPHYXIA

***Perinatal asphyxia**, neonatal asphyxia, or birth asphyxia is the medical condition resulting from deprivation of oxygen to a newborn infant that causes physical harm, mainly to the brain.*

*The **Perinatal Asphyxia** may be defined as hypoxic insult to the fetus severe enough to cause metabolic acidosis, neonatal encephalopathy, and multiorgan system dysfunction.*

ESSENTIAL CRITERIA FOR PERINATAL ASPHYXIA AAP AND ACOG

- *Profound metabolic or mixed acidemia (pH< 7.00) in umbilical cord blood*
- *Persistence of low Apgar scores less than 3 for more than 5 minutes*
- *Signs of neonatal neurologic dysfunction (e.g., seizures, encephalopathy, tone abnormalities)*
- *Evidence of multiple organ involvement (such as that of kidneys, lungs, liver, heart and intestine).*

ETIOLOGY

Pathologically, any factors which interfere with the circulation between maternal and fetal blood exchange could result in the happens of perinatal asphyxia.

These factors can be maternal factor, delivery factor and fetal factor.

ETIOLOGY—HIGH RISK FACTORS

Maternal factor:

- *Hypoxia*
- *Anemia*
- *Diabetes*
- *Hypertension*
- *Smoking*
- *Nephritis*
- *Heart disease*
- *Too old or too young*

Delivery condition:

- *Abruption of placenta*
- *Placenta Previa*
- *Prolapsed cord*
- *Premature rupture of membranes*

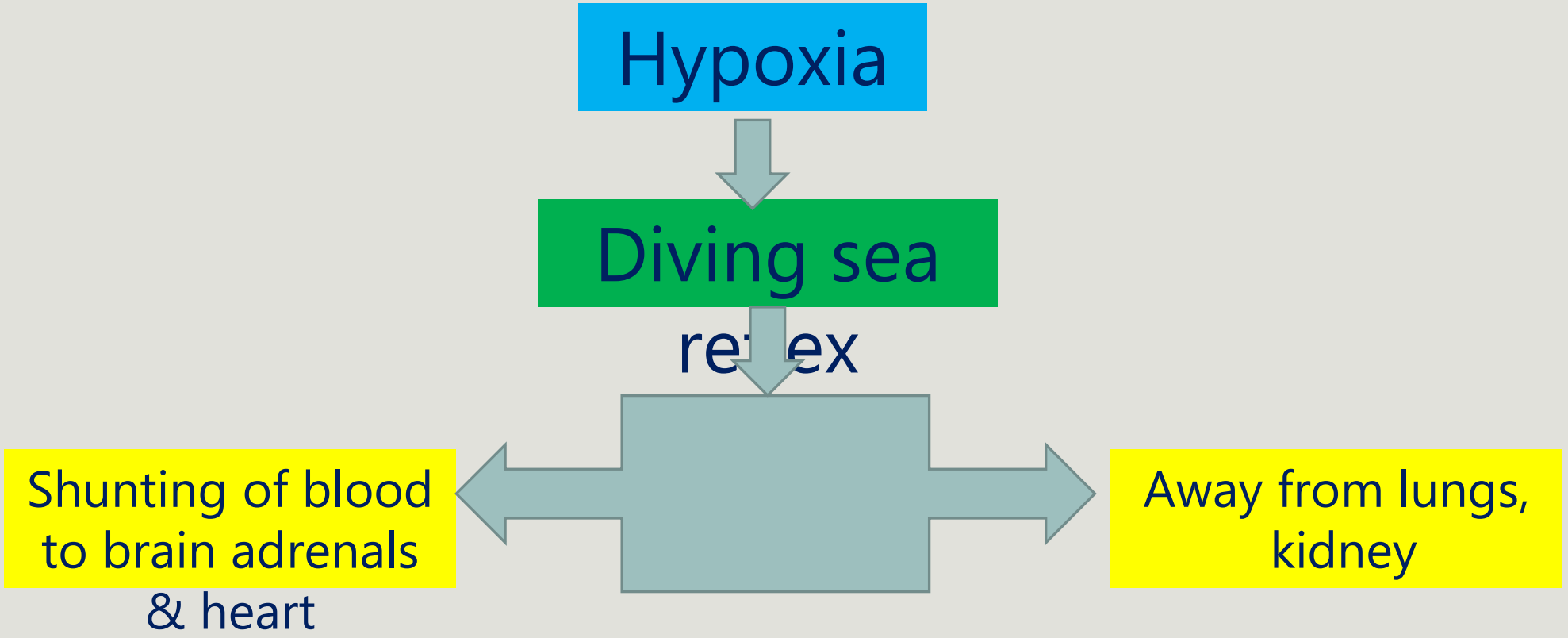
Fetal factor:

- *Multiple birth*
- *congenital or malformed fetus*

PATHOPHYSIOLOGY

When fetal asphyxia happens, the body will show a self-defended mechanism which redistribute blood flow to different organs called "inter-organs shunt" in order to prevent some important organs including brain, heart and adrenal from hypoxic damage.

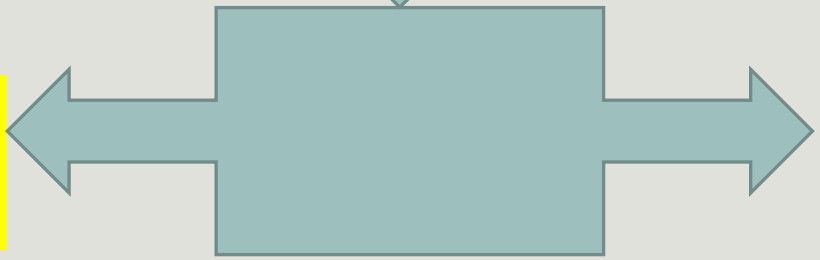
PATHOPHYSIOLOGY



PATHOPHYSIOLOGY

Asphyxia continues

Shunting within the brain



Anterior Circulation Suffers

Posterior Circulation Maintained

CEREBRAL CORTICAL LESIONS

PATHOPHYSIOLOGY

Hypoxia – ABRUPT & SEVERE

No time for compensation

THALAMUS & BRAIN STEM INJURY, CORTEX SPARED

PATHOPHYSIOLOGY (I)

Hypoxic cellular damages:

a. ***Reversible damage(early stage):*** *Hypoxia may decrease the production of ATP, and result in the cellular functions . But these change can be reversible if hypoxia is reversed in short time.*

b. ***Irreversible damage:*** *If hypoxia exist in long time enough, the cellular damage will become irreversible that means even if hypoxia disappear but the cellular damages are not recovers. In other words, the complications will*

PATHOPHYSIOLOGY (II)

Asphyxia development:

a. Primary apnea:

- Breathing stops but normal muscular tone or hypertonia, tachycardia (quick heart rate), and hypertension .
- Happens early and shortly, self-defended mechanism

PATHOPHYSIOLOGY (II)

b. Secondary apnea

Features of severe asphyxia or unsuccessful resuscitation, usually result in damage of organs function.

CLINICAL MANIFESTATION

- **Fetal asphyxia**
  - Fetal heart rate: Tachycardia → Bradycardia
  - Fetal movement: Increase → Decrease
  - Amniotic fluid: Meconium-stained

ASSESSMENT

- *Fetal heart rate slows*
- *Electronic fetal monitoring*
  - *Persistent late deceleration of any magnitude*
  - *Persistent severe variable deceleration*
  - *Prolonged bradycardia Decreased or absent beat-to-beat variability*
- *Thick meconium-stained amniotic fluid*
- *Fetal scalp blood analysis show pH less than 7.2*

EFFECTS

- *Hypoxic damage to most of the infant's organs (heart, lungs, liver, gut, kidneys), but brain damage is of most concern and perhaps the least likely to heal.*
- *In more pronounced cases, an infant will survive, but with damage to the brain manifested as either mental or physical disability, such as developmental delay or intellectual disability, or physical, such as*

EFFECTS CONT....

- ***Central nervous system*** : *Intracranial hemorrhage*

  Hypoxic-ischemic encephalopathy
- ***Cardiovascular*** : *Bradycardia*

  Arrhythmia

  Hypotension

  Myocardial ischemia

EFFECTS CONT....

- ***Respiratory system*** *: Apnea*
- ***KUB****: Acute tubular necrosis*
- ***Gastrointestinal tract*** *: Necrotizing enter colitis*
- **Hematology**: Disseminated intravascular coagulation
- **Metabolic:** Hypoglycemia
  Hyperglycemia
  Hypocalcaemia

EFFECTS CONT....

- ***Mental Disability***: *Developmental Delay, Intellectual Disability.*
- ***Physical Disability:***

  Spasticity,

  Motor Deficit.
- ***Cerebral Palsy***.

SPECIFIC MANAGEMENT
PREVENT FURTHER BRAIN DAMAGE

- *Maintain temperature, perfusion, oxygenation & ventilation.*
- *Correct & maintain normal metabolic & acid base milieu .*
- *Prompt management of complications.*

MANAGEMENT OF A NEONATE WITH PERINATAL ASPHYXIA

- ***Delivery room care:***

  Obtain arterial cord blood for analysis

- ***Transfer the infant to NICU if***

  Apgar score 0-3 at 1 minute

  Prolonged bag and mask ventilation (60 seconds or more)

  Chest compression

MANAGEMENT OF A NEONATE WITH PERINATAL ASPHYXIA

NICU care

1. ***Maintain normal temperature :***

- *Avoid Hyperthermia*

2. ***Maintain normal oxygenation and ventilation***

- *Maintain saturations between 90% and 95% and avoid any hypoxia or hyperoxia*
- *Avoid hypocarbia, as this would reduce the cerebral perfusion*
- *Avoid hypercarbia, which can increase*

MANAGEMENT OF A NEONATE WITH PERINATAL ASPHYXIA

NICU care

3. Maintain normal tissue perfusion

- *Start intravenous fluid*
- *Administer dobutamine (preferred) or dopamine to maintain adequate cardiac output, as required.*
- *Do not restrict fluid as this practice may predispose the babies to hypo perfusion.*
- *Restrict fluid only if there is hypernatremia(Sodium<120mg%) secondary to syndrome of inappropriate secretion of ADH (SIADH) or if there is renal failure.*

MANAGEMENT OF A NEONATE WITH PERINATAL ASPHYXIA

NICU care

4. Maintain normal hematocrit and metabolic milieu

- *Maintain blood glucose levels between 75 mg/dL and 100 mg/dl.*
- *Correct Anaemia and maintain hematocrit between 45% and 55%.*
- *Check blood gases to detect metabolic acidosis as needed and maintain pH above 7.30.*

MANAGEMENT OF A NEONATE WITH PERINATAL ASPHYXIA

NICU care

5. **Treat seizures**
6. **Nutrition:**

- Start oral feeding once baby is hemodynamically stable

7. **Miscellaneous**

- Administer Vitamin K (1 mg IM) to all infants with perinatal asphyxia

NEWER MODES OF THERAPY

1. ***Therapeutic hypothermia***
   - *33degree C to 34degreeC*
   - *in infants of at least 36 wk.*
   - *moderate to severe encephalopathy*
   - *initiated within 4- 6 hr*
   - *continued for 72 hr of age*
   - *reduce mortality and neuromorbidity by 18 months of age.*
   - *selectively cooling the head or the whole body*

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NEWER MODES OF THERAPY

2. Prophylactic phenobarbitone

- A dose of 40 mg/kg administered prophylactically was associated with a better neuro-developmental outcome at 3 years of age

3. Drugs under investigation

- A large number of drugs are under investigation for neuroprotection in HIE which need to be used in the early period.
  - Blockade of free radical generation (allopurinol, oxypurinol)
  - Scavenging of oxidants (superoxide dismutase, glutathione, Nacetyl cysteine and alpha tocopherol)
  - Calcium channel blockage (flunarizine, nimodipine)
  - Blockage of NMDA receptors (magnesium, MK801, dextromethorphan)
  - Blockage of inflammatory mediators (phospholipase A2, indomethacin).

PREDICTORS OF POOR NEURO DEVELOPMENTAL OUTCOME

- *Failure to establish respiration by 5 minutes*
- *Apgar 3 or less in 5 mts*
- *Onset of Seizure in 12 hrs.*
- *Refractory convulsion • Stage III HIE*
- *Inability to establish oral feed by 1 wk.*
- *Abnormal EEG & failure to normalize by 7 days of life*
- *Abnormal CT, MRI, MR spectroscopy in neonatal period*

PROGNOSIS

- Apgar score < 5 at 10 minutes : nearly 50 % death or disability (Leicester)
- No spontaneous respiration after 20 min :60 % disability in survivors (USA).
- No spontaneous respiration after 30 minutes : nearly 100 % disability in survivors


THANK YOU!