# 1. Perinatal asphyxia

**Definitions:-**

- WHO defines birth asphyxia as failure to initiate and sustain breathing at birth
- It can also be defined as placental or pulmonary gas exchange impairment leading to hypoxemia and hypercarbia.

# Cont. …

- As a result of **anaerobic glycolysis** lactate is produced and will accumulate in the brain, heart and other tissues with a resultant effect of metabolic acidosis (evidenced by low cord PH< 7).
- Manifestations of perinatal asphyxia are low APGAR score less than or equal to 6 at 5th minute or less than 3 at 10th minute and abnormal muscle tone.

# Epidemiology:

- Perinatal asphyxia is the second commonest cause of neonatal mortality only preceded by infection and the commonest cause of disability in surviving newborns.

- **Timing of Insult:** Asphyxia can occur during ***antepartum, intrapartum or postpartum period.***
- The following are risk factors for asphyxia.

# Cont. …

- **Antepartal events (20%)**
  - Maternal hypotension
  - Severe anemia
  - Cardiopulmonary diseases
  - Placental abruption
  - Maternal hypertension
  - Preeclampsia/eeclampsia
  - Maternal diabetes

# Cont. …

- **Intrapartum (70%)**
  - Problems with umbilical circulation (E.g. Cord prolapse)
  - Meconium aspiration
  - Prolonged labor (maternal/ fetal causes)

# Cont. …

- **Postpartum Asphyxia (10%)**
  - Prematurity
  - Cardiovascular abnormalities
  - Pulmonary malformations
  - Neurologic abnormalities
  - Severe infections
  - Bleeding, shock

# Pathophysiology:

- When there are factors, which result in low oxygen delivery to the newborn, the initial response is ***increased respiratory*** rate followed by ***apnea***.
- This is a critical time that newborns would require drying and stimulation.

- If asphyxia is prolonged the following two scenarios would happen.

1. **Brief asphyxia:** There is transient increase followed by a decrease in heart rate, elevation of blood pressure and essentially, there will be redistribution of cardiac output.

# Cont. …

- There will be gasping respiration.
- This is followed by increased blood flow to brain, heart and adrenal glands, which is referred to as **DIVING REFLEX**.

# Cont. …

2. **Prolonged asphyxia:** It leads to decreased blood flow with further compromisation of the heart cascaded by hypotension and increased anaerobic metabolism in the brain.

# Cont. …

- Decreased cerebral flow with anaerobic metabolism later complicates energy failure, increased glucose metabolism and ATP depletion.
- The final outcome is diffuse cortical and sub cortical injury.

# Cont. …

- **Clinical findings:-**
  - Renal failure: Oliguria (< 0.5ml/kg/hr)
  - Respiratory distress,
  - Tachycardia
  - Cardiomegaly
  - Hepatomegaly
  - Shock
  - Necrotizing enterocolitis /Bloody stool
  - Hypoglycemia
  - Hypocalcaemia

# Multiorgan Systemic Effects of Asphyxia

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# Cont. ….

**Laboratory evaluation**

- CBC
- RBS
- Urine analysis
- Stool for blood
- Renal function
- Liver function test
- Echocardiography as needed
- Serum electrolytes,
- EEG
- CXR
- Brain imaging

# Management of asphyxia in the newborn

I. Perinatal management of high risk pregnancies and early detection of fetal hypoxia
II. Neonatal resuscitation and post care
III. Post natal management
    a. Keep NPO for 48 hrs or until passing of meconium, normoactive bowel sound
    b. Fluid management:- two third maintenance fluid
    c. Oxygenation:- maintain between 90-95%

# Cont. ….

d. Do not warm asphyxiated babies:- cooling therapy is used
e. Correction f metabolic states:-
   - Hypoglycemia with 2ml or 4ml (if seizure) of 10% dextrose followed by maintenance,
   - Hypocalcaemia:- 1-2ml/kg of 10% calcium gluconate
f. Seizure treatment

***Parent counseling has to be the integral part of management!***

# Hyaline Membrane Disease (RDS type 1)

# 2. Hyaline Membrane Disease (RDS type 1)

- Respiratory Distress Syndrome (RDS) is the name for a cluster of symptoms that indicate severe malfunctioning of the lungs.
- In infants, RDS is termed Infant Respiratory Distress Syndrome (IRDS). Commonly found in premature infants –
- IRDS results when the alveoli fail to fully expand during inhalation.

# Hyaline Membrane Disease…

- Expansion of the alveoli requires a chemical called **surfactant**, but in many premature infants, the alveoli are not developed enough to produce this vital substance. Incidence by age:-
  - Less than 28 weeks 60 – 80%,
  - 32-36 weeks 15-35% in
  - >37 weeks 5%.

# Hyaline Membrane Disease…

- Adult Respiratory Distress Syndrome (ARDS) results when lungs are severely injured, for example, in an automobile accident, by poisonous gases, or as a response to inflammation in the lungs.
- ARDS is a life-threatening condition with a survival rate of about 50 percent.

# Hyaline Membrane Disease…

- **Associated factors:-**
  - Infants of diabetic mothers
  - Delivery by cesarean section
  - Multiple pregnancies
  - Precipitous delivery
  - Asphyxia
  - Cold stress and a prior history of affected infants.

# Hyaline Membrane Disease

**Etiology**

- **Surfactant deficiency** is the primary causes of HMD.
- The failure to develop a functional residual capacity and the tendency of affected lungs to become atelectalic correlate with high surface tensions and absence of pulmonary surfactant.

# Hyaline Membrane Disease

## Clinical features

- Signs of HMD usually appear with in minutes of birth, peak at 1-3 days, onset recovery is at 72hrs
- The late onset of tachypnea should suggest other conditions.
- Characteristically, **respiratory distress** (tachypnea, nasal flaring, subcostal and intercostal retractions, expiratory grunting) and **cyanosis** are noted.

# Hyaline Membrane Disease

- Decreased bilateral air entry may be noted on auscultation.
- The natural course is characterized by progressive worsening of cyanosis and dyspnea.
- In most cases, the symptoms and signs may reach a peak within 3 days, after which improvement is gradual.

# Hyaline Membrane Disease

**Investigation:-**

- CBC
- CXR
- BGA
- Septic work up
- SaO2

# Hyaline Membrane Disease

## 3. Fluid and metabolic management

- Maintenance of fluid requirements usually begin at 60 to 80 ml/kg/days as a 10 % dextrose solution and increases gradually to 150 ml/kg/day by the fifth day of life.

# Hyaline Membrane Disease

## 4. Antibiotics

- **Fear of** congenital pneumonia and the difficulty in distinguishing it from HMD.
- A penicillin combined with an aminoglycoside is the antibiotic regimen of choice.

# Hyaline Membrane Disease…

## 5. Surfactant therapy

- Multi-dose endotracheal instillation of exogenous surfactant for the treatment of HMD has dramatically improved survival and reduced the incidence of pulmonary air leaks.

# COMPLICATION OF SURFACTANT THERAPY

- Transient hypoxia, and hypotension,
- Blockage of the endotracheal tube,
- Pulmonary hemorrhage are complications related to RDS.

# Hyaline Membrane Disease…

## Prevention:-

- Most important is the prevention of prematurity
- Administration of **dexamethasone** to women 24-48 hours before delivery of fetuses less than 34 weeks of gestation significantly reduces the incidence and the mortality and morbidity of HMD.

# Complication and Prognosis

- Air leaks (pneumothorax, pneumomediastinum)
- Intracranial bleeding, pulmonary hemorrhage
- Bronchopulmonary dysplasia
- Retinopathy of prematurity

# Meconium Aspiration Syndrome

# 3. Meconium Aspiration Syndrome

**Definition:-**

- MAS refers to a condition in which the fetus or newborn develops respiratory distress after inhaling meconium mixed with amniotic fluid.
- Meconium is a thick, pasty, greenish-black substance that is present in the fetal bowel as early as 10 weeks' gestation.

# Cont. …

- Meconium staining of amniotic fluid usually occurs as a reflex response condition like hypoxia that allows the rectal sphincter to relax.
- Subsequently, meconium is released into the amniotic fluid.
- The fetus may aspirate meconium while in utero or with his or her first breath after birth that block the airway .

- Prompt Significant aspiration of thick meconium can induce 4 major pulmonary effects:
  - Airway obstruction,
  - Surfactant dysfunction,
  - Chemical pneumonitis and
  - Pulmonary hypertension

- **Risk factors**
  - Prolonged labor
  - Post maturity
  - Maternal illness,  diabetes, hypertension
  - Umbilical cord complications
  - Intrauterine growth retardation

# Cont. …

**Clinical Features**

- Meconium staining, nails, skin, umbilical cord
- Increased AP diameter of the chest
- Tachypnea, retractions, grunting, and cyanosis
- Pneumothorax, or pneumomediastinum, or both.

# Prevention

- Timely identification of fetal distress and initiating prompt delivery
- However, routine intrapartum nasopharyngeal suctioning in pregnancies with meconium-stained amniotic fluid does not reduce the risk for MAS and, may cause nasopharyngeal trauma or cause a cardiac arrhythmia, no more recommended.

# Cont. …

- Immediately after delivery of the baby, suck the oropharynx before drying and stimulation.
- Identify and treat for any complication.
- Suctioning via endotracheal tube is indicated for depressed infants before initiation of PPV.

- Investigation
  - CXR
  - Septic workup

# Management

- Vigorous infants born through meconium-stained fluid do not need routine intubation to aspirate the lungs.
- Endotracheal intubation and suctioning is indicated for depressed infants (those with hypotonia, bradycardia, or apnea).

# cont. . ...

- Those who fail to breathe spontaneously should not be stimulated until the airway is cleared to prevent aspiration during the first breath.
- Oxygen is critically important in infants with MAS
- Antibiotics are indicated

# Complications

- Persistent pulmonary hypertension
- Air leak — pneumomediastinum, pneumothorax , cystic lung disease
- Pulmonary hemorrhage
- Perinatal asphyxia

# Neonatal infections

- Neonatal Sepsis can be divided as early and late onset depending on the time of occurrence.
- Early-onset neonatal sepsis occurs with in the first 72 hrs of life
  - Is caused by organisms prevalent in the maternal genital tract, in the labor room or operation theatre.
  - Majority are caused by Gp B streprococcus,E-coli, klebsiela and enterobacter.
  - Majority manifest with respiratory distress due to an early intrauterine pneumonia.

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- Associated with certain high risk obstetric factors:
  - Birth asphyxia
  - Unclean vaginal examination
  - Foul smelling liquor
  - Prolonged labor (>24 hrs)
  - Preterm and low birth weight neonate
  - Prolonged rapture of membrane (>24 hrs)
  - Maternal pyrexia

...

- Late onset neonatal sepsis occurs after 72 hrs most of which is after the first week of life.
- Most are caused by gram negative bacteria's.
  - Klebsiela,enterobacter,E-coli,pseudomonas and salmonella,
  - Gram positives like staph **aurous** and **staph.albus** also contribute as causes of late onset sepsis.

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- Late-onset infections are acquired as **nosocomial** after delivery in the normal newborn nursery, neonatal intensive care unit (NICU) or the community.
- Usual sources include:-
  - Incubators, resuscitation equipment's,
  - feeding bottles catheters, infusion sets and sites

# Clinical features

- It may be subtle especially in those who are very small.
- Early manifestations could be change in behavior or feeding patterns.
- Gradual/sometimes suddenly:-
  - Lethargic, inactive and unresponsive
  - Failure to suck
  - HSM , Cyanosis
  - hyper/hypothermia
  - Evidences of respiratory problems (RD or apnea)
  - Failure to gain weight or loss of weight
  - Localizing features depending the organ/system affected

# Bacterial meningitis

- About a third of babies with neonatal sepsis can have coexisting meningitis.
- It is more common with late onset neonatal sepsis.
- Clinical evidence of meningial irritation are usually absent in the new born period
- In a baby with sepsis the finding of:-
  - Convulsions/twitching
  - Staring look
  - Bulged anterior fontanel
  - Abnormal high pitched cry/excessive cry
    - Should arouse the suspicion of meningitis and lumbar puncture should be done.

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- Abnormal CSF analysis
  - >30 cells/cubic mm
  - >60% polymorphs
  - CSF glucose /blood glucose ratio <50%
  - Protein>150 mg/dl
  - Presence of microorganisms

# Pneumonia in the newborn

- Is more common in early onset neonatal sepsis
- Some particularities of pneumonia in the NB
  - Minimal clinical signs.
  - Generalized signs of sepsis predominate esp. in premature NBs
  - Some neonates can have apnea rather than tachypnea.
  - Clinical signs of pneumonic consolidation may not be evident in the neonatal period

# Diagnostic work up

- Clinical + presence of high risk obstetric factors.
- Blood culture and sensitivity
- CSF analysis when indicated
- Chest X-ray
- Urin analysis and /or urine culture

# Management

- Depending on the etiologic agent
- Till etiologic agent is identified:-
  - Good broad spectrum coverage for gram +ve and gram –ve organisms is essential
- First line drugs
  - Crystalline penicillin 100,000iu/kg /day in two divided doses and
  - Gentamicin 5 mg/kg/day in two divided doses
  - Change crystalline penicillin with Ampicilline Lysteria monocytogens is implicated as the causative agent.
- Meningitis the dose of Ampicilline and crystalline is doubled.

# Management

- Second line drugs
  - Ceftriaxone 50mg/kg/dose BID +
  - Aminoglycoside dose as above
- Dose of ceftriaxone is doubled in cases of meningitis
- Supportive therapy to correct metabolic complications.
- Duration of therapy
  - Suspected sepsis (blood culture –ve) = 7 days
  - Proven sepsis (Blood culture +ve) = 14 days
  - Gram positive meningitis = 14 days
  - Gram negative meningitis = 21 days
  - Septic arthritis (osteomyelitis) = 4 to 6 weeks

# Prevention

- Universal precaution
  - Hand washing  before entering labor ward, before and after examine each infant.
    - Single most important means of preventing infection in NICU
  - Wear gowns and slippers when entering NICUs.
  - HCP with acute infections (Fever,ARI,GE,Skin lesions and viral exanthems ) should be restricted from providing care to the neonates.
  - Keep clean illness of the neonatal and labor ward(cleaning and fumigating at regular intervals)
  - Proper skin and cord care
  - Keep all equipments clean

# Jaundice and Hyperbilirubinemia in the Newborn

# Jaundice and Hyperbilirubinemia in the Newborn

**Hyperbilirubinemia** is a common and, in most cases, benign problem in neonates.Howevere,

- Untreated, severe indirect hyperbilirubinemia is potentially neurotoxic, and
- Conjugated-direct hyperbilirubinemia often signifies a serious hepatic or systemic illness.
- Jaundice is observed during the 1st wk of life in approximately 60% of term infants and 80% of preterm infants.

**Bilirubin occurs in plasma in four forms:**

1. U*nconjugated bilirubin* tightly bound to albumin;
2. ***Free or unbound bilirubin (the form responsible for kernicterus, because it can cross cell membranes);***
3. *Conjugated bilirubin* (the only fraction to appear in urine); and
4. D*elta* (d) *fraction* (bilirubin covalently bound to albumin), which appears in serum when hepatic excretion of conjugated bilirubin is impaired in patients with hepatobiliary disease.

## Etiology

**Unconjugated hyperbilirubinemia** may be caused or increased by any factor that:-

1. Increases the load of bilirubin to be metabolized by the liver
    - Hemolytic anemias,polycythemia, shortened red cell life as a result of immaturity or transfused cells, increased enterohepatic circulation, infection),
2. Damages or reduces the activity of the transferase enzyme
    - Genetic deficiency, hypoxia, infection, possibly hypothermia and thyroid deficiency),
3. Competes for or blocks the transferase enzyme
    - drugs and other substances requiring glucuronic acid conjugation for excretion
4. Leads to an absence or decreased amounts of the enzyme or to reduction of bilirubin uptake by liver cells
    - genetic defect, prematurity.

# Factors that increase the toxic effects of unconjugated bilirubin in serum

1. Agents that reduce the retention of bilirubin in the circulation
   - hypoproteinemia, displacement of bilirubin from its binding sites on albumin by competitive binding of drugs such as sulfisoxazole ,acidosis, increased free fatty acid concentration secondary to hypoglycemia, starvation, or hypothermia
2. Factors that increase the permeability of the blood-brain barrier or nerve cell membranes to bilirubin or increase the susceptibility of brain cells to its toxicity,
   - Asphyxia, prematurity, hyperosmolality, and infection.

# Risk Factors for Neonatal Hyperbilirubinemia

- **J**aundice visible on the 1st day of life
- **A** sibling with neonatal jaundice or anemia
- **U**nrecognized hemolysis (ABO, Rh, other blood group,incompatibility);
- **U**DP-glucuronyl transferase deficiency (Crigler-Najjar, disease)
- **N**onoptimal feeding (formula or breast-feeding)

- **D**eficiency of glucose-6-phosphate dehydrogenase
- **I**nfection (viral, bacterial).
- **I**nfant of diabetic mother.
- **I**mmaturity (prematurity)
- **C**ephalohematoma or bruising.
- Polycythemia **(C**entral hematocrit >65%)

# Clinical Manifestations

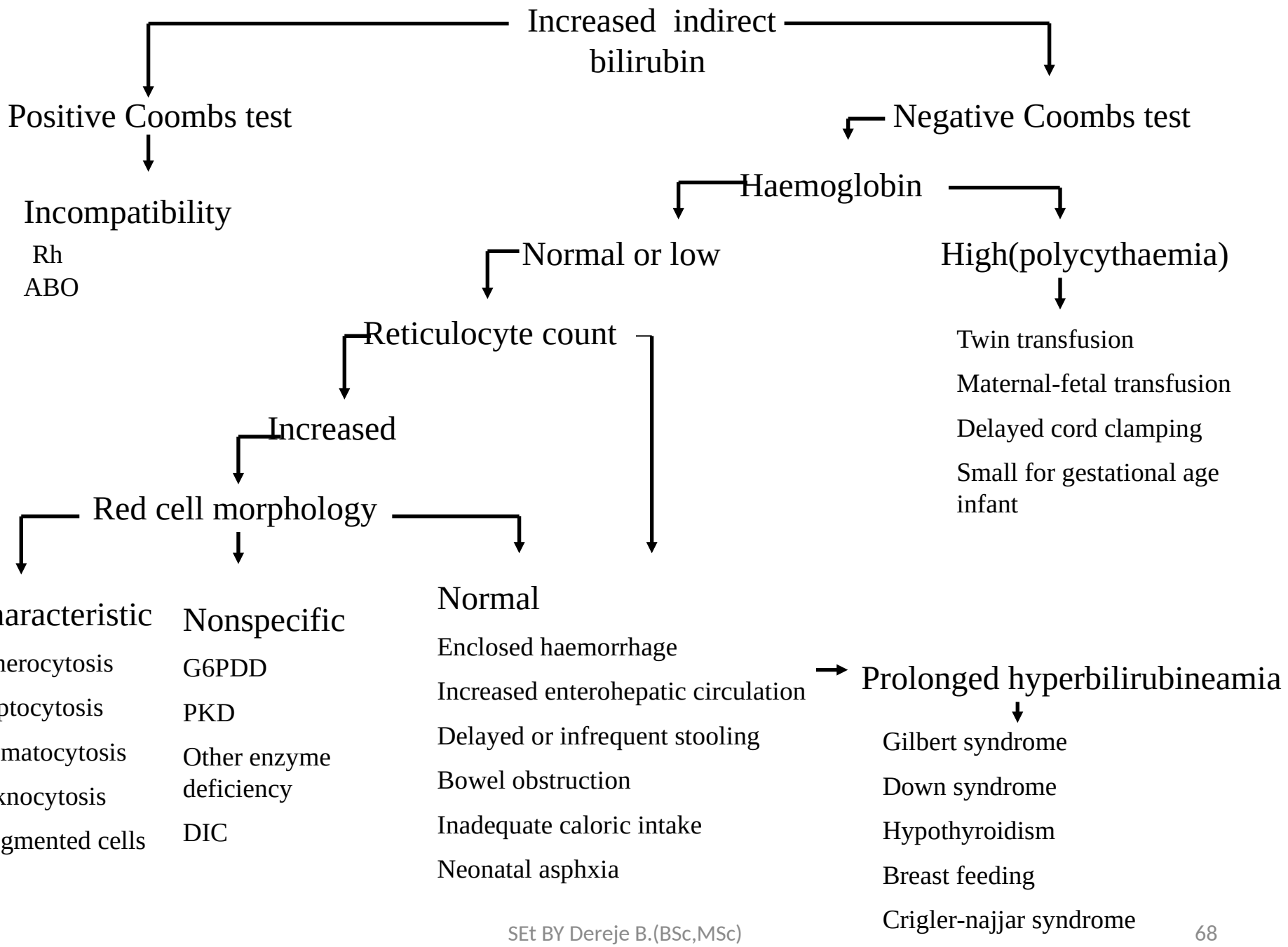
- Jaundice may be present at birth or may appear at any time during the neonatal period.
- Jaundice usually begins on the face and, as serum levels increase, progresses to the abdomen and then the feet.
- Dermal pressure may reveal the anatomic progression of jaundice (face, 5 mg/dL; mid-abdomen, 15 mg/dL; soles, 20 mg/dL), but clinical examination cannot be depended on to estimate blood levels.
- Jaundice resulting from deposition of indirect bilirubin in the skin tends to appear bright yellow or orange, and jaundice of the obstructive type (direct bilirubin) has a greenish or muddy yellow cast. This difference is usually apparent only in severe jaundice.
- Affected infants may be lethargic and may feed poorly. Signs of kernicterus rarely appear on the 1st day of jaundice

# Differential Diagnosis

- **Hemolysis** is suggested by a rapid rise in serum bilirubin (>0.5 mg/dL/hr), anemia, pallor, reticulocytosis, hepatosplenomegaly, and a positive family history.
- An unusually high proportion of direct-reacting bilirubin may characterize jaundice in infants who have received **intrauterine transfusions.**
- Familial nonhemolytic icterus **(Crigler-Najjar syndrome)** and early-onset breast-feeding jaundice are seen initially on the 2nd or 3rd day.
- *Jaundice appearing after the 3rd day and within the 1st wk should suggest bacterial sepsis or urinary tract infection;* it may be due to other infections, notably syphilis, toxoplasmosis, cytomegalovirus, or enterovirus.

- Jaundice that is noted **initially after the 1st wk of life** suggests breast milk jaundice, septicemia, congenital atresia or paucity of the bile ducts, hepatitis, hypothyroidism, congenital hemolytic anemia (spherocytosis), or possibly the crises of other hemolytic anemias

- Rarely, physiologic jaundice may be prolonged for several weeks, as in infants with **hypothyroidism or pyloric stenosis.**


Increased indirect bilirubin
Positive Coombs test
Incompatibility
Rh
ABO
Negative Coombs test
Haemoglobin
Normal or low
High(polycythaemia)
Twin transfusion
Maternal-fetal transfusion
Delayed cord clamping
Small for gestational age infant
Reticulocyte count
Increased
Red cell morphology
Characteristic
Spherocytosis
Eliptocytosis
Stomatocytosis
Pyknocytosis
Fragmented cells
Nonspecific
G6PDD
PKD
Other enzyme deficiency
DIC
Normal
Enclosed haemorrhage
Increased enterohepatic circulation
Delayed or infrequent stooling
Bowel obstruction
Inadequate caloric intake
Neonatal asphxia
Prolonged hyperbilirubineamia
Gilbert syndrome
Down syndrome
Hypothyroidism
Breast feeding
Crigler-najjar syndrome

# Increased direct bilirubin

***Sepsis***

***Intrauterine infection (TORCHS)***

***Biliary and bile duct abnormalities***

***Disorders of bile acid metabolism***

***Severe haemolytic disease***

# *Physiologic Jaundice (Icterus Neonatorum)*

- **Under normal circumstances, the level of indirect-reacting bilirubin in umbilical cord serum is 1–3 mg/dL and rises at a rate of less than 5 mg/dL/24 hr; thus, jaundice becomes visible on the 2nd–3rd day, usually peaking between the 2nd and 4th days at 5–6 mg/dL and decreasing to below 2 mg/dL between the 5th and 7th days of life.**

- **Jaundice associated with these changes is designated "physiologic" and is believed to be the result of increased bilirubin production after the breakdown of fetal red blood cells combined with transient limitation in the conjugation of bilirubin by the liver.**

- **Overall, 6–7% of full-term infants have indirect bilirubin levels greater than 12.9 mg/dL and less than 3% have levels greater than 15 mg/dL.**

***Persistent indirect hyperbilirubinemia*** beyond 2 wk suggests hemolysis, hereditary glucuronyl transferase deficiency, breast milk jaundice, hypothyroidism, or intestinal obstruction. Jaundice associated with **pyloric stenosis** may be due to

- Caloric deprivation,
- Deficiency of hepatic UDP-glucuronyl transferase, or
- Ileus-induced increased enterohepatic circulation of bilirubin.
- The diagnosis of physiologic jaundice in term or preterm infants can be established only by precluding known causes of jaundice

**Features of pathologic Hyperbilirubinemia**

(1) It appears in the 1st 24hr of life,

(2) Serum bilirubin is rising at a rate faster than 5 mg/dL/24hr,

(3) Serum bilirubin is greater than 12mg/dL in full-term (especially in the absence of risk factors) or 15mg/dL in preterm infants,

(4) Jaundice persists after 10–14 days of life, or

(5) Direct-reacting bilirubin is greater than 2 mg/dL at any time.

**Jaundice Associated with Breast-Feeding**

- **Occures in about 2% of breast fed teerm infants**
- **Usually after the 7th day of life.**
- **Maximum concentration can reach as high as 10 to 30 mg/dl during the 2nd to 3rd week of life**
- **Gradually decreases even if the baby is continued on breast feedingMay persist for 3 to10 weeks at lower levels**
- **Drops fast if breast feeding is stopped for a short period of time**
- **Cause is not known but may be due to the presence of glucornidase in breast milk**

# Kernicterus

- **Kernicterus, or bilirubin encephalopathy, is a neurologic syndrome resulting from the deposition of unconjugated bilirubin in the basal ganglia and brainstem nuclei**
- **The pathogenesis of kernicterus is multifactorial**
- **The precise blood level above which indirect-reacting bilirubin or free bilirubin will be toxic for an individual infant is unpredictable**
- **In previously healthy, predominantly breast-fed term infants, kernicterus has developed when bilirubin levels exceed 30 mg/dL,**

# Clinical Manifestations

## ACUTE FORM

- Phase 1 (1st 1–2 days): poor sucking stupor, hypotonia, seizures
- Phase 2 (middle of 1st wk) hypertonia of extensor muscles, opisthotonos, fever
- Phase 3 (after the 1st wk): hypertonia

## CHRONIC FORM

- First year: hypotonia, active deep tendon reflexes, obligatory tonic neck reflexes, delayed motor skills
- After 1st yr: movement disorders (choreoathetosis, ballismus, tremor) sensor neural hearing loss

# Incidence and Prognosis

- **By pathologic criteria, kernicterus will develop in one third of infants (all gestational ages) with untreated hemolytic disease and bilirubin levels in excess of 25–30 mg/dL**
- **Overt neurologic signs have a grave prognosis**
  - **75% or more of such infants die**
  - **80% of affected survivors have**
    - **Bilateral choreoathetosis with involuntary muscle spasms**
    - **Mental retardation**
    - **Deafness and**
    - **Spastic quadriparesis**

# Potentially preventable causes of kernicterus

- Early discharge (<48 hr) with no early follow-up (within 48 hr of discharge).Failure to check the bilirubin level
- Failure to recognize the presence of risk factors for hyperbilirubinemia
- Underestimating the severity of jaundice by clinical (i.e., visual) assessment.
- Lack of concern regarding the presence of jaundice
- Delay in measuring the serum bilirubin level despite marked jaundice or delay in initiating phototherapy in the presence of elevated bilirubin levels and
- Failure to respond to parental concern regarding jaundice, poor feeding, or lethargy

# TREATMENT OF HYPERBILIRUBINEMIA.

- The goal of therapy is to prevent indirect-reacting bilirubin related neurotoxicity .The risk of injury to the CNS can be reduced by phototherapy and if unsuccesful,echange transfusion

**Phototherapy**

The mechanisms by which phototherapy works are:-

a. Photoisomerization

   It will convert the toxic bilirubin to non toxic molecule.

b. Intramolecular cyclization of bilirubin to lumirubin.

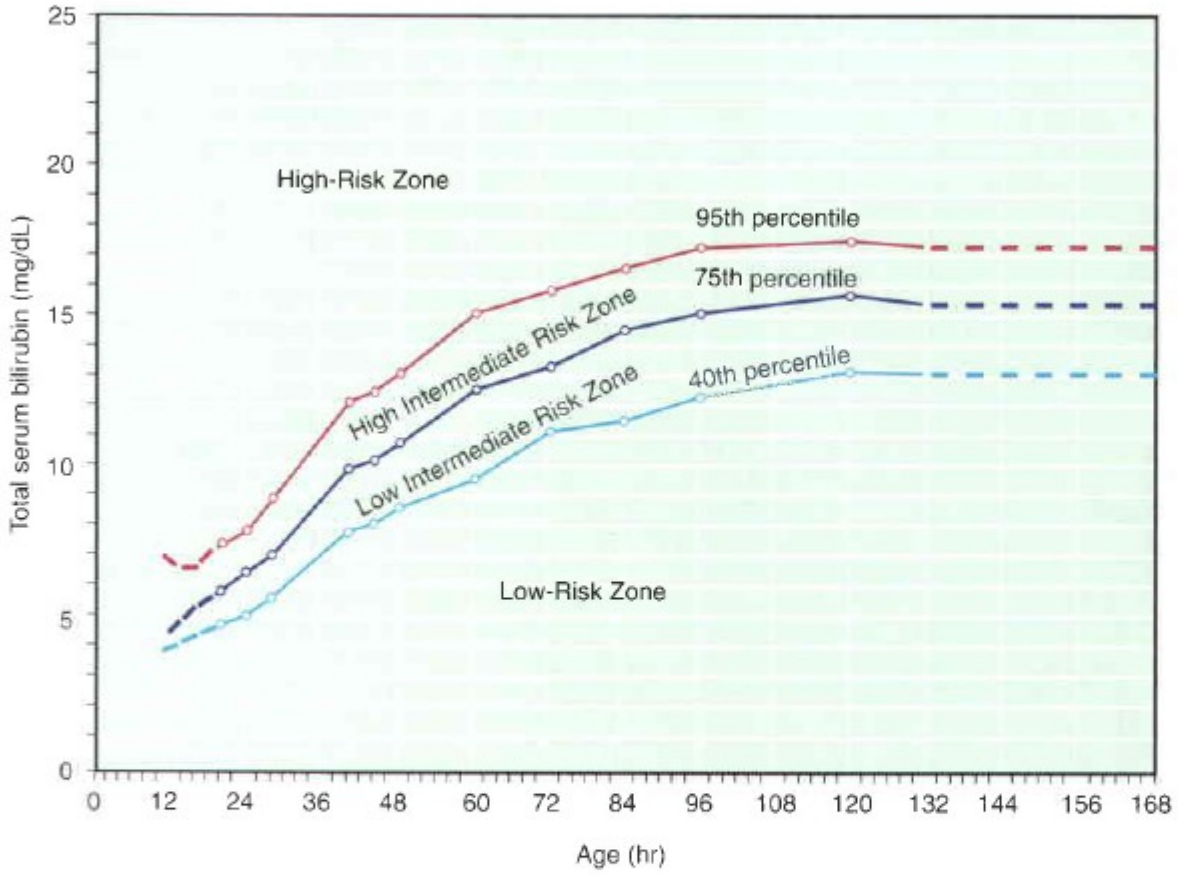
c. Photoxidation:- Conversion of bilirubin to polar,colorless product

Bilirubin absorbs light maximally in the blue range (420-470 nm).

- Bilirubin in the skin absorbs light energy, causing Several photochemical reactions.

- **While baby is under phototherapy:**
  - **Babies eye should be securely covered**
  - **Take baby of phothotherapy every 2 to 3 hours**
  - **Monitor fluid and electrolyte status**
  - **Increase fluid intake by 20 ml/kg**
  - **Monitor temperature regularly**
- **Clinical assessment of jaundice in babies under phototherapy May be fallaceaus**

# Indication of phototherapy



**Conventional phototherapy is applied continuously, and**

- **The infant is turned frequently for maximal skin surface area exposure.**
- **It should be discontinued as soon as the indirect bilirubin concentration has reduced to levels considered safe with respect to the infant's age and condition.**
- **Serum bilirubin levels and hematocrit should be monitored every 4-8 hr in infants with hemolytic disease or those with bilirubin levels near toxic range for the individual infant.**

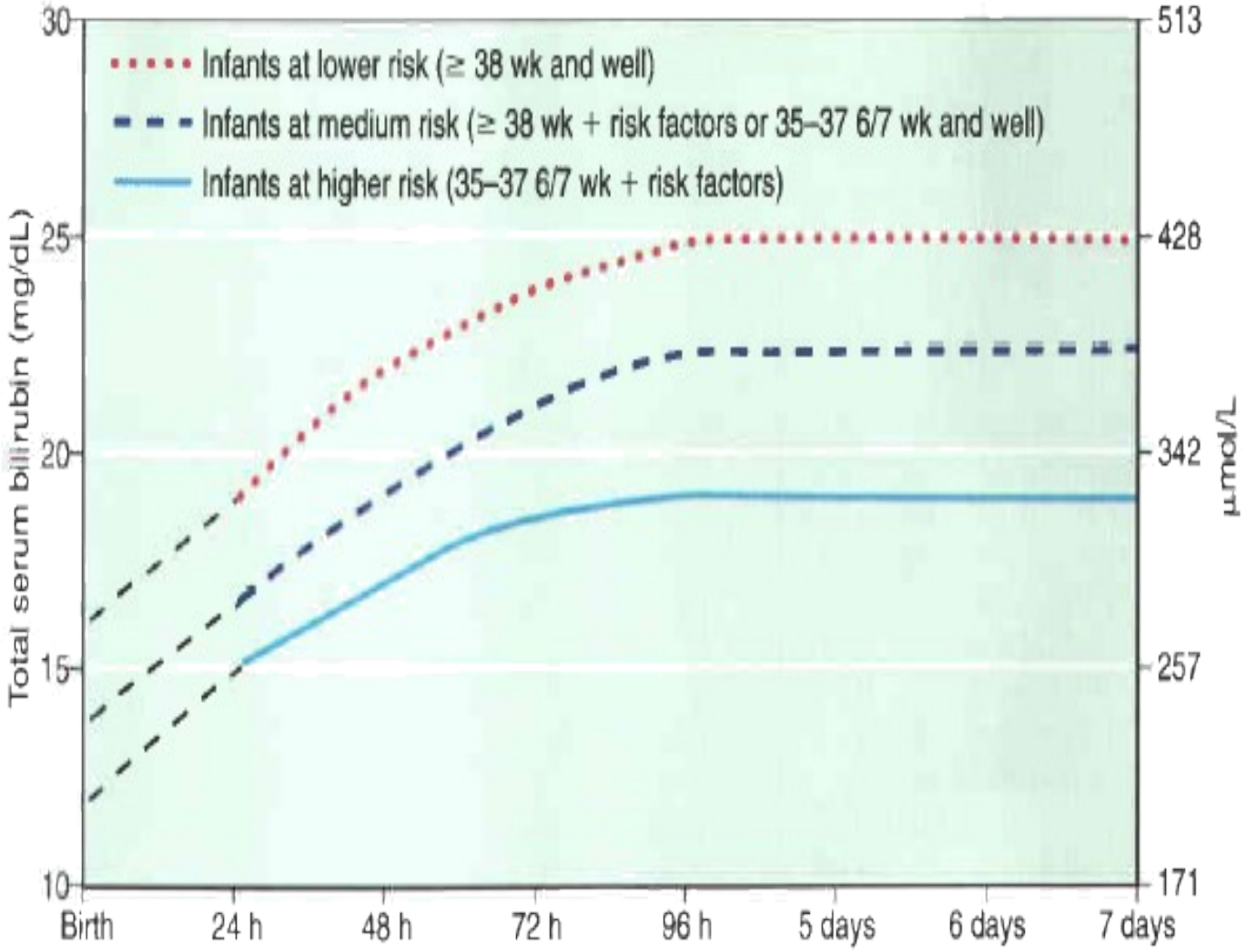
# Complications of phototherapy

- **Increased insensible water loss**
- **Retinal damage**
- **Erythematous skin rash**
- **Retinal damage**
- **Bronze baby syndrome**
- **Hypo or hyper thermia**
- **Sterility if tests not well covered**

# Exchange Transfusion

- **Most effective and reliable methode of reducing bilirubin level thereby reduce the risk of brain damage and kernicterus**
- **Can also help treat sever anaemia**
- **One double volume exchange transfusion lowers serum bilirubin to half pre exchange value**

# Indication of exchange transfusion



# Indications

- **Hydrops fetalis**
- **History of previous sibiling requiring an exchange transfusion because of Rh isoimmunization in a baby born with palor HSM and +ve direct coombs test**
- **Cord Hgb < 11gm**
- **Cord TSB >5 mg**
- **Rate of rise of TSB > 5mg /24hrs Despite phototherapy**
- **Rate of rise of TSB > 0.5mg/hr Despite phototherapy**
- **Any TSB > 12 mg in the 1st 24 hrs**
- **Any TSB >20 mg In the neonatal period**

# Preparation of infant

a. **Ensure pulse, temperature and respiration are stabilized and maintained.**

b. **Continue feeding the child and omit only the LAST feed before exchange transfusion. If needed, empty gastric content by doing NG aspiration before exchange transfusion.**

c. **Proper restraint.**

d. **Check resuscitation equipment.**

e. **Set a peripheral IV line.**

f. **Get a signed informed consent from parent (mortality from ET is 1%).**

# The procedure

- **Blood for exchange transfusion should be as fresh as possible.**
- **Type of blood used**
  - **Blood group of the baby and RH of the mother or O-ve blood in case of Rh incompatability**
  - **Blood group of the mother and Rh of the baby in case of ABO incompatibility**
- **Blood should be gradually warmed and maintained at a temperature between 35 and 37°C throughout the exchange transfusion.**
- **The infant's stomach should be emptied before transfusion to prevent aspiration,**
- **With strict aseptic technique, the umbilical vein is cannulated a distance no greater than 7 cm in a full-term infant.**

## The exchange

- Should be carried out over a 45-60 min period,
- Usually volume taken out and given will be 20 ml at a time but smaller aliquots (5-10 mL) may be indicated for sick and premature infants.
- The goal should be an isovolumetric exchange of approximately two blood volumes of the infant (2 **x**85 mL/kg).
- Exchange should start with removal of blood
- If child anaemic (Hb < 15) give an extra aliquot volume of blood at the end, leaving a positive balance).
- Place back under phototherapy lights after the procedure
- Feed after 3 hours.

# Post exchange

- **Give VITamin K 1mg**
- **Repeat all medications which the baby was receiving**
- **Monitor VS ½ hrly for the first two hrs**
- **Watch for bleeding from umblical stump**
- **Continue photo therapy**
- **Check for blood sugar,TSB And HCT after two hrs**
- **Start feeding after 2 to 3 hrs if baby is well and stable**

# Complications of ET

1. Catheter related
   - Infection
   - Haemorrhage
   - Portal and splenic vein thrombosis
   - Air embolism
2. Hemodynamic problems
   - Overload cardiac failure
   - Hypovolaemic shock
   - Arrhythmia (Catheter tip near sinus node in Rt Atria)

# Electrolyte imbalance

- Hyper kalemia (old blood)
- Hypocalcaemia
- Increase or decrease Blood glucose
- Acidosis (sometimes late alkalosis due to breakdown of citrate)
- Tissue hypoxia (old blood )

# Follow-up

- **Serial folow up of Serum bilirubin level till discharge and then 2 days after discharge**
- **For infant who had exchange transfusion, follow-up for 2 years**
  - **Assess every time for the late complications**
    - **Signs of deafness**
    - **Cerebral palsy and**
    - **Mental retardation**