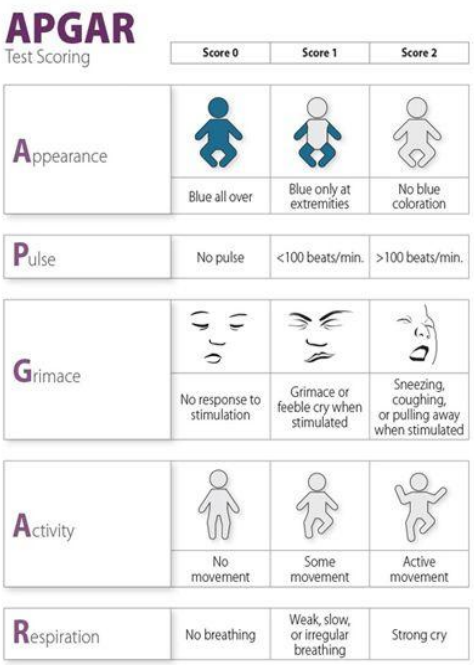
- **Neonatal resuscitation إنـ عاش-إحـ ياء:**
  - Steps which are done when there is abnormality in breathing or circulation of the neonate.
  - Resuscitation steps:
    - ✓ Place the neonate under radiant warmer.
    - ✓ Dry the neonate completely.
    - ✓ Gentle suctioning of mouth, oropharynx and nares.
    - ✓ Evaluation with APGAR score:
      - ❖ *1 minute*: is resuscitation needed?
      - ❖ *5 minutes*: was resuscitation effective?



Sign	0	1	2
Appearance	Pale/blue	Pink/blue extremities	All pink
Pulse	Absent	< 100	> 100
Grimace	No response	Grimace	Sneezing & coughing
Activity	Flaccid	Some flexion	Active motion
Respiration	Absent	Slow & irregular	Normal and crying

Score > 7	**Good condition** → wrapping baby, cutting the cord and do neonatal examination
Score 4-6	**Moderate asphyxia** →stimulate breathing by slapping soles or rubbing sternum → no response → bag & msk ventilation with 100% oxygen
Score < 4	**Severe asphyxia** → endotracheal intubation and cardiac massage until it rises above 80 beats/minute → if not → inset umbilical catheter with resuscitative drugs

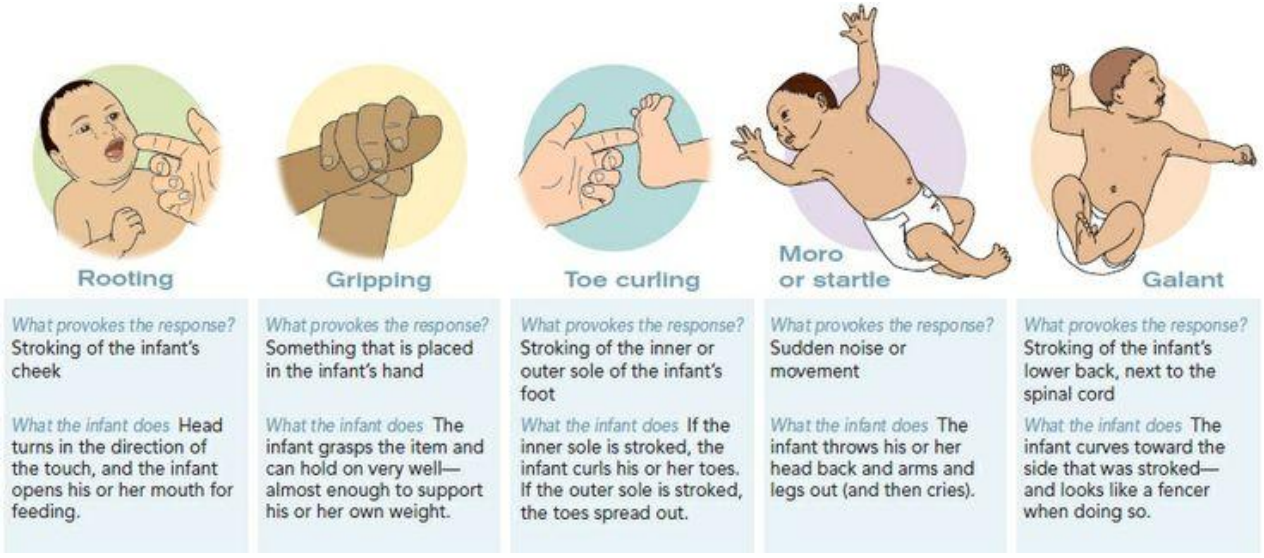
  - **Resuscitative drugs:**

Epinephrine	IV from 1:10,000 solution when there is bradycardia < 80 beats/minute
Naloxone	IV when mother received opiate analgesic during delivery because this will cause transient respiratory depression in her baby
$NaHCO_3$	IV if there is metabolic acidosis
Volume expanders	0.9% NaCl when there is hypovolemic shock
Dopamine	IV when there is cardiogenic shock due to prolonged asphyxia

- **Developmental reflexes (primitive reflexes):**
  - **Cerebral cortex in neonates is immature** → with time, maturation will occur with subsequent disappearance of these reflexes.
  - **Why are they important?**
    - ✓ If absent → damage to spinal cord and brainstem
    - ✓ If persistent → damage to cortex (no maturation).

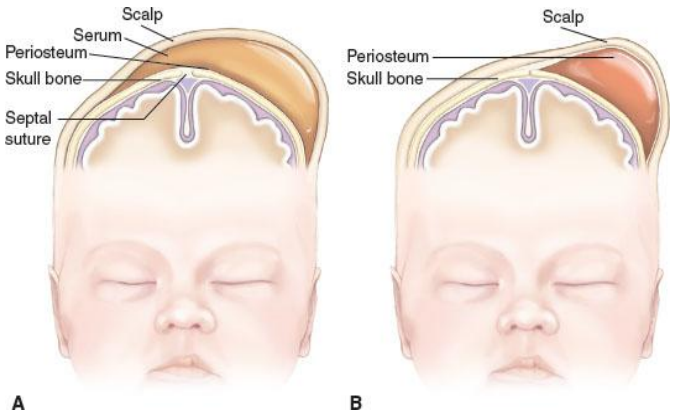
- **Important reflexes:**

Moro	• Present from birth – 6 months • Done by sudden dropping of the head from semi-sitting position on examiner's hand → response → abduction-extension followed by adduction-flexion of upper limbs with loud crying • Absent reflex: ✓ Bilateral: prematurity (< 28 weeks); CNS depression; bilateral injury to brachial plexus or clavicles. ✓ Unilateral: Erb's palsy
Grasp	• There is palmar grasp (present from birth-2 months) and solar grasp (present from birth-10 months). • If palmar grasp absent → klumpke's palsy (injury to C8,T1 of brachial plexus)
Rooting	• From birth -4 months • Finger stimulation near the angle of the mouth → turning of mouth to the stimulus.
Suckling	• From birth -4 months • Stimulation of lips → suckling movements
Tonic-neck	• From 1 month-6 months • While infant is supine the head is rapidly turned to one side → extension of upper and lower limbs on the side of turning with flexion on the other side
Landau	• From 3 months – 24 months • Infant is raised in prone position supported from beneath abdomen by the hand → extension of head, trunk and limbs

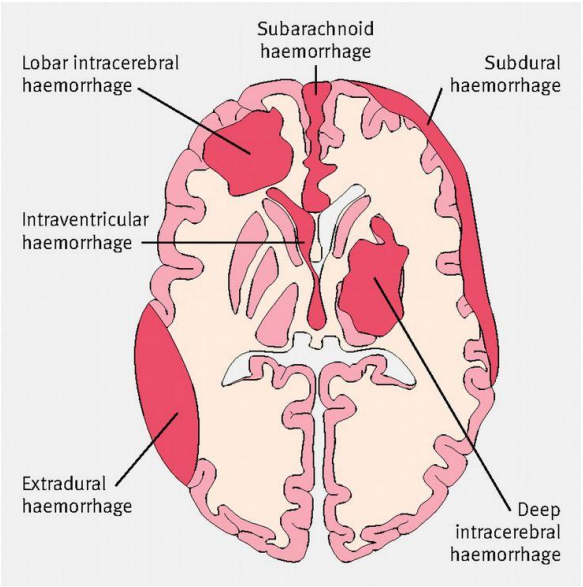


- **Birth injuries:**

	Caput succedaneum (A)	Cephalohematoma (B)
What is it?	Sub-cutaneous fluid accumulation	Sub-periosteal hemorrhage (parietal or occipital)
Onset	Immediately after birth	Within hours after birth
Crossing suture lines	Yes	No
Fate	Disappear within days	Disappear within 6-8 weeks



- **Intra-Cranial Hemorrhage (ICH):**
  - ✓ <u>What are the risk factors?</u>
    - ❖ Birth trauma (such as using forceps).
    - ❖ Bleeding disorder.
    - ❖ Vascular malformation.
  - ✓ <u>Types of ICH:</u>

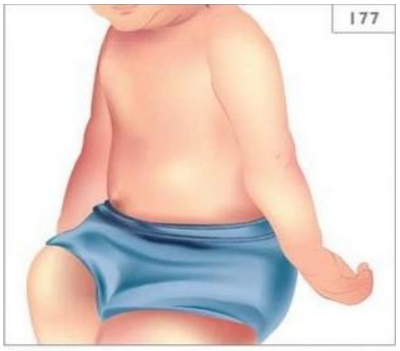


Sub-dural hemorrhage	Caused by trauma
Sub-arachnoid hemorrhage	Spontaneous (due to vascular malformations) or 2ry to perinatal asphyxia
Intraventricular hemorrhage	Mainly in preterms within first 3 days of life

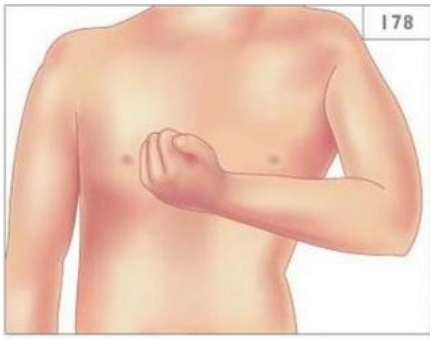
  - ✓ <u>Clinical features (seen with large hemorrhages) and treatment:</u>
    - ❖ *Hypovolemic shock (↓BP; tachycardia):* oxygen and IV fluids
    - ❖ *Pallor (anemia):* blood transfusion
    - ❖ *Seizures*: IV phenobarbitone
    - ❖ *Bulging fontanels (↑intracranial pressure):* IV mannitol
      <u>Notice that sub-dural hemorrhage requires surgical evacuation.</u>
  - ✓ <u>Investigations</u>: CBC (for anemia); coagulation profile (PT, PTT and platelets to rule-out the presence of a bleeding disorder); cranial ultrasound (mainly for intraventricular hemorrhage); CT/MRI (very accurate in defining lesions).
- **Nerve injuries:**
  - ✓ <u>Facial nerve injury (cranial nerve 7):</u>
    - ❖ *Peripheral facial nerve injury (LMN lesion) results in paralysis of half of the face on the same side of the lesion. It is characterized by:*
      - ➢ Inability to close the eye completely.
      - ➢ Absence of nasolabial fold.
      - ➢ Deviation of mouth towards the healthy side.
    - ❖ *If recovery doesn't occur within 3 months* → neuroplasty.

✓ Brachial plexus injury:

	Erb's palsy	Klumpke's palsy
Injury to	Upper trunk (C5,C6)	Lower trunk (C8,T1)
Presentation	Adduction, internal rotation and pronation of affected side → thus resulting in ABSENT MORO REFLEX on the affected side	Loss of all intrinsic muscles of the hand → thus resulting in ABSENT GRASP REFLEX
Treatment	Splint opposite to lesion (abduction, external rotation and supination) followed by physiotherapy after 1 week	Hand is kept in a neutral position with a cotton pad in the fist + physiotherapy
Prognosis	No recovery within 3 months → neuroplasty	



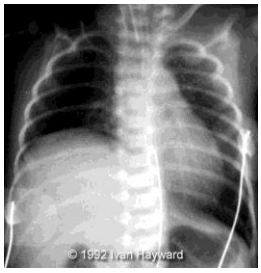
177 Erb's palsy.



178 Klumpke palsy.

✓ Phrenic nerve injury (C3, C4, C5):

- ❖ It will result in respiratory distress with no abdominal bulge during in spiration.
- ❖ *CXR shows*: elevation of diaphragm on the affected side.
- ❖ *Management*: placement on affected side + mechanical ventilation + oxygen.



- **Neonatal sepsis:**
  - **It is defined as systemic illness + positive blood culture (bacteremia).**
  - **Types:**

Early sepsis	Late sepsis
Within the 1st week	After the 1st week
Prematurity, prolonged ROM > 18 hours, chorioamnionitis and maternal bacteuria	Prematurity, endotracheal intubation, mechanical ventilation and umbilical catheterization
E.coli, GBS or Listeria	S.aureus

  - **Clinical features:**

Early manifestations (not specific)	Lethargy, vomiting and poor feeding, hypothermia and respiratory distress
Late manifestations (focal)	**Respiratory**: pneumonia with respiratory distress. CXR is done
	Neurologic: meningitis. Lumbar puncture is done
	Cardiac: ✓ Shock: pallor, hypotension, oliguria and cold skin ✓ Heart failure: tachycardia, tachypnea and cardiomegaly
	GI: vomiting and diarrhea, hepatosplenomegaly or necrotizing enterocolitis
	Hematologic: DIC
	Skin: sclerema (thickening of the skin) = poor prognosis

- **Investigations:**

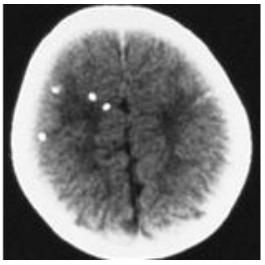
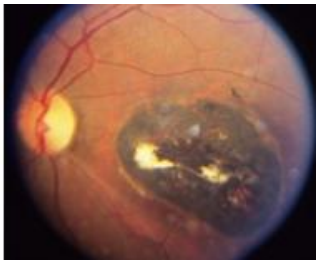
CBC	• ↓WBCs (< 5000 cells/mm^3) • Neutropenia (<1000 cells/mm^3) • Band cells (> 20% of total neutrophil count)
Markers of inflammation	• ↑ESR and CRP
Positive blood culture	

- **Management:**

Curative	Slow rewarming, oxygen and mechanical ventilation, hydration
Specific	Empiric antibiotic therapy after obtaining blood for culture: ampicillin, gentamicin and 3rd generation cephalosporin.

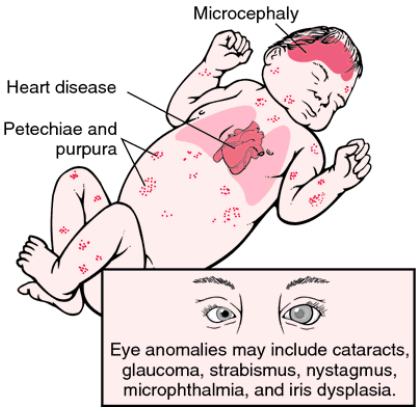
- **Congenital infections (TORCH):**

- **Toxoplasmosis:**
  - ✓ Caused by: protozoan Toxoplasma gondii that it transmitted through cat feces contaminating food and water.
  - ✓ Clinical features: Intracranial calcifications and chorioretinitis.

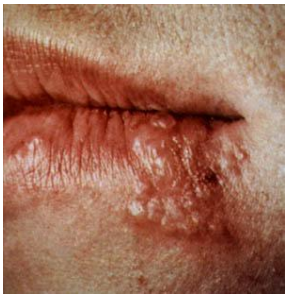
  - ✓ Diagnosis: serology or isolating organism from blood.
  - ✓ Treatment:
    - ❖ *Infected pregnant mother*: spiramycin.
    - ❖ *Infected baby*: sulphadiazine and pyrimethamine.

- **Rubella:**
  - ✓ It is an RNA virus causing German measles.
  - ✓ Clinical features: congenital deafness, congenital cataracts and congenital heart disease.
  - ✓ Diagnosis: serology or isolating organism from urine.
  - ✓ Management: rubella or MMR vaccine. In pregnant lady: induction of abortion or IV immunoglobulin (v accine cannot be given during pregnancy).

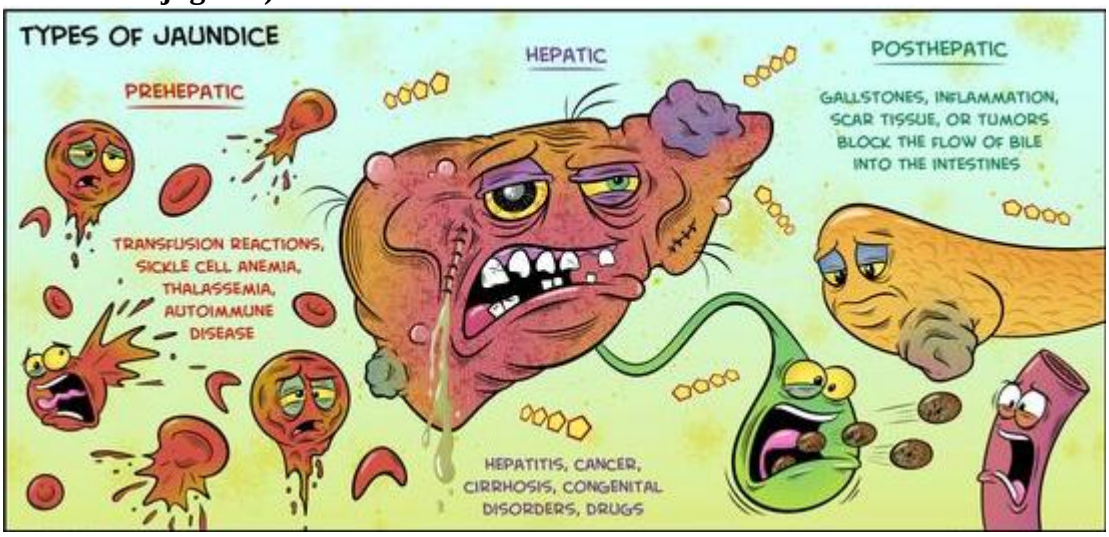


- **Cytomegalovirus (CMV):**
  - ✓ It is a DNA virus with vertical transmission (transplacental, secretions from birth canal or breast milk).
  - ✓ Clinical features: sensory neural deafness and neonatal thrombocytopenia and purpura.
  - ✓ Diagnosis: serology or isolating organism from urine.
  - ✓ Treatment: gancilovir.

- **Herpes Simplex Virus type-II (HSV):**
  - ✓ It is a DNA virus which is acquired from genital lesions (vesicles) during delivery.
  - ✓ Clinical features: skin/mouth vesicles and ulcers, keratoconjunctivitis and encephalitis.
  - ✓ Diagnosis: serology or isolating organism from vesicles or urine.
  - ✓ Treatment: acyclovir.



- **Neonatal jaundice:**
  - **Jaundice is defined as the yellowish discoloration or sclera (sclera icterus), skin and mucous membranes due to increased serum bilirubin (either unconjugated or conjugated) above the normal levels.**



  - **Indirect/unconjugated hyperbilirubinemia:**
    - ✓ It occurs when total bilirubin is > 5 mg/dL and direct bilirubin is < 15% of total bilirubin.
    - ✓ Causes: hereditary spherocytosis, α-thalassemia, G6PD deficiency, Rh/ABO incompatibility, large cephalohematoma, infections and breast-feeding jaundice.
    - ✓ Clinical features: yellowish discoloration of sclera and skin but no change in color of urine or stool. Extension of jaundice is from face to feet and this depends on the severity:

Face	Total bilirubin > 5 mg/dL
Abdomen	Total bilirubin > 10 mg/dL
Feet	Total bilirubin > 20 mg/dL

    - ✓ Complications: kernicterus which occurs when indirect bilirubin exceeds binding sites on albumin. Indirect bilirubin is lipid-soluble and can cross the blood-brain barrier and deposit in basal ganglia.
    - ✓ Physiologic jaundice: it is the most common cause of neonatal indirect hyperbilirubinemia which occurs after 24 hours of delivery and is characterized by decreased capacity of the enzyme UDP-glucouronyl transferase to conjugate bilirubin. Usually no treatment is needed and there is no risk of kernicterus.

	Full-term baby	**Pre-term baby**
Incidence	40%	60%
Onset	2^{nd} – 3^{rd} day	3^{rd} – 4^{th} day
Peak at	4^{th} day	6^{th} – 8^{th} day
Disappear	End of 1^{st} week	End of 2^{nd} week
Peak level	12 mg/dL	15 mg/dL

    - ✓ Criteria of pathological jaundice:
      - ❖ Occurring within 24 hours after delivery.
      - ❖ Rise in bilirubin level is > 5 mg/dL/day (compared to 3 mg/dL/day).
      - ❖ Persistent hyperbilirubinemia > 1 week in term infant and > 2 weeks in pre-term infant.
      - ❖ Peak level > 12 mg/dL in term infant and > 15 mg/dL in pre-term infant.
      - ❖ Notice that direct hyperbilirubinemia is always pathological.

✓ Treatment of indirect hyperbilirubinemia:

Phototherapy	• **It is avoided in direct hyperbilirubinemia (why?)** → bronzed baby syndrome. • **How it works?** by exposing the infant to light with different colors -white being most effective followed by blue and green- and a wave length between 425-475 nm to convert indirect bilirubin to more soluble forms that can be excreted through urine. • **Side effects**: skin rash, tanning of skin, hypo or hyperthermia and damaged to exposed eyes or genitalia.
Exchange transfusion	• **Done when bilirubin level exceeds:** ✓ 10 mg/dL at the 1st day. ✓ 15 mg/dL at the 2nd day. ✓ or 20 mg/dL at any time • It works by reducing the concentration of unconjugated lipid-soluble bilirubin in the blood. • O (-) blood is used with removal of small amounts (10-20 ml) of infant's blood and replacement by the new blood. IV glucose and calcium gluconate will be given at 100 ml intervals.

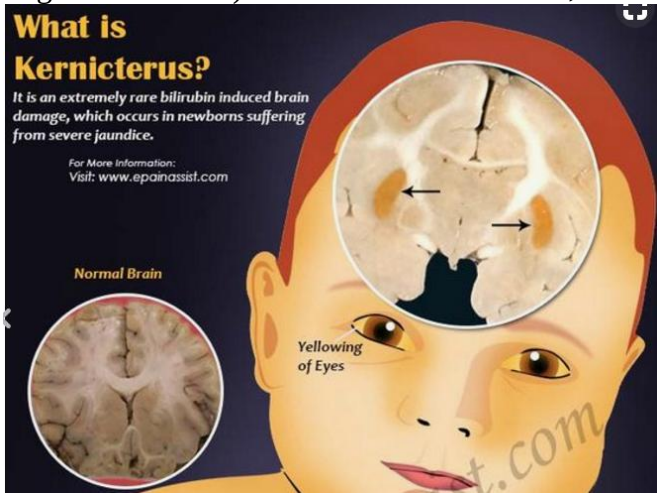
- **Kenicterus:**
  - ✓ It is defined as the deposition of the unconjugated lipid-soluble bilirubin in basal ganglia resulting in neuronal necrosis.
  - ✓ Risk factors:

Increased permeability of blood brain barrier	Displacement of unconjugated bilirubin from albumin
• Prematurity • Hypoxia • Acidosis • Anemia • Sepsis	• Hypoalbuminemia • Drugs (ampicillin and aspirin) • Hypothermia

  - ✓ Clinical features:

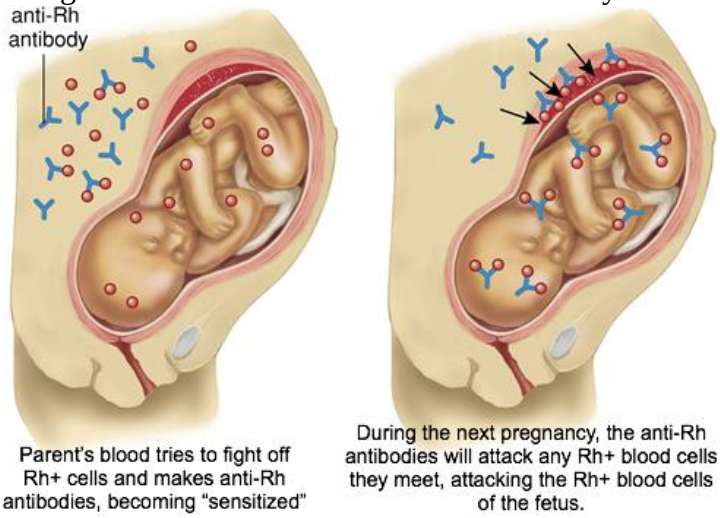
Acute	• **Phase 1**: 1st-2nd day; lethargy, poor suckling, loss of Moro reflex, hypotonia and seizures • **Phase 2**: hypertonia, fever and high-pitched cry • **Phase 3**: hypertonia and stiffness
Lucid (months)	Recovery or few symptoms
Chronic	Picture of cerebral palsy (chorio-athetosed or spastic)

  - ✓ Prevention: treatment of indirect hyperbilirubinemia (through phototherapy or exchanged transfusion). Once kernicterus occurs, it is not curable.



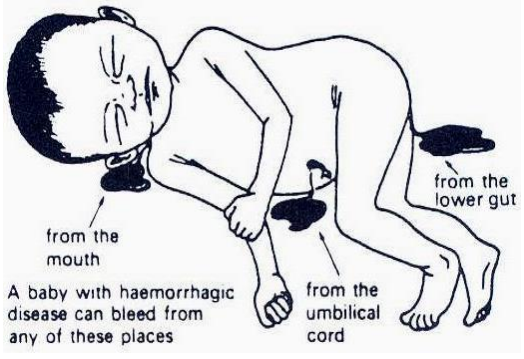
- **Conjugated hyperbilirubinemia:**
  - ✓ It occurs when total bilirubin is > 5 mg/dL and direct bilirubin is > 15% of total bilirubin. It occurs due to cholestasis which means that conjugated bilirubin cannot be excreted.
  - ✓ Causes: neonatal hepatitis (idiopathic), viral hepatitis, congenital intrahepatic biliary atresia, congenital extrahepatic biliary atresia or biliary stones/tumors.
  - ✓ Clinical features: jaundice with sclera appearing olive-green, dark urine, pale stool, malabsorption and FTT.
  - ✓ Investigations: LFT; total, direct and indirect bilirubin; HIDA scan and liver biopsy.
  - ✓ Management: Treatment of underlying cause when possible (biliary atresia is managed through Kasai portenterostomy operation); fat-soluble vitamins, ursodeoxycholic acid and liver transplantation for end-stage liver failure.

- **Hemolytic disease of newborn (Rh & ABO isoimmunization):**
  - **Rh isoimmunization:**
    - ✓ It occurs when mother is Rh (-) and her fetus is Rh (+). When getal blood escapes and enters maternal circulation she will develop anti-Rh antibodies. Usually, not consequences will occur in 1st pregnancy (unless there is previous formation of antibodies due to abortion of Rh (+) baby or from blood transfusion). In subsequent pregnancy, anti-Rh antibodies will cross the placenta into fetal circulation causing hydrops fetalis (which is characterized by severe hemolytic anemia, hepatosplenomegaly and jaundice. Notice that is is incompatible with life). Direct Coomb's test is strongly positive.
    - ✓ If mother is Rh (-) and baby is Rh (-) → anti-D injection will be given at 28 weeks gestation and within 72 hours after delivery.



  - **ABO incompatibility:**
    - ✓ Mother's blood group is O while baby's blood group is A, B or AB. The first baby can be affected as anti-A and anti-B antibodies are naturally present. Direct Coomb's test is weakly positive.

- **Hemorrhagic disease of the newborn:**
  - It occurs due to deficiency of vitamin K and its dependent factors (II, VII, IX and X).
  - Nowadays it is rare to occur because newborns are routinely injected with 1mg IM vitamin K immediately after delivery unless they are delivered at home with no medical care.
  - It tends to occur between 2nd-7th day after delivery (in the 1st day: there is still no



depletion of vitamin K which is derived from mother; in the 7th day: baby's interstinal bacteria will start synthesizing vitamin K). It also occurs more in prematures due to liver immaturity.

- **Clinical features**: baby looks fine except for severe hemorrhage (GI; umbilical; circumcision site) or intra-cranial hemorrhage. Hemorrhagic anemia also occurs.
- **Investigation:**
  - ✓ CBC: to look for anemia and platelet count (which is normal).
  - ✓ Coagulation studies: ↑PT and PTT but normal bleeding time.

- **Neonatal polycythemia:**
  - It is defined as an increase in number of RBCs relative to total blood volume with a Hct > 60%.
  - **Causes**: infant of a diabetic mother or chronic placental insufficiency (which results in chronic intrauterine hypoxia that leads to increased production of erythropoietin.
  - **Clinical features:**

Asymptomatic	Plethoric face
Symptomatic	Respiratory distress, lethargy and indirect hyperbilirubinemia

  - **Treatment**: for those who are symptomatic or Hct is > 75% → partial exchange transfusion.
- **Neonatal bleeding**

Bleeding in a healthy baby	Bleeding in a sick baby
• **Swallowed maternal blood**: this occurs during delivery (especially with CS) or from a cracked nipple resulting bloody vomit or stool. Apt test is done to differentiate maternal blood from fetal blood: ✓ Maternal blood → yellow ✓ Fetal blood → pink • **Hemorrhagic disease of newborn** • **Coagulation disorders (hemophilia or vWD)**	• **Necrotizing enterocolitis.** • **Intussusception or volvulus.** • **DIC.** • **Gastric stress ulcer.**

- **Neonatal anemia:**
  - **Physiologic anemia of infancy:**
    - ✓ **Normal Hb at birth = 14-20 gm/dL**
    - ✓ **After birth** → blood oxygen saturation increases → ↓ erythropoietin production → ↓Hb to reach a nadir (lowest point) of 10-11 gm/dL → again restimulating erythropoietin production.
  - **Pathologic anemia of infancy:**

	Blood loss	Hemolysis	↓RBC production
Cause	Twin-to-Twin Transfusion; feto-maternal transfusion; placental malformations; GI bleeding, caphalohematoma; frequent sampling; hemorrhagic disease of newborn	Rh/ABO incompatibility; spherocytosis; G6PD deficiency; α-thalassemia	Congenital leukemia
Investigations	↓Hb, normal/↑ retics and normal bilirubin	↓Hb, ↑retics and ↑bilirubin	↓Hb, ↓retics and normal bilirubin

  - **Management**: blood transfusion (in severe anemia or blood loss) and treatment of the underlying cause.
- **Necrotizing enterocolitis (acute intestinal necrosis):**
  - **Risk factors:**
    - ✓ Weak intestinal wall in prematures (most common).
    - ✓ Intestinal wall ischemia due to perinatal asphyxia.

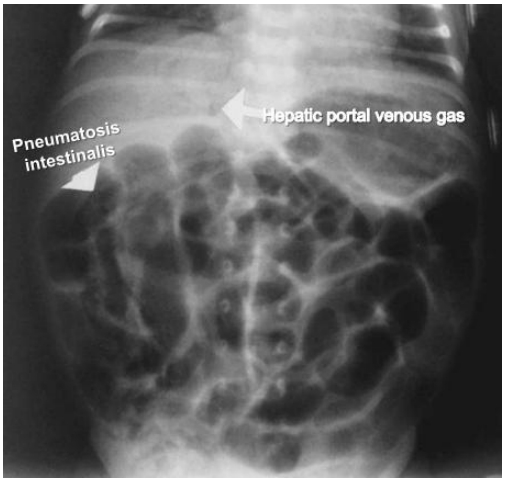
- ✓ Aggressive feeding with formula.

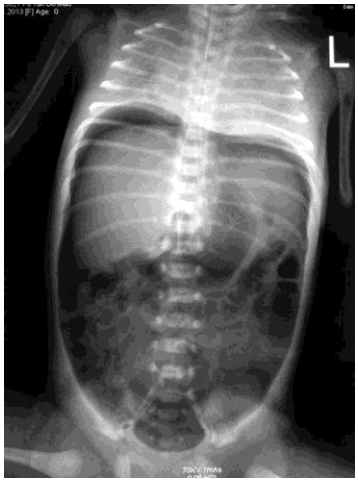
- **Pathogenesis:**
  - ✓ There will be sloughing and injury of intestinal wall especially in terminal ileum and proximal colon.
  - ✓ Superadded infections (with E.coli, Enterobacter or rotavirus) will cause extensive necrosis with gas production that might result in perforation and peritonitis.
- **Clinical features (occurring within first 2 weeks of life):**

Septicemic manifestations	Lethargy, poor Moro/suckling reflexes, poor feeding/vomiting, respiratory distress and hypothermia
Abdominal manifestations	Abdominal distention, abdominal pain, ileus (absent intestinal sounds) and bloody stool (gross or occult)

- **Investigations:**

Abdominal X-ray	• Pneumatosis-intestinalis (gas in abdominal wall) • Intrahepatic portal venous gas. • Pneumoperitoneum (gas under diaphragm indicating intestinal perforation)
Laboratory findings	• **Triad of**: thrombocytopenia, hyponatremia and metabolic acidosis.





- **Prevention**: prevention of prematurity, breast-feeding with avoidance of aggressive feeding.
- **Management:**

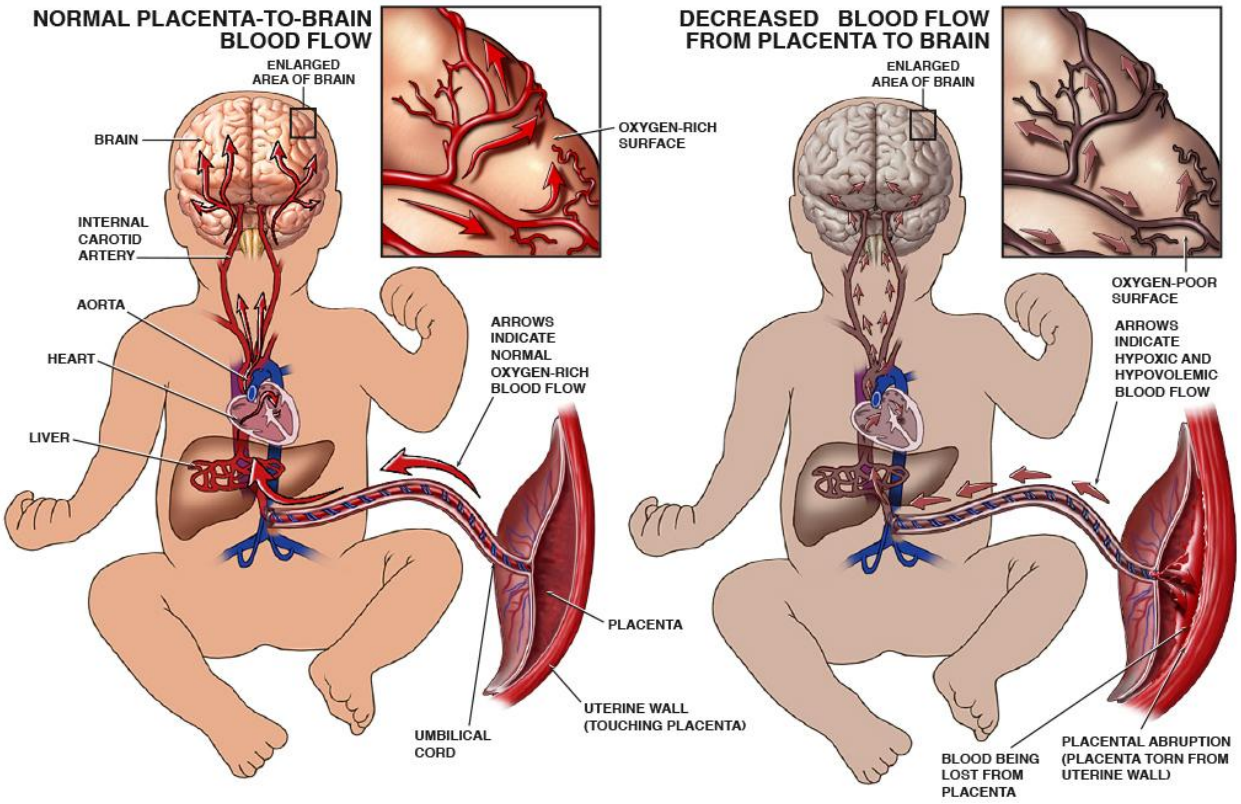
Medical	• Re-warming, stop enteral feeding, IV fluids, oxygen and mechanical ventilation. • $NaHCO_3$ (for metabolic acidosis), platelet transfusion (for thrombocytopenia) and correction of Na level.
Surgical	Resection of the bowel when there is failed medical treatment of perforation. Complication: short bowel syndrome.

- **Perinatal asphyxia:**

- **Perinatal asphyxia (APGAR score < 4):** it is defined as failure of the newborn to establish spontaneous regular breathing immediately after birth resulting in death or survival with permanent neurologic damage.
- **Normally, the first breath is stimulated by:**
  - ✓ $\downarrow PaO_2$ (by cutting the umbilical cord).
  - ✓ $\uparrow PaCO_2$
  - ✓ Hypothermia.
  - ✓ Tactile stimulation of breathing in delivery room (by slapping soles of feet or rubbing the sternum).

- **Causes:**

Intra-uterine	• **Maternal**: hypoventilation or hypotension. • **Placental**: insufficiency with postmaturity (where it will become calcified) or eclampsia (where there is hypertension causing decreased placental blood flow) • **Umbilical cord**: compression or knots
Intra-partum (during delivery)	Prolonged or obstructed labour.



- **Hypoxic-Ischemic Encephalopathy (HIE):** a late manifestation of severe perinatal asphyxia:
  - ✓ Pathology includes: brain edema and intra-cranial hemorrhage.
  - ✓ Pathogenesis: hypoxia results in anaerobic glycolysis with energy depletion which will cause primary neuronal death. Then, when reperfusion occurs, secondary neuronal death ensues due to release of neurotoxic mediators (such as NO, free radical and lactate).
  - ✓ Clinical staging:

Stage-I	Pupils dilated; prognosis 100%
Stage-II	Pupils constricted; prognosis 75%
Stage-III	Coma; death or severe deficits

  - ✓ Diagnosis: brain CT/MRI.
- **Management of perinatal asphyxia:**

Prevention	• Preventing risk factors (mentioned previously) • Neonatal resuscitation steps.
Curative	• Re-warming, IV fluids, oxygen and mechanical ventilation. • Symptomatic treatment for: ✓ Brain edema: fluid restriction by 20% and mannitol. ✓ Convulsions: phenobarbitone. ✓ Renal failure: peritoneal dialysis. ✓ Ensure normal blood glucose, calcium, magnesium and pH.

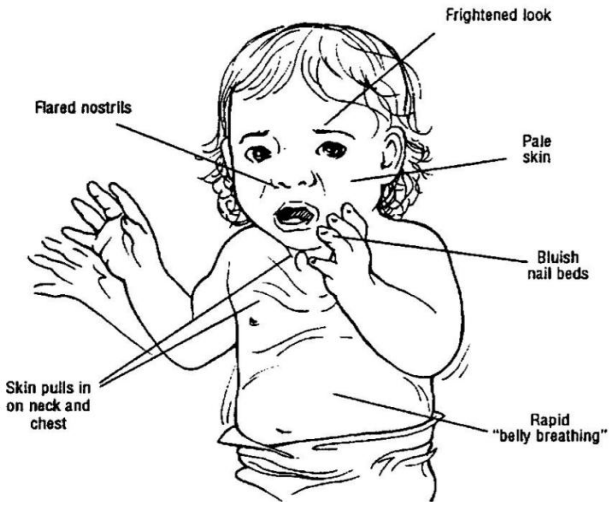
- **Neonatal seizures:**
  - **Causes:**

CNS	• Hypoxic-Ischemic Encephalopathy (40%) • Intra-cranial hemorrhage and CNS trauma (15%) • Meningitis and encephalitis (5%) • CNS malformations (5%) • Kernicterus
Metabolic	• **Hypoglycemia (< 40 mg/dL):** due to being an infant of diabetic mother, preterm or perinatal asphyxia. • **Hypocalcemia (serum calcium < 7 mg/dL):** ✓ Early-onset: infant of diabetic mother, preterm or perinatal asphyxia. ✓ Late-onset: ↓intake, hypoparathyroidism or hyperphosphatemia. • **Hypomagnesemia**: accompanying hypocalcemia. • **Hyponatermia (< 130 mEq/L) or hypernatremia (> 150 mEq/L)**
Others	• Neonatal epileptic syndromes

  - **Types of seizures:**
    - ✓ Subtle seizures خ ف يف:
      - ❖ *Eye movements*: blinking, nystagmus or sustained eye opening.
      - ❖ *Repetitive oral movements*: suckling, lip smacking or chewing.
      - ❖ *Limb movement*: bicycling or boxing.
      - ❖ *Epileptic apnea.*
    - ✓ Tonic seizures: the body becomes stiff.
    - ✓ Clonic seizures: characterized by jerky movements.
    - ✓ Myoclonic seizures: sudden, rapid, shock-like movement of the limb.
  - **Investigations**: serum electrolytes, metabolic screen (ammonia and pH), brain CT/MRI, EEG and sepsis screen (complete blood picture, blood culture and CSF examination).
  - **Management:**

Step-1 (stabilizing vital functions: ABC)	Suction of secretions, 100% oxygen inhalation and IV fluids.
Step-2 (correct transient metabolic disturbances)	• **Hypoglycemia**: glucose • **Hypocalcemia**: calcium gluconate • **Hypomagnesemia**: magnesium sulphate
Step-3 (anticonvulsants)	• Start with IV benzodiazapines • If no response, Phenobarbital or phnytoin • If no response, anesthesia with propofol

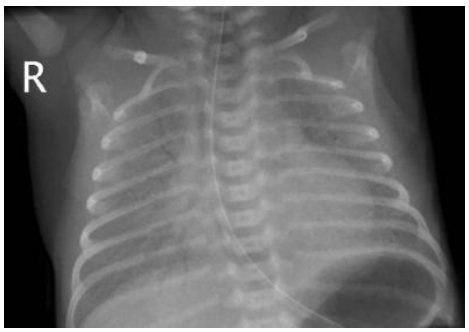
- **Neonatal respiratory distress:**
  - **Signs of respiratory distress**: nasal flaring, tachypnea (> 60 breaths/minute), retractions (intercostal or subcostal), grunting, cyanosis and altered consciousness.
  - **Respiratory Distress Syndrome (RDS):**
    - ✓ It is the most common cause of neonatal death and occurs due to deficiency of surfactant.
    - ✓ Surfactant is produced by type-II alveolar cells and

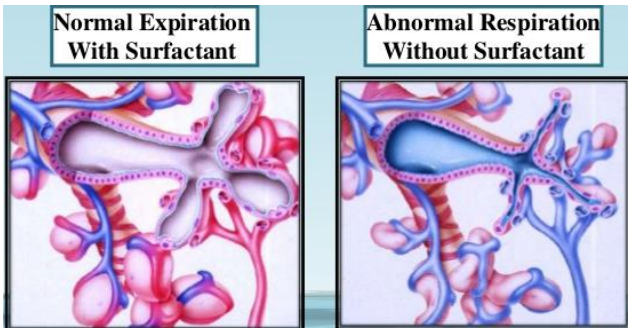


mature after 35 weeks of gestation. It is composed of dipalmitoyl phosphatidylcholine and it functions by reducing surface tension in alveoli thus preventing their collapse at the end of expiration.

- ✓ There are three main causes of respiratory distress syndrome:
  - ❖ Prematurity.
  - ❖ Infant of diabetic mother: maternal hyperglycemia → fetal hyperinsulinemia → ↓fetal cortisone.
  - ❖ Delivery by CS: lack of stressful NVD → ↓fetal cortisone.
- ✓ Clinical features: signs of respiratory distress which begin hours after delivery (4 hours); diminished air entry on auscultation (due to alveoli collapse); gradual improvement after the 3rd day in mild cases while severe cases will result in death.
- ✓ Complications: pneumothorax.
- ✓ Investigations:

Pre-natal diagnosis	• Lecithin/sphingomyelin ratio: if > 2.5 → mature lung with no risk of RDS
Post-natal diagnosis	• CXR shows: ground-glass appearance, air bronchogram and small lung volumes. • ABG (in severe RDS): ↓pH, ↓PaO_2 and ↑$PaCO_2$



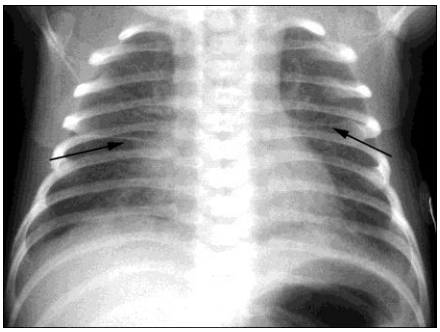


- ✓ Prevention:

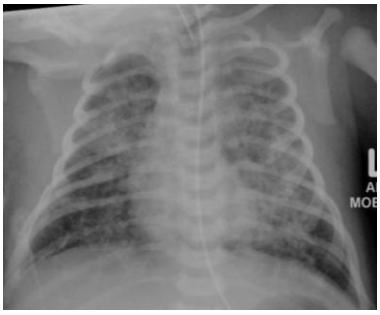
Avoid risk factors	• Avoid prematurity. • Avoid unnecessary CS. • Control maternal diabetes.
Steroid therapy	• Dexamethasone injection for pre-terms < 34 weeks of gestation

- ✓ Treatment of RDS: oxygen and IV fluids, antibiotics (as it is difficult to differentiate between RDS and congenital pneumonia: ampicillin + gentamicin), give surfactant (there is bovine type of porcine type).

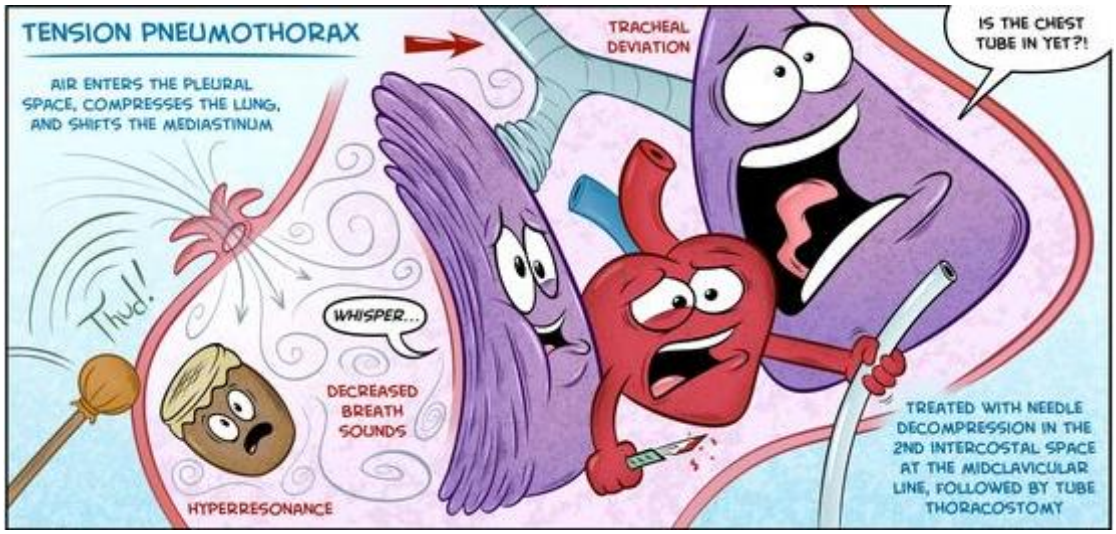
- **Transient tachypnea of newborn:**
  - ✓ It occurs due to delayed abso rption of lung fluids by pulmonary lymphatics due to maternal diabetes, CS or perinatal asphyxia.
  - ✓ Spontaneous resolution occurs within 2-3 days.
  - ✓ CXR shows: fluid in costophrenic angle and horizontal fissures.
  - ✓ Treatment: low-concentration oxygen and antibiotics.



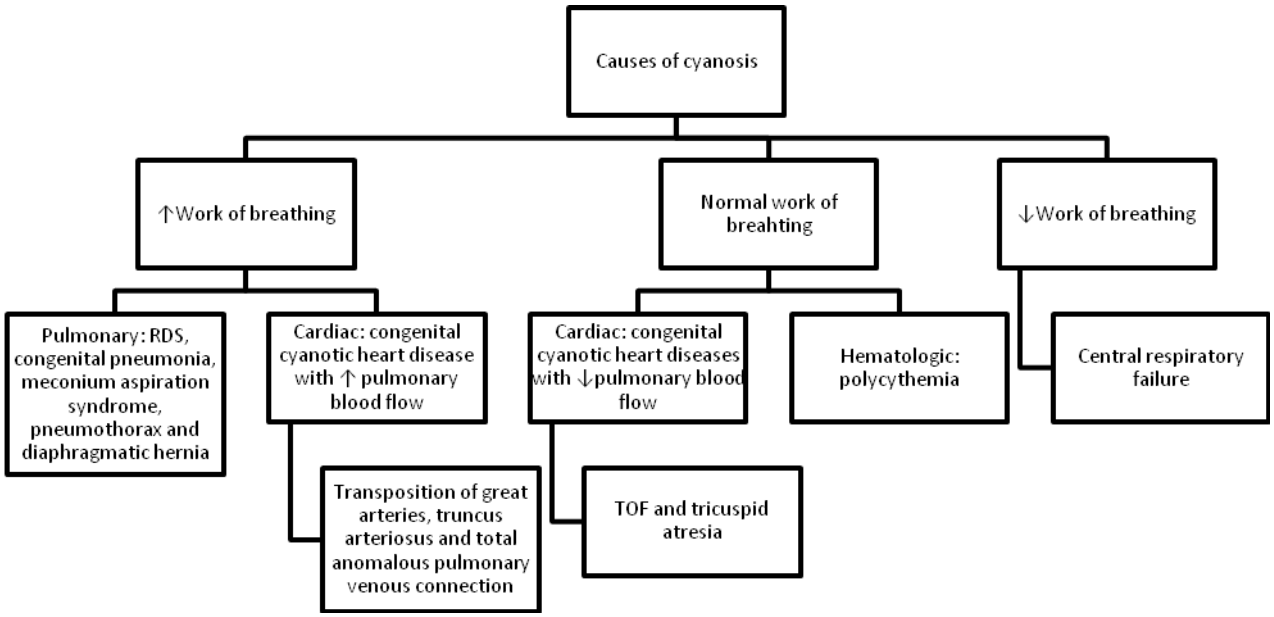
- **Meconium aspiration syndrome:**
  - ✓ Occurring in post-maturity when there is meconium-stained liquor that will be aspirated into lungs causing:
    - ❖ Patchy collapse.
    - ❖ Secondary infection and chemical pneumonitis.
  - ✓ CXR shows: patchy opacity of lungs.
  - ✓ Treatment: oxygen and antibiotics.



- **Pneumothorax:**
  - ✓ It is a complication of RDS in which CXR shows jet black opacity.
  - ✓ Treatment of tension pneumothorax:
    - ❖ Air aspiration by a needle.
    - ❖ Then, insert an intercostals chest tube for gradual drainage under water seal.



- **Neonatal cyanosis:**
  - It is defined as the bluish discoloration of skin and mucous membranes due to presence of > 5 gm/dL deoxygenated hemoglobin. It can be central (affecting the tongue) or peripheral (no affecting the tongue).



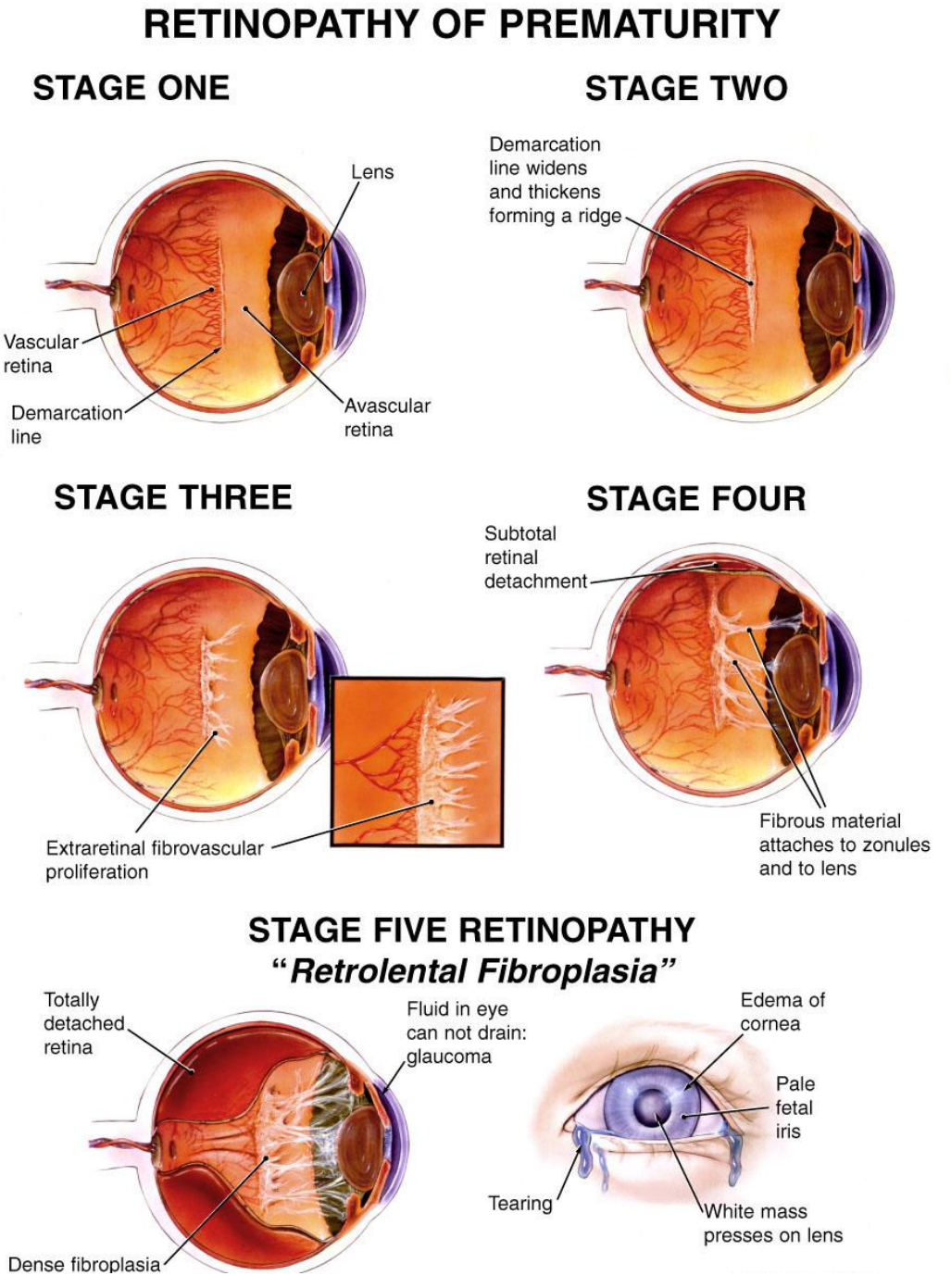
- **Hyperoxia test**: it helps in differentiating between pulmonary and cardiac causes of cyanosis.

- ✓ Do ABG in room air → then give 100% oxygen to the patient → measure ABG:
  - ❖ *If PaO_2 > 100 mmHg after 100% oxygen* = pulmonary cause of cyanosis.
  - ❖ *If PaO_2 remains < 100 mmHg after 100% oxygen* = cardiac cause of cyanosis.

- **Retinopathy of prematurity:**
  - It is a vasoproliferative retinal disorder which occurs in prematures who are exposed to high oxygen tension for long duration or have vitamin E deficiency.
  - **Stages:**

Stage-I	Retinal vasoconstriction
Stage-II	Retinal vasodilation with hemorrhage
Stage-III	Neovascularization
Stage-IV	Retinal detachment

  - **Management**: lowest oxygen tensions for shortest duration of time (when indicated to prematures) + screening with ophthalmoscope examination at 1 and 3 months.



- **Infant of a diabetic mother:**
  - **Features:**
    - ✓ Comonly delivered pre-term with increased birth weight (why?)
    - ✓ Maternal hyperglycemia → fetal hyperglycemia → ↑fetal glycogen synthesis, lipogenesis and protein synthesis → macrosomia.
    - ✓ Complications:
      - ❖ Increased risk of Intra-Uterine Fetal Death (IUFD).
      - ❖ Hypoglycemia due to fetal hyperinsulinemia that is stimulated by maternal hyperglycemia.
      - ❖ RDS.
      - ❖ Congenital anomalies (e.g. congenital heart disease and neural tube defects).
      - ❖ Macrosomia predisposing to shoulder dystocia.