DISEASE OF THE NEWBORN

Definitions and classifications

- **Newborn:** A live born infant during the first 4 weeks of life.
- **Live birth:** A live born infant that shows signs of life (movements, respiratory efforts, heart beats or pulsating umbilical cord).
- **Still birth:** A fetus born dead at / ≥ 24 weeks gestation.
- **Abortion:** A fetus born dead at /< 24 weeks gestation.
- **Preterm:** A live born infant < 37 weeks gestation.
- **Term:** A live born infant 37- <42 weeks gestation.
- **Post-term:** A live born infant ≥ 42 weeks gestation**.**
- **Normal birth weight:** 2500 – < 4000 g.
- **Macrosomia:** birth weight ≥ 4000 g.
- **Low birth weight:** ≤ 2500 g.
- **Very low birth weight:** < 1500 g.
- **Extremely low birth weight:** < 1000 g.
- **Appropriate for gestational age (AGA):** a birth weight lies between 10^{th} and 90^{th} percentile for the G.A.
- **Large for gestational age (LGA):** birth weight > growth percentile for the G.A.
- **Small for gestational age (SGA):** a birth weight < 10^{th} percentile for the G.A.

Care of the Newborn

Examination of the Newborn

Apgar score:-

1. It is a practical method to assess the condition of the newborn immediately after birth, done at 1 minute and at 5 minutes, to identify those requiring resuscitation.
2. Five objective signs are evaluated and each is given a score of 0, 1, or 2 according to the following illustrative table:

APGAR score

Sign		0	1	2
A	Appearance (Color)	Blue Pale	Pink Body Blue Extremities	Pink
P	Pulse	Absent	Below 100	Over 100
G	Grimace (Response to nasal Catheter)	No Response	Grimace	Cough Sneezing
A	Activity (Muscle Tone)	Limp	Some Flexion & Extension	Active Motion
R	Respiratory Effort	Absent	Slow - Irregular	Good Crying

Significance

APGAR Score	Diagnosis	Management
0 – 3	Cardiopulmonary Arrest	Resuscitation
4 – 6	Close attention	Resuscitation
7 – 10	Normal Infant	Discharge

Assessment should be carried on at: 1 – 5 – 10- 15 minutes after Birth

Interpretation:

1. Apgar score of 0-3 at 1 min; requires immediate resuscitation.
2. Apgar score of 4-6 at 1 min; requires resuscitation & Observation
3. Apgar score of 7-10 at 1 min; is normal.
4. Apgar score of 0-3 at 5 min; requires intubation, positive pressure ventilation, and admission to NICU.
5. Apgar score of 4-6 at 5 min; indicates perinatal asphyxia and requires continuing resuscitation efforts, and assessment of apgar at 10, 15 & 20 min.

Ordinary Care of Newborn infants

1) Assure patent airways by the following steps :

a) Suctioning of the oropharynx and nasopharynx.

b) Gentle tapping on the soles or back of the baby.

c) Aspiration of gastric contents using a gastric tube.

2) Maintenance of body temperature to guard against hypothermia by :

a) Wrapping the baby using a sterile warm towel.

b) Put the baby under a source of external heat (radiant warmer).

3) Care of the cord (To guard against tetanus neonatorum and omphalitis) **by:**

a) Use sterile disposable umbilical clamp.

b) Antiseptic lotion (Alcohol 70% or Betadin) till falling of the stump.

4) Care of the eyes :

Use prophylactic antibiotic eye drops to guard against ophthalmia neonatorum other mucopurulent conjunctivitis.

5) Vitamin K: IM 1 mg single dose to guard against hemorrhagic disease of the newborn.

Resuscitation of the newborn

1. Neonates tolerate asphyxia better than any other age.
2. Delayed or inadequate resuscitation may lead to death of the baby or brain damage. Therefore, a person skilled in resuscitation must attend all high-risk deliveries.
3. Examples of high-risk deliveries are
   a. Maternal Diseases (hypertension, toxemia, diabetes, cardiac, pulmonary, renal…).
   b. Caesarean section.
   c. Breech delivery.
   d. Preterm labor.
   e. Premature rupture of membrane (PROM).
   f. Ant partum hemorrhage.
   g. Multiple pregnancies.

Steps of resuscitation

1. Start by rapid assessment of **color** of the baby, **breathing** and **heart rate**:
2. If the baby is pink, has good respiration and his/her heart rate is less than 100/min, dry the baby, wrap in warm towels and give to the mother.
3. If the baby is blue, has inadequate respiration and his/her heart rate is more than 100/min, perform
   i. Gentle suction of the mouth and nose.
   ii. Gentle tactile stimulation.
   iii. Give oxygen by mask:
      a. If improved, give to the mother.
      b. If not, start bag & mask ventilation.
4. If the baby is blue, has apnea and his/her heart rate is less than 100/min, start bag &mask ventilation.

a. If responds, give to the mother.
b. If not, start intubation & ventilation.

5. If heart rate is less than 60/min or absent, start intubation & ventilation, cardiac massage. Consider giving IV adrenaline.

Examination of the newborn

1. Measurements: weight, length, head circumference.
2. Vital signs: temperature, RR, HR, BP& capillary refill time.
3. Color: cyanosis (central or acrocyanosis), pallor, jaundice, plethora, meconium stained, mottling, Mongolian spots.
4. Respiration: respiratory rate, intercostal and subcostal retractions, grunting, stridor, wheeze, air entry, breath sounds, rhonchi, crepitations.
5. Cardiovascular: heart rate, peripheral pulses, murmurs, capillary refill time.
6. Abdomen: abdominal distension, concavity, palpation of organs: liver, spleen, kidneys, intestinal sounds, passage of meconium, voiding of urine, genitalia.
7. CNS: level of consciousness, head circumference, anterior fontanels, sutures, pupils, muscle tone, reflexes, abnormal movements, convulsions.
8. Musculoskeletal: spines, extremities.
9. Congenital anomalies.

Neonatal reflexes

- The primitive neonatal reflexes are present since birth.
- They occur at the subcortical level, as the cerebral cortex is functionally deficient.
- They disappear at 4-6 months of age with maturation of the cerebral cortex.

1- Moro reflex:-

How to elicit (stimulus):-

1. Placing the baby in a semi-upright position, with shoulders and back supported by the examiner's hand. The head is suddenly allowed to fall backward, with immediate support by the examiner's hand.
2. Sudden withdrawal of the blanket from underneath the infant.
3. Sudden loud sound behind the infant's ear.

Normal Response:

- Sudden jerking movements consisting of abduction and extension of both arms, followed by adduction and flexion in an embracing manner.
- Time of appearance: ≥ 28 weeks gestation.
- Time of disappearance: at 4 months of age.

Significance:-

- Normal response: it indicates a normal neurological condition.
- Absence or poor response: it indicates either marked prematurity or depression of CNS (HIE, ICH, sedation, or anesthesia given to the mother during delivery).
- Asymmetrical or unilateral response: it indicates focal neuromuscular lesion: Erb`s palsy, fractured clavicle, dislocated shoulder.
- Abnormal persistence of the reflex beyond 4-6 months of age indicates CNS damage.
- Exaggerated response: it indicates brain irritation, as in early stages of HIE, kernicterus, meningitis.

2- Sucking reflex:-

***Stimulus*:** introduction of a finger into the mouth of the baby.
***Response*:** sucking movements of the mouth.

- Time of appearance: ≥ 28 weeks gestation.
- Time of disappearance: at 4 months of age.

Birth Trauma

Predisposing Factors:

1. Large baby.
2. Breech delivery& abnormal presentations
3. Preterm birth.
4. Precipitate deliveries
5. Contracted pelvis/ Cephalo pelvic disproportion.

- **Caput-Succedaneum:**

1. It consists of edema of the scalp of the presenting part.
2. It occurs in obstructed or prolonged labor.
3. It is a soft swelling in the head of the baby.
4. It may cross sutures (Cephalhematoma does not cross sutures).
5. Although it's appearance is very alarming, it is a benign condition.
6. No treatment is required. It resolves within few days.

- **Cephalhaematoma:**

1.This is a subperiosteal bleeding.
2. It occurs in obstructed labour.
3. It takes days to reach a full size.
4. A small fracture in cranial bone(s) leads to bleeding into subperiostium.
5. It is usually limited to one cranial bone
6. Anemia, jaundice and parental anxiety are the main complications

7. Treatment:

a. Reassurance
b. Symptomatic treatment
   i. Blood transfusion (if there is severe pallor).
   ii. Observe for jaundice and treat accordingly.

- **Intracranial Hemorrhage:**

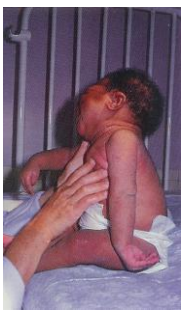
This is the most catastrophic complication of birth trauma. It may be epidural, subdermal parenchyma or intraventricular (preterm)

- ***Clinical features***:

Asymptomatic
Pallor, shock, tachypnea, tachycardia.
Jaundice.
Apnea.
Poor reflexes, fits, coma.
The diagnosis is confirmed by cranial ultrasound &CT.

- ***Treatment*:**

a. General supportive care
b. Minimal handling
c. Vitamin K, fresh-frozen-plasma
d. Symptomatic treatment e.g., treat fits
e. Ginformation for parents



Low birth weight:

Definitions

Low birth weight (LBW) is defined as a live born infant weighting $\leq$ 2500 g at

It includes preterm baby (live - born baby delivered before 37 weeks of gestation) and small for date baby (birth weight less than 10^{th} percentile for the gestational age).

Preterm baby (Prematurity)

Causes of Prematurity

- Idiopathic.
- Secondary:
  - Congenital malformation - Congenital anomalies
  - Twin Pregnancy. - Ante – partum hemorrhage.
  - Toxemia of pregnancy. - Maternal diabetes.
  - Malformations of maternal genital tract.

Criteria of the premature infant

- Weight < 2500 gm.
- Length < 45 cm.
- Skull circumference < 33 cm.

Physiologic handicaps and complications of premature infants:

The vast majority of body systems are physiologically immature. This will make the premature liable to the following complications:

1. Respiratory complications :
   - Hyaline membrane disease.
   - Apnea.
   - Respiratory infections.

2. Hyperbilirubinemia and kernicterus. 3. Neonatal infections .
4. Hypothermia. 5. Intra –cranial hemorrhage.
2. Retrolental fibroplasias. 7. Necrotizing enterocolitis.
8. Hypoglycemia. 9. Hypocalcemia
10. Nutritional deficiencies (rickets and iron deficiency)

Small for date baby
(Intrauterine growth retardation)

Causes

1. Placental dysfunction syndrome.
3. Congenital infections.
4. Congenital malformations.
5. Chromosomal disorders of the baby.
6. Maternal diseases affecting nutritional states of the fetus.

Criteria: Wasting of soft tissues and muscles.
The skin is thin loose and dry.

Management of L B W

1- Incubator care will provide:

- Regulation of body temperature.
- Regulation of relative humidity.
- Regulation of relative oxygen concentration.
- Minimize the possibility of infections.

2- Feeding:

- If breast milk is available and the baby is able to suck , breast feeding is the best .
- If breast feeding is not possible start formula feeding (LBW formula) using, bottle, spoon, dropper or even nasogastric tube.
- If enteral (oral) feeding is impossible use intravenous alimentation (Parenteral nutrition).
- Vitamin and mineral supplements

3- Prevention of infections:

In addition to the incubator care the following measures are needed:

- No or minimal visitors.
- Strict hand washing for those handling the preterm & LBW babies.
- Doctors and nursing staff should be free from infection and should wear masks.
- Proper isolation of the infected baby.
- Prophylactic antibiotics.
- Prophylactic immunoglobulin.

Neonatal convulsions

Definition: Twitching, rolling of the eyes, apnea or generalized tonic stiffness.

Causes

1- Hypoxic ischemic encephalopathy.
2- Intracranial hemorrhage.
3- Metabolic: Hypoglycemia, Hypocalcemia, Hypomagensemia, Hyponatremia – Hypernatremia and Pyridoxine dependency
4- Infections:- Sepsis, Meningitis and TORCH
5- Inborn errors of metabolism: Maple syrup urine disease and Methylmalonic acidemia
6- Drugs: theophylline, corticosteroids.
7- Hydrocephalus.
8- Polycythemia

Causes according to the age:

1- At birth→ a - maternal anesthetic agents
b - Hyponatremia.
c - Water intoxication
2- Day l→ acute metabolic abnormalities

3- Day 2-3→ a - meningitis
b - Drug withdrawal
4- Day ≥ 5→ a - Congenital infection (TORCH).as Toxoplasmosis – Rubella- Cytomegalovirus, Herpes & Other.
b - Developmental defects.
c – Galactosemia.
d – Hypocalcaemia.
5- > 1- 2 wks→ - addict mother after withdrawal e.g. opiates

Clinical picture and classification:

1- **Focal** (rhythmic twitching of muscle groups of extremities and face) usually due to local structural lesions, infections and subarachnoid hemorrhage.
2- **Multi focal clonic,** as before but include many muscle groups.
3- **Tonic** (rigid extremities and trunk ± fixed deviation of the eyes).
4- **Myoclonic** (brief focal or generalized jerks of distal muscle groups of the extremities or body).
5- **Subtle** (chewing, excessive salivation, apnea, blinking, nystagmus, bicycling, pedaling, changes in color and tongue thrusting) especially in premature.

Jitteriness is startle response to minor physical stimuli or loud noises.

1. Polycythemia: partial plasma exchange.

NEONATAL RESPIRATORY DISORDERS (RD)

Introduction:

Respiratory disorders are responsible for a major percentage of neonatal morbidity and mortality, especially in preterm infants.

Respiration involves:

-Air ways, Lungs & Muscles of respiration (Diaphragm, Chest wall).

-Control of respiration involves: *Brain* (Respiratory center), *Sensors* (chemoreceptors) that respond to hypoxia and hypercarbia, and *Nerves* that conduct impulses to and from these structures.

Clinical manifestations of RD are:

- Tachypnea (R.R. ≥ 60 breaths/min)
- Retractions (Use of accessory muscles of respiration)
- Grunting (self-induced CPAP).
- Cyanosis (failure of the compensatory mechanisms)
- Apnea (may be primary or secondary).

It is better to use R.D. scoring system (e.g. Downe's score) for accurate assessment and follow up.

Etiology of RD:

A: Air ways Obstruction

B: Lungs:

Extrapulmonary causes of RD.

C: CNS disorders: (HIE, ICH, Infection)
D: CVS: (Heart failure, PDA, VSD)
E: Hematologic: (Anemia, Polycythemia)
F: Metabolic:
1 Hypothermia & hyperthermia
2 Hypoglycemia

3 Hypocalcemia
4 Infection (Septicemia)

HYALINE MEMBRANE DISEASE (RESPIRATORY DISTRESS SYNDROME, RDS)

RDS is the most common respiratory disorder in neonates.
RDS occurs primarily in preterm infants (< 35 wk).

Risk Factors

Increased Risk	Decreased Risk
- Prematurity (< 35 wk)	- Chronic intrauterine stress
- C.S.	- Maternal hypertension
- Infants of diabetic mothers	- IUGR
- Male sex	- Narcotic use
- Familial predisposition	- Maternal CPD
- Perinatal asphyxia	- Tocolytic agents
- Multiple pregnancy (2^{nd} twin)	- Prenatal corticosteroids
- Hydrops fetalis	- PROM

Etiology & Pathophysiology

- RDS is due to surfactant deficiency (a surface tension reducing substance secreted by type II alveolar cells that prevents collapse (atelectasis) of the alveoli and small air spaces during expiration).
- Surfactant is a phospholipid complex consists mainly of phosphatidylcholine (lecithin), phosphatidyl-glycerol, cholesterol and surfactant proteins A, B, C, D.
- Surfactant deficiency leads to progressive alveolar collapse (atelectasis) during expiration and poor lung compliance during inspiration (stiff lung).

Clinical Manifestations:

- Signs of RDS usually appear within few hours (2-4 hr) after birth: tachypnea, intercostal and subcostal retractions, nasal flaring, grunting, cyanosis on room air, diminished breath sounds and air entry, and fine rales may be heard posteriorly over lung bases.
-The natural course of RDS is characterized by progressive worsening of cyanosis and dyspnea, peaks at 12-36 hr, and starts to resolve at 72 hr.

X-RAY CHEST

ARTERIAL BLOOD GAS (ABG)

Treatment:

A: Prevention:

- Adequate antenatal care for early identification and proper management of high risk pregnancies (DM, Hypertension) to prevent premature labor.
- Ante-natal Betamethasone (12mg IM) for expected premature delivery.

B: Supportive:

1 Thermal regulation (incubator care).
2 I.V. fluids & nutrition.
3 Medications (Antibiotics).
4 Correction of acid-base balance.
5 Maintain adequate tissue perfusion (fluid resuscitation, dopamine).
6 Monitoring of vital signs, pulse oximetry & ABG.
7

C: Oxygen therapy and respiratory support:

D: Specific therapy

- Surfactant replacement (Survanta, Exosurf).

- It is administered through the endotracheal tube.
- It is used either prophylactic (before lung injury) or rescue therapy.

TRANSIENT TACHYPNEA OF THE NEWBORN (TTN)

It is a benign disease of term or near-term infants. It is due to delayed absorption of lung fluids. It is a state of transient pulmonary edema.

ASPIRATION SYNDROME (MAS)

-It is a severe disorder secondary to meconium aspiration by the newborn either in utero or more often with the first breath.
-The aspirated meconium can cause airway obstruction (thick meconium), and/or an intense inflammatory reaction (thin meconium).

PNEUMONIA

Congenital Pneumonia:

-Ingestion and/or aspiration of bacteria in amniotic fluid lead to congenital pneumonia or septicemia with manifestations apparent prior to delivery (fetal distress, fetal tachycardia), at delivery (perinatal asphyxia, low Apgar score), or after a latent period of few hours (respiratory distress, shock, hypothermia, jaundice...).

-Risk factors (PROM, chorioamnionitis, maternal UT infection, premature labor, Endotracheal intubation, Umbilical vessel catheterization).

Postnatal-acquired Pneumonia:

-Infection is acquired from the care-givers or contacts harboring the organisms (by direct contact or droplet infection).

Aspiration Pneumonia:

-History of chocking and vomiting followed by cough and dyspnea.
-Risk factors (cleft lip & palate, TOF, GORD, bulbar palsy)

Clinical manifestations:

- Infants with pneumonia present with signs of RD (tachypnea, intercostal and subcostal retractions, nasal flaring, grunting, and cyanosis).
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APNEA

Definition: Cessation of breathing for ≥ 20 seconds usually accompanied by cyanosis and bradycardia (HR < 100/min.).

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II: Apnea of Prematurity (Primary or Idiopathic):

- It occurs in the absence of any identifiable predisposing disease.
- The onset of apnea occurs on the 2^{nd} -7^{th} day of life.
- Immaturity of the brain stem respiratory center is manifested by an attenuated response to CO_2 and a paradoxical response to hypoxia.

N.B.: In preterm infants, the onset of apnea during the 1^{st} day, or after the 2^{nd} week of life or in term infants at any time denotes a pathological apnea.

NEONATAL CYANOSIS

Definition: Bluish discoloration of the skin and mucus membranes.

Central Cyanosis is seen in the tongue and lips, and is also seen allover the body. It usually improves with O_2 and is associated with clubbing.

Peripheral Cyanosis is seen in the hands, feet and ears, due to peripheral arteriolar constriction. It improves with warmth and no clubbing.

Neonatal Hematology

Normal values (in the first few days of life)

- Blood volume 80-85 ml/kg - Hb 15-19 gm% - Hct 45% : 60 %
- MCV 95—110 fl - RBCs 5-6 millions/mm^3
- Reticulocytes 5-7 %

- WBCs 9,000-25,000/mm^3, Neutrophils constitute 60 %
- Platelets 150-250,000/mm^3
- Total serum bilirubin: cord < 2.5 mg/dl
  24 hr < 7.0 mg/dl
  2-6 days < 12.0 mg/dl

7 days < 7.0 mg/dl

Neonatal Anemia

Definition: Hematocrite (Hct) < 40- 45 % (0.40-0.45).

Causes:

A: Hemolysis:

1- Feto-maternal blood group incompatibility: Rh, ABO, and minor blood groups (e.g. C, E, Kell, Duffy).
2- Spherocytosis and other abnormalities of red cell morphology.
3- Glucose -6- phosphate dehydrogenase deficiency and other red cell enzymes deficiencies.
4- Hemoglobinopathies, e.g. α- and γ- thalassemia syndromes.
5- Infections, e.g. Bacterial, Viral (TORCH).
6- Drugs, e.g. penicillin, cephalosporins, vitamin K3.

B: Blood loss:

1- Obstetric causes: placenta previa, abruptio placenta, incision of placenta at C.S, hematoma or rupture of cord.
2- Fetōmaternal transfusion.
3- Fetōfetal, (twin-to-twin) transfusion.
4- External bleeding, e.g. bleeding from umbilicus, bleeding after circumcision.
5- Internal bleeding, e.g. cephalhematoma, subgaleal hemorrhage, adrenal hemorrhage, ICH, pulmonary hemorrhage, hepatic subcapsular hemorrhage, GIT hemorrhage.
6- Repeated blood sampling.

C: Diminished RBCs production (rare):

1- Diamond-Blackfan syndrome
2- Congenital leukemia..
3- Infections, e.g. parvovirus B19, rubella
4- Osteopetrosis
5- Drug-induced bone marrow depression
6- Physiolologic anemia or anemia of prematurity.

Clinical features:

1- Acute blood loss manifests with pallor and shock, with hypotension, poor tissue perfusion, and acidosis.
2- Chronic blood loss manifests with pallor, irritability, and poor weight gain, and if severe enough may manifest with tachypnea, tachycardia, and tender liver (HF).
3- Hemolysis is associated with pallor, jaundice, and hepatosplenomegaly.

Investigation:

1- Complete blood count: ↓ Hb, ↓ Hct, ↓ RBCs count.
2- Reticulocytes: ↑ in hemolysis and acute blood loss, ↓ in diminished RBCs production.
3- RBCs morphology, e.g. spherocytosis, and fragility test.
4- Total and differential WBCs, platelets count, and CRP for infection.
5- Infant's and mother's blood groups, and coomb's test.
6- G6PD assay.
7- TORCH screen if suspected.

Treatment:

1- Packed RBCs transfusion (15-20 ml/Kg) over 2 hours, if the baby has Cardio-pulmonary Compromise, (e.g. respiratory distress, congenital heart disease), or severe anemia Hct < 30 %.
2- Partial exchange transfusion using packed RBCs, in severely anemic infants, when routine transfusion to correct anemia, would result in circulatory overload.
3- Iron-fortified formula (2-4mg/Kg/day), if they are not breast-fed.

4- Vitamin E (15-25 IU day, water-soluble) is given to preterm infants until the baby is 38-40 wk post-conception.
5- Recombinant human erythropoietin offers a promise in anemia of prematurity.

Polycythemia

Definition: Hct ≥ 65%, (≥ 0.65).

Causes:

A: Passive polycythemia

1- Delayed cord clamping.
2- Materno-fetal transfusion.
3- Twin-to-twin transfusion.

B: Active polycythemia (increased erythropoiesis secondary to chronic intra-uterine hypoxia):

1- Placental insufficiency (IUGR, SGA)
2- Maternal hypertension (pregnancy-induced, preeclampsla, renal, essential)
3- Maternal hypoxemia (pulmonary disease, heart disease)
4- Large for gestational age (IDM, constitutional LGA).
5- Beckwith-Wiedmann syndrome.
6- Trisomy syndromes (21, 13, and 18).
7- Congenital cyanotic heart disease**,** (e.g. TGA)

Clinical Features:

- All polycythemic infants appear plethoric.
- Most polycythemic infants are asymptomatic.
- Clinical symptoms and signs of polycythemia / hyperviscosity include:

- General: plethora, cyanosis, jaundice
- CNS: lethargy, jitteriness, tremors, seizures, hypotonia, cerebral venous thrombosis.
- Cardiopulmonary: cyanosis, tachypnea, tachycardia, cardiomegaly heart murmurs, congestive heart failure, increased vascular markings on chest x-ray.
- Renal: decreased glomerular filtration, decreased sodium excretion, renal vein thrombosis, hematuria, proteinuria.
- GIT: poor feeding, NEC.
- Hematopoietic: jaundice, thrombocytopenia, DIC (purpura and bleeding tendency), other thromboses (priapism, testicular infarcts)
- Metabolic: hypoglycemia, hypocalcaemia.

Treatment:

- All symptomatic polycythemic infants should have a partial exchange transfusion using fresh frozen plasma, normal saline or albumin 5% solution, that will bring Hct to 50-60%.
- Volume of exchange= Blood volume x (observed Hct- Desired Hct) / observed Hct.
- Treatment of complications accordingly.

Hemorrhagic Diseases of the Newborn (HDN)

Definition: It is a bleeding disorder due to deficiency of vitamin K dependent clotting factors II, VII, IX and X.

Vit. K is synthesized by the intestinal bacterial flora. Because the newborn's intestine is sterile, and bacterial colonization is delayed by breast-feeding, HDN is common among newborns especially breast-fed ones.

Clinical features:

A) Classic type: it usually manifests after the 2nd day of life to the 5th day with GIT bleeding (hematemesis, melena, bleeding per rectum), bruises, bleeding from nose, mouth, venipuncture sites, heel sticks and circumcision.

B) Early-onset type: it manifests with bleeding tendency on the first 24hr of life.

- The mothers have received phenytoin, phenobarbital, salicylates, warfarin, rifampin, isoniazide during pregnancy.
- Hereditary clotting factors deficiency.

C) Late-onset type: It manifests with bleeding tendency after the 1st week of life (1-6 mo).

- It occurs in infants with malabsorption, cholestasis, biliary artesia, hepatitis, cystic fibrosis.
- Intracranial hemorrhage is common.

Diagnosis:

- Complete blood count: usually within normal range, except if bleeding is severe, normocytic normochromic anemia with mild reticulocytosis is present.
- Bleeding time is normal
- Clotting time, PT, and PTT are prolonged.

Treatment:

- **Prevention:** All newborn infants should receive vit. K□, 1mg, IM at birth.
- **Therapy:**
- Slow infusion of 1-5mg vit. K usually stops bleeding within few hours.
- Infusion of 10 ml/kg Fresh Frozen Plasma, may be required for serious active bleeding, and may be repeated every 8-12 hours, if needed.
- Packed RBCs, or fresh whole blood transfusion to correct anemia.

NEONATAL HYPERBILIRUBINEMIA (NH)
NEONATAL JAUNDICE (NJ)

Introduction:

NH is a biochemical term indicates elevated total serum bilirubin (TSB) >1 mg/dL.
NJ is a clinical term denotes yellowish discoloration of sclera and skin. Adults appear jaundiced when TSB > 2 mg/dL and newborns appear jaundiced when TSB >7mg/dL.
NH is a common and in most cases benign problem. Because neonates have an increased bilirubin production and decreased bilirubin secretion, 60% of term and 80% of preterm infants develop jaundice during the first week of life.
A small fraction of neonates who develop marked indirect hyperbilirubinemia are at risk to develop neurologic sequelae, bilirubin encephalopathy (Kernicterus), but direct hyperbilirubinemia indicates serious hepatic or metabolic disorders.

Diagnosis

History:

- Family history of jaundice, anemia, splenectomy, early gallbladder disease, liver disease may suggest chronic hemolytic anemia or metabolic disease.
- Previous sick sibling with jaundice or anemia may suggest blood group incompatibility or breast milk jaundice.
- Maternal illness during pregnancy may suggest congenital infection (TORCH). Infants of diabetic mothers tend to develop jaundice.
- Maternal drug intake during pregnancy (e.g. Sulphonamides, Nitrofurantoin) may initiate hemolysis in G_6PD deficient infants or displace bilirubin from albumin binding sites.
- Delivery history may show trauma, vacuum extraction, oxytocin infusion, delayed cord clamping and asphyxia, all are associated with increased incidence of hyperbilirubinemia.
- Infant's history of poor caloric intake, vomiting and decreased stools & urine suggest increased enterohepatic re-circulation of bilirubin.

- Breast feeding may lead to hyperbilirubinemia either secondary to decreased enteral intake (early) or due to breast milk inhibitory hormone (late).

Physical Examination:

- Gestational age (prematurity)
- SGA (Polycythemia, congenital infections).
- Microcephaly, chorioretintis, (congenital infections).
- Extravascular blood (bruises, cephalhematoma, other enclosed hemorrhages).
- Pallor (hemolytic anemia, extravascular blood).
- Petechiae (infections, sepsis, erythroblastosis fetalis).
- Hepatosplenomegaly (hemolytic anemia, infections, liver disease)
- Prolonged jaundice (hypothyroidism).

Investigations:

- Total and direct serum bilirubin
- Complete blood count (Hb, Hct, RBCs morphology, Retics, WBCs and platelets.
- Blood type and Rh (for both mother and baby)
- Coombs test (for both the mother and the baby)
- Sepsis screen (Cultures, CRP).
- Liver function profile (increased CB, cholestasis).
- TORCH screen (specific IgM & IgG).
- Screen for chronic hemolytic anemia (G6PD assay, osmotic fragility test)

PHYSIOLOGICAL JAUNDICE

A) Mechanism:

1)Increased RBCs volume/kg and decreased RBCs life span (90-day vs 120-day) lead to increased RBCs destruction and increased bilirubin production.

2)Increased enterohepatic circulation due to high levels of β-glucuronidase, decreased enteral intake, decreased intestinal bacteria, and decreased gut motility.

3)Decreased conjugation due to decreased activity of UDPG-T.

B) Characteristics:

-In Full Term: it appears on day 2-3, peaks on day 4-5, and disappears on day 6-8. Peak level is < 12 mg/dL.

-In Preterm: it appears on day 3-4 and, peaks on day 6-8, and disappears on day 10-12. Peak level is < 15 mg/dL.

-Rate of bilirubin rise is < 0.2 mg/dL/hr or < 5 mg/dL/24 hr.

-On examination: no any abnormal sign (poor feeding, lethargy, hypothermia….).

-Unconjugated hyperbilirubinemia.

PATHOLOGICAL JAUNDICE (Characteristics):

1. Clinical jaundice on the first 24 hr of life.
2. Clinical jaundice on day ≥ 14.
3. TSB more than l2 mg in full term or more than 15 mg in preterm infants.
4. A rate of rise in TSB > 0.2 mg/dL/hr, or > 5 mg/dL/24hr.
5. Sings of an underlying illness (e.g. lethargy, poor feeding, apnea, tachypnea, temperature instability, vomiting, hepatosplenomegaly, bleeding ...).
6. Direct hyperbilirubinemia, direct bilirubin ≥ 2 mg/dL or ≥ 20 % of TSB.

Late Breast Milk Jaundice:

-By day 6-8, bilirubin level continues to rise instead of the normal decline. It may reach 20-30 mg/dL, and starts to decrease by 4 weeks of age gradually returning to normal.
-Stopping breast feeding leads to a rapid fall in serum bilirubin within 48 hrs, may be the only therapeutic and diagnostic test.
-It is different from **EARLY BREAST-FEEDING JAUNDICE** which is occurring on day 2-4, and it is mainly due to decreased enteral intake, both in volumes and calories, and increased enterhepatic re-circulation of bilirubin.

l Blood Group Incompatibility (Hemolytic Disease of the Newborn, Isoimmune Hemolytic Disease, Erythroblastosis Fetalis):

A) Rh-incompatibility:

It occurs when an Rh-negative woman marry Rh-positive husband and the fetus is Rh-positive. During pregnancy, abortion, delivery, and trauma (amniocentesis, chorionic villus sampling) fetomaternal transfusion occurs and the maternal immune system recognizes Rh-positive fetal RBCs (containing D-antigen) as a foreign antigen forming anti-D antibodies (maternal sensitization). During the 1st pregnancy (1st exposure to Rh-positive RBCs) anti-D antibodies are of IgM class which cannot cross the placenta and the infant is not affected. During the 2nd pregnancy (re-exposure) anti-D antibodies are of IgG class which crosses the placenta and cause hemolysis in the fetus and the newborn. In case of severe hemolysis, fetal anemia and anemic heart failure with generalized edema may develop, Hydrops Fetalis (treated with intrauterine fetal RBCs transfusion). In case of mild to moderate hemolysis early neonatal hyperbilirubinemia will develop.

B) ABO-incompatibility:

It occurs when a woman with blood group "O" marry husband with blood group "A, B, or AB" and the fetus has a blood group "A or B". Mothers with blood group "O" have in their sera, naturally occurring, anti-A and anti-B antibodies of IgM (major fraction) and IgG (small fraction) which cross the placenta and cause mild to moderate hemolysis in the fetus and the newborn, affecting the 1st pregnancy (no need for maternal sensitization).

Neonatal Hypoglycemia

Diagnosis

- Blood glucose <40 mg/dL regardless of gestational age or clinical condition is too low
- Neonates with hypoglycemia often display no symptoms
- Symptoms—lethargy, irritability, jitteriness, poor feeding, seizures, ↓level of consciousness, coma
- Increased risk—infants of diabetic mothers, infants with intrauterine growth retardation, stressed infants, preterm infants

Differential Diagnosis

- Genetic hyperinsulinism
- Inborn errors—galactosemia, glycogen storage diseases
- Endocrine—hypopituitarism
- Complication of birth asphyxia
- Complication of sepsis

Treatment

- Blood sugar <40 mg/dL with symptoms—start IV within 10 minutes

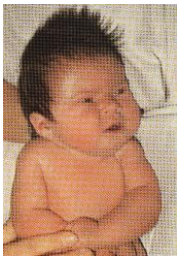
- Blood sugar <40 mg/dL without symptoms—feed and repeat glucose and give 2 mL/kg of 10% dextrose bolus followed by3.6 mL/kg/h constant infusion. Monitor glucose within 30 minutes
- Blood sugar <20 mg/dL with/without symptoms—start IV within10 minutes and give 2 mL/kg 10% dextrose bolus followed by3.6 mL/kg/h constant infusion. Monitor glucose within 30 minutes

Infants of Diabetic Mothers (IDM)

Maternal diabetes may be pre-existing (before pregnancy) or gestational (during pregnancy). Tight glycemic control during the preconceptional period and throughout pregnancy, (using insulin), is paramount to decrease fetal and neonatal complications.

Maternal diabetes may adversely affect the fetus and the newborn:

- Congenital anomalies (cardiac, neural tube, renal, vertebral and skeletal malformations)
- Hypoglycemia (plasma glucose level < 40 -45 mg/dL)
- Hypocalcemia,
  Serum tCa < 7mg/dL (< 1.75 mmol/L) or iCa < 4.4 mg/dL (< 1.1 mmol/L)
- Hypomagnesemia, serum Mg < 1.5 mg/dL (< 0.62 mmol/L)



- Abortion
- Still birth
- Preterm delivery
- Macrosomia
- Birth Trauma
- RDS
- Hyperbilirubinemia
- Polycythemia
- Renal vein thrombosis
- Small left colon syndrome (with delayed passage of meconium, meconium blug)

Management:

- IDM is a high risk newborn, thus delivery should be carried out at a hospital equipped for neonatal care.
- Resuscitation and stabilization of the newborn.
- Monitor blood sugar level at 1,2,3,6,12,24,36 and 48 hours after birth.
- Treatment of hypoglycemia (blood sugar level < 40 mg/dL). Bolus of IV dextrose 10% 2-4 ml/kg/min
- Treatment of hypomagnesemia: Bolus $MgSO_4$ 50% 0.1-0.2 mL/kg (50-100 mg/kg) IM.
- Complete physical examination and follow up searching for complications of IDM and treat accordingly.

NEONATAL INFECTIONS

Infections are a common cause of neonatal morbidity and mortality.

Neonatal infections are classified according to the timing of exposure:

A) Intrauterine infections (Transplacental, Congenital):

TORCH infections

T= Toxoplasmosis

O= Others (Syphilis, Varicella, Parvovirus B19)

R= Rubella

C= Cytomegalovirus, CMV

H= Herpes simplex

Consequences and clinical manifestations of intrauterine infections:

- Early spontaneous abortion/ late stillbirth
- Congenital malformations
- Premature delivery
- IUGR/SGA
- Petechiae, purpura, bleeding tendency
- Hepatosplenomegaly
- Jaundice/ pallor
- Microcephaly/ cranial calcifications
- Chorioretinitis
- Developmental delay
- Hearing loss

- Congenital Rubella Syndrome (cataract, hearing loss, congenital heart disease: PDA/ PS, microcephaly, microphthalmia, retinopathy, glaucoma, meningo-encephalitis, HSP, thrombocytopenic purpura, DM)

- Congenital Parvovirus B19 infection (RBCs aplasia, severe anemia, CHF, edema, nonimmune Hydrops Fetalis)

Diagnosis:

- Serum TORCH titers (specific IgM & IgG against each infectious agent).
- Rising IgG titer (4 fold rise in a 2-4 wk interval).
- Identification of the virus in clinical specimens (urine, saliva, CSF).
- Assessment of the different system organs functions.

Treatment:

- Supportive treatment.
- Hyperimmune specific immunoglobulin & IVIG.
- Specific treatment, according to the infectious agent (Ganciclovir for CMV, Acyclovir for Herpes &Varicella, Spiramycin, Pyrimethamine, & sulfadiazine for Toxoplasmosis, Penicillin & Ceftriaxone for Syphilis).

B) Intrapartum (Intranatal) infections:

I- Viral: HSV, HIV, HBV, HCV
II- Bacterial: Group B Streptococcus, E coli, Listeria monocytogenes
III- Mycoplasma & Chlamydia
IV- Fungal (Candida)

C) Postpartum (Postnatal) infections:

I- Viral: HIV, CMV
II- Bacterial: Coagulase-negative Staph., Staph. Aureus, E coli, Klebsiella, Pseudomonas, TB (infected mother)
III- Mycoplasma & Chlamydia
IV- Fungal (Candida)

Neonatal Bacterial Sepsis (Septicemia)

It is a clinical syndrome of symptoms and signs due to the presence of bacteria and its toxins in the blood stream and /or other body fluids and tissues.

Risk Factors:

A) Maternal Risk Factors:

1- Premature rupture of membranes ≥ 18 hrs.

2- Choriamnionitis (intrapartum fever ≥ 38°C, foul vaginal discharge, uterine tenderness, leukocytosis, increased CRP.

3- Premature delivery.

4- Maternal UT infection.

B) Neonatal Risk Factors:

1-Preterm & low birth weight newborns.

2- Invasive procedures: IV catheters, endotracheal tube, chest tube.

3-Congental defects: meningocele, TOF, obstructive uropathy, cleft lip & palate.

4-Male sex

C) Environmental Risk Factors:

1- Contaminated place, equipments, and personnel.

2- No adherence to infection control measures (nosocomial infection).

Pathogens:

Causative organisms differ according to the pattern of infection:

I-Early-onset sepsis (first 3 days) intrapartum infection is acquired from the mother

Group B Streptococcus, gram-negative enterococci, E coli, Listeria monocytogenes

II-Late-onset sepsis (> 3 days) postpartum infection is acquired from community

Coagulase-negative Staph., Staph. Aureus, E coli, Klebsiella, Pseudomonas

Clinical manifestations:

Symptoms are nonspecific and may be subtle (just not doing well), they include:

1- Sleeps more, awake little, decreased movements, little cry, poor feeding
2- Hypothermia or fever, pallor, jaundice, cyanosis, shock
3- Respiratory distress: apnea, tachypnea, retractions, grunting, cyanosis.
4- Tachycardia, bradycardia, Hypotension, poor perfusion, HF.
5- Poor feeding, vomiting, diarrhea, distension, ileus.
6- Oliguria
7- Pallor, jaundice, Petechiae, purpura, bleeding tendency, hepatomegaly, splenomgaly.
8- Lethargy, irritability, seizures, hypotonia, hyporeflexia, diminished Moro & sucking reflexes, bulging fontanel, neck rigidity, high pitched cry.

Investigations:

1- Chest X-ray: (Pneumonia)

2- ABG: Metabolic acidosis

3- CBC: Leucocytosis > 20,000/cmm or Leucopenia < 5000/cmm, Neutropenia <1500/cmm or Neutrophilia >8000/cmm, I/T > 0.18, Thrombocytopenia, platelets < 150,000/cmm, Anemia, Hct < 40%.

4- C-reactive protein (> 0.6 mg/dl).

5- Cultures: blood, CSF, urine, local sites of infection.

6- Check blood glucose and serum electrolytes for any abnormality.

Treatment:

1- Start IV antibiotics after collection of samples for cultures.