BABY AT RISK

COURSE OBJECTIVES

By the end of the course, the student should be able to:

1. Describe the normal physiology of a newborn
2. Describe the resuscitation of a newborn
3. State the indications for admission to special care baby unit (SCBU)
4. Describe the set-up of the SCBU
5. Describe Low birth weight babies, Preterm and light for dates
6. Identify clinical presentations of respiratory distress syndrome and asphyxia neonatorum

7. Describe the pathophysiology of rhesus incompatibility and jaundice
8. Identify the distinguishing features in various congenital malformations
9. Identify the etiology and pathophysiology of

neonatal sepsis
10. Identify the features and diagnostic findings in caput succedaneum and cephalohematoma

11. Describe fetal alcohol syndrome, etiology, and features
12. Identify the various types of intracranial hemorrhage
13. Describe various injuries of the newborn including nerve injuries and bone fractures
14. Discuss the nursing management of various conditions of the newborn
15. Outline the diagnostic approaches for various conditions in a newborn

EVALUATIONS

1. Pre-block quiz
2. Mid block Quiz
3. End block exams
4. Assignments

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NORMAL PHYSIOLOGY OF THE NEWBORN

On leaving the intrauterine environment, the baby's basic needs are to:

- establish respiration and subsequently adequate nutrition
- maintain normal body temperature
- avoid contact with infection.

NEWBORN REFLEXES

- **Moro Reflex -** Support the child with your hand and forearm. Then gently but swiftly, tilt the child backwards. In normal circumstances, the child should outstretch their arms (and legs). This is a positive moro reflex.
- **Rooting reflex –** when you gently touch next to the baby's mouth, the baby will turn their head and try to suckle.

- **Grasp reflex –** place your finger in the baby's palm. The baby should grasp with a firm grip
- **Stepping reflex –** holding upright above the bed, and then gently brush the bed against the baby's feet / shins. The baby should step. They may also be able to support some of their own weight.
- **Others???**

GOALS OF NEWBORN NURSING CARE

a. To continue assessment of the newborn throughout hospital stay:
   (1) Observe and record the infant's vital signs.
   (2) Monitor weight loss or gain
   (3) Monitor bowel and bladder function.
   (4) Monitor activity and sleep patterns.
   (5) Monitor interactions and bonding with parents.

b. To provide safeguards against infection.

c. To initiate feedings.

d. To provide guidance and health instruction to parents.

PRINCIPLES OF ESSENTIAL NEWBORN CARE

- Resuscitate and maintain an airway
- Keep the newborn warm and avoid unnecessary hypothermia or cold stress
- Encourage early breast feeding, and feed high-risk newborns more frequently
- Maintain hygiene during delivery and cord cutting; treat infections promptly
- Ensure the newborn infant stays close to its mother, and mothers have open access to their newborn infant if he or she requires special care

IMMEDIATE CARE AND ASSESSMENT

- Observe infection prevention practices
- Deliver baby onto the mother’s abdomen
- Dry the baby and keep warm
- Assess baby’s breathing, skin color and tone
- Wipe eyes
- Clamp, tie and cut umbilical cord at 2 and 5 cm from the abdomen
- Initiate breastfeeding within one hour

APGAR

- **A**ppearance
- **P**ulse
- **G**rimace
- **A**ctivity
- **R**espiration

RESUSCITATION OF THE NEWBORN

- Resuscitation is an emergency measure taken to sustain life or to revive when life has just ceased. The aim is to establish the heart and lung function following cardio-pulmonary arrest.

INTRODUCTION

- **Preparation**
- Check and arrange equipment/supplies at all times (use equipment checklist) as follows:
- - A firm stable surface
- - Source of heat e.g. heater, heater lamp or resuscitaire
- - Adequate lighting
- - Source of oxygen, flow meter, tubing and key

- - Suction equipment i.e. suction machine, suction catheters sizes F6, 8, 10
- - Ambu bag (500mls)
- - Face masks sizes 0 and 1, preferably round
- - Wall clock
- - At least two pieces of warm dry linen
- - Syringes and needles/swabs, (preferably 1ml, 2ml and 10mls)

- - Stethoscope
- - Artificial Airways
- - Nasal prongs
- - Nasogastric tubes
- - Scissors and tape
-

THE DRUG TRAY

- should contain the following :
- Sodium bicarbonate 8.4% or 7.5% for correction of metabolic acidosis.
- Fifty percent dextrose for correction of hypoglacemia (survival of the newborn infant correlates with glycogen stores).
- Adrenaline if the heart rate is falling after several minutes of vigorous resuscitative efforts; it may be given directly by cardiac puncture and then external cardiac massage started. This drug is also a vaso-dilator.

- Aminophylline in small doses is given to aid in respiration if distress is present (3mg kg/body weight intravenously). This drug is a vaso-dilator and also stimulates the respiratory centre.
- Calcium gluconate can be administered intravenously and slowly to a baby who displays tetany due to calcium deficiency. It is also a cardiac muscle stabliliser.
- Source of oxygen to be administered after clearing the airway.

PRINCIPLES OF RESUSCITATION

ABCDO of resuscitation:

- **A**irway should be cleared either by suction or positioning the infant.
- **B**reathing, that is, establish respiration if not breathing.
- **C**irculation should be noted through pulse, color of mucus membrane and heart beat. Start cardiac massage if there is no heart beat or pulse.
- **D**rugs
- **O**bservations to make a diagnosis.

NEONATAL RESUSCITATION PROCEDURE

- **Temperature:**
- Dry the baby, remove wet clothing and wrap baby in dry warm clothes. At the same time observe the baby’s breathing, color and activity
- Place the baby on a firm warm surface or under a radiant heat warmer

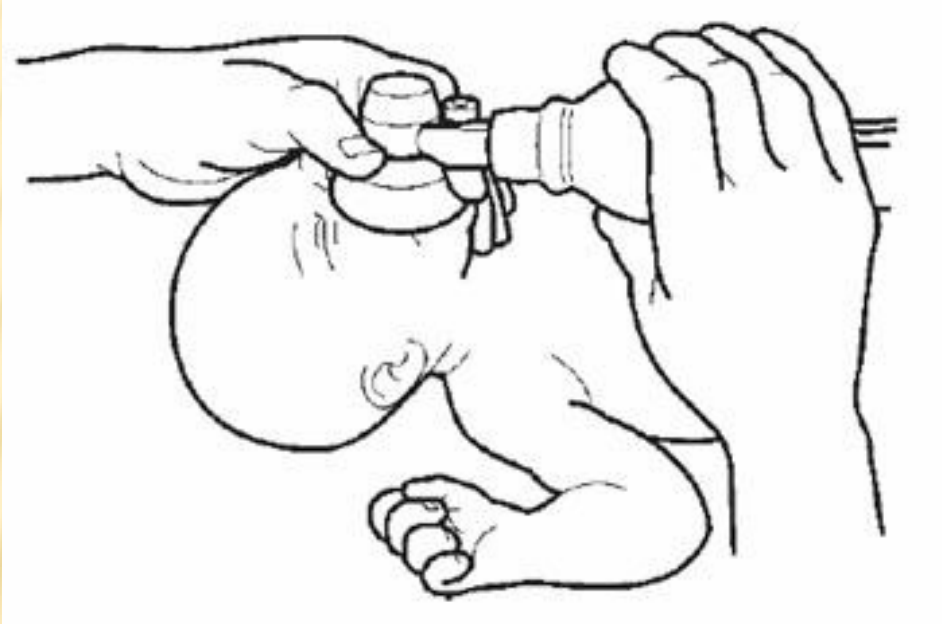
AIRWAY:

- Position baby’s head in a neutral position (slightly extended position) to open airway.
- Open airway
- Suction –If there is meconium stained fluid and baby is not crying and moving limbs (Do not suck for more than 3 seconds)
- - Suck only what you can see
- - Suck the mouth and then the nose
- - Suck only while withdrawing the catheter
-

VENTILATING WITH BAG AND MASK

- Place mask to cover chin, mouth and nose
- Form a seal
- Squeeze bag attached to the mask with two fingers or whole hand about 30 to 50 times per minute
- Watch chest for movement, do not over inflate, allow baby to breathe out. If chest is not rising:
- - Reposition the head
- - Check mask seal
- - Squeeze bag harder
- Start ventilation immediately using room air (bag and mask). If the baby does not improve then use oxygen

Pull the jaw forward towards the mask with the third finger of the hand holding the mask. Do not hyperextend the neck.



- **Circulation**
- Assess and count heart rate.
- **Chest compression**
- Using two fingers method, or thumb method compress the chest (1cm below the line connecting the nipples and the sternum) pushing down 1.5cm.
- Give 90 compressions coordinated with 30 breaths per minute (3 compressions for every breath).

NEONATAL EMERGENCIES

OVERVIEW

- Respiratory distress syndrome
- Asphyxia neonatorum

RESPIRATORY DISTRESS SYNDROME

LEARNING OBJECTIVES

- Explain the science behind RDS
- Be able to identify who is at risk of RDS
- State the signs of increased work of breathing
- Outline other causes of respiratory distress in a new born baby
- Discuss management of a baby with RDS

RESPIRATORY DISTRESS SYNDROME

- Also called the hyaline membrane disease
- This is a developmental disease of the preterm infant._
- **PATHOPHYSIOLOGY** – in RDS the alveolar and the ducts are filled with a sticky exudate, a hyaline material that prevents aeration.

- This could be due to lack of surfactant that reduces alveolar ventilation and promotes atelectasis.
- Reduction in alveolar surfactant may occur due to:-
- Immature cells lining the alveoli
- Decreased rates of production due to early neonatal stress
- Inadequate release of surfactant from the alveolar cells
- Death of cells producing surfactants.

SIGNS OF RDS.

- Increased work of breathing
  - Grunting
  - Respiratory rate >60
  - Cyanosis/low saturations
  - Recession: subcostal, intercostal, sternal
  - Head bobbing
  - Nostrils flaring
- Apneas
- Symptoms typically worsen over first 48-72h

- **Investigations:**
- Full hemogram / full blood count
- Blood culture
- Blood sugar
- X-ray where possible

TREATMENT

- Oxygen
- Intubation and ventilation
- Surfactant administration directly to the lungs
- Feeding support with NG feeds or IV fluids
- Antibiotics (incase respiratory distress due to infection)

NURSING CARE

- Administer oxygen using the appropriate delivery method
- If secretions are present use suction for brief periods using intermittent pressures
- Weigh the infant daily and monitor laboratory values as ordered
- For IV lines change tubing every 24hrs
- Provide adequate calories per kg body weight
- Prepare to administer surfactant replacement therapy (instilled into the endotracheal tube).

NURSING CARE CONT…

- Provide nutrition
- Support bonding
- Provide realistic information to the parents
- Observe and report any signs of complications

PREVENTIVE MEASURES TO RDS.

- Prevent preterm labour if possible
- Give dexamethasone to the mother at least 48 hours before delivery
- Prevention of hypothermia at birth
- Prevention of perinatal asphyxia
- Adequate resuscitation

COMPLICATIONS

- Respiratory – pneumothorax ,bronchopulmonary dysplasia
- C.V.S – hypotension, PDA,
- CNS – cerebral edema, intracranial hemorrhage, sepsis, renal failure

BIRTH ASPHYXIA

- Asphyxia means lack of oxygen
- Means that baby's organs don't get enough oxygen before, during or right after birth

WHERE CAN THE BABY SUFFER FROM ASPHYXIA?

- In Utero
- During Delivery
- After Birth

CAUSES OF ASPHYXIA

- Decreased placental flow e.g. cord prolapse
- Decreased placental gas exchange e.g. placental abruption, uterine rupture and placenta previa.
- Decreased placental perfusion
- Maternal hypoxia e.g. maternal seizure
- Inadequate postnatal cardiopulmonary circulation e.g. blocked airway

- Poor APGAR SCORE mostly indicates asphyxia and the infant goes into apnea.
- Poor placenta function that may occur with high blood pressure or in post-term pregnancies.

SIGNS AND SYMPTOMS

- Before delivery, symptoms may include:
- Abnormal heart rate or rhythm
- An increased acid level in a baby's blood
- At birth, symptoms may include:
- Bluish or pale skin color
- Low heart rate
- Weak muscle tone and reflexes

- Weak cry
- Gasping or weak breathing
- Meconium — the first stool passed by the baby — in the amniotic fluid, which can block small airways and interfere with breathing

ORGANS

- Brain – Hypoxic Ischemic Encephalopathy
- Kidneys – Poor urine output, Drugs e.g.. Gentamycin can have decreased clearance
- Liver – clotting problems
- Cardiac – impaired function
- Bowel ischemia

EXAMINATION

- Poor breathing effort
- Prolonged bleeding
- Poor urine output
- Neurological signs – depends on severity:
  - Tone – Increased (stiff) or decreased (floppy)
  - Level of consciousness – hyperalert , lethargic
  - Reflexes
  - Seizures
  - Poor suck and difficulty feeding
  - Lack of gag reflex

TREATMENT

- Immediate management:
- 1.Establish effective ventilation
- 2.Assist circulation if necessary
- Early management:
- 1.Restrict fluids by 20% for first two days
- 2.Monitor blood pressure and treat hypotension vigorously
- 3.Assess respiratory effort and
- (A)ventilate if baby breathing spontaneously with arterial carbon dioxide tension >7

- (b)if baby ventilated maintain arterial carbon tension at 4.5
- 4.If clinical signs of raised intracranial pressure give Mannitol 1g/kg over 20minutes and repeat if necessary every 4-6hours
- Anticonvulsants

ASPHYXIA NEONATORUM

OVERVIEW

Definition

Pathophysiology

Types of asphyxia

Signs and symptoms

Predisposing factors

DEFINITION

- Defined as the impaired respiratory gas exchange accompanied by the development of acidosis
- According to WHO, it is as a failure to initiate breathing at birth

PATHOPHYSIOLOGY

1. Anaerobic metabolism
2. Profound metabolic acidosis
3. Persistence of apgar score of 0 to 3 beyond 5 minutes
4. Clinical neurologic sequelae caused by Hypoxic ischemic encephalopathy (HIE)
5. Multiorgan system dysfunction in the immediate neonatal period

TYPES OF ASPHYXIA

- The degree of asphyxia is determined by apgar score.
- An apgar score between 8-10 does not show asphyxia
- There are three types:

1. **Mild asphyxia** apgar score: 6-7 (at 5 minutes). It requires clearing of airway and application of external stimuli to initiate breathing.
2. **Moderate asphyxia**: apgar score 4-5. it requires rescuscitation, administration of oxygen, and drugs to initiate breathing
3. **Severe asphyxia:** apgar score 0-3. requires intensive resuscitative measures and intubation to survive

CLINICAL FEATURES

- Apnea
- Bradycardia
- Altered respiratory patterns (grunting, Gasping)
- Cyanosis
- Pallor
- Hypotonia
- Unresponsiveness

1. Mild and moderate asphyxia

- Apex beat (pulse) 100/ min or less
- Skin color is pink with blue extremities
- Response to stimuli may be present
- Cry may be weak or strong
- Makes effort to breathe and may gasp with irregular respiration

2. Severe asphyxia

- No attempt to breathe and may gasp periodically
- It does not cry
- Entire body skin is blue i.e. cyanosed
- No response to stimuli
- Pulse rate very low or absent
- Poor muscle tone

PREDISPOSING FACTORS

Depression of the central nervous system of the fetus is the most common basic cause for failure to breathe at birth. However there are also maternal and fetal factors

Maternal causes:

- Medical conditions e.g. chronic hypertension
- Antenatal conditions e.g. abnormal uterine contractions, antepartum hemorrhage, prolapsed cord, malpositions etc.
- Drug abuse

Fetal causes:

- Multiple pregnancies,
- Big baby with C.P.D
- Fetal anomalies e.g. anomalies of the lungs

NURSING MANAGEMENT

1. Clear the airway as soon as the baby is born
2. Nurse the baby in an incubator for at least 48 hours to keep it warm at body temperature
3. Resuscitation may be needed to promote ventilation and ensure effective circulation to prevent acidosis, hypoglycemia and intracranial hemorrhage
4. Do suctioning whenever necessary
5. Closely observe the baby for skin color, TPR

6. Administer oxygen by mask, ambu bag or nasal catheter whenever there is an apneic attack.
7. Give iv fluids for rehydration
8. Aspirate mucus to unblock the airway or may intubate the baby
9. Give fluids with electrolytes to maintain fluid electrolyte balance
10. If the mother was given narcotics during labor, administer its antidote, naloxone, through the umbilical vein
11. Give anticonvulsants to control convulsions if present

12. Maintain accurate input output charts to prevent overhydration or underhydration
13. When the baby is stable, pass NG tube and start feeding
14. Observe aseptic technique to prevent cross infection
15. Administer broad spectrum antibiotics prophylactically

Drug administration

- Sodium bicarbonate 1-2 mls to combat acidosis
- Vitamin K 0.5- 1 mg i.m to prevent hemorrhagic disorders
- Aminophylline to improve respiration
- Calcium gluconate to strengthen heart muscles
- Administer broad spectrum antibiotics prophylactically

PREVENTION OF ASPHYXIA

- Proper screening of mothers to detect those at risk and advise on delivery in the hospital for proper management
- Pelvic assessment should be done at 36 weeks to rule out CPD
- Proper management of maternal diseases in pregnancy

- Drugs that depress respiratory centre e.g. sedatives, General anesthesia and narcotics should be avoided in late first stage.
- Early detection and management of fetal distress
- Clearing baby’s airway as soon as the head is born
- Avoiding instrumental deliveries but rather prepare for c/s

COMPLICATIONS

- Renal abnormalities (50%), e.g. azotemia, elevated creatinine levels, acute tubular necrosis
- Hypoxic Ischemic Encephalopathy (HIE)
- Brain damage
- Cardiac arrest
- Respiratory distress syndrome
- Respiratory acidosis

THE PREMATURE INFANT (PRETERM)

INTRODUCTION

- A preterm infant is a baby born before the **37th completed week** of gestation.
- This is most often due to the early over distension of the uterus, which leads to the fetus being born prematurely.
- Premature births also occur more frequently in low socio economic and illiterate groups in the population, largely as a result of the poor nutritional intake of the pregnant mother.
- Additionally, they tend to be more common when there is concurrent maternal metabolic or systemic disease, for example, hypertension and Diabetes.

CLASSIFICATION OF PRETERM

- WHO Classification is based on gestational age:
- Extremely preterm (< 28 weeks)
- Very preterm (28 to < 32 weeks)
- Moderate to late preterm (32 to <37 weeks)

PREDISPOSING FACTORS TO PREMATURITY

- **Fetal factors**
- Polyhydramnios which causes over distension
- Congenital malformation of the fetus
- Rhesus incompatibility interfering with fetal viability.
- Multiple births (twins or more), which causes early distension of the uterus hence early birth
- **Maternal factors**
- Hypertension associated with the pregnancy leading to early induction of labor, for example, preeclampsia and eclampsia
- Premature rupture of membranes due to physical or psychological stress

- Chronic infections or diseases in the mother, such as syphilis, tuberculosis, chronic nephritis, cardiac disease, diabetes and thyroid disease, may lead to premature labor
- Acute infections in the mother, like pneumonia, influenza, rheumatic fever and malaria could induce premature labor
- Strenuous exercise, excessive drinking of alcohol, smoking

- Physical stress caused by non obstetric surgery may lead to premature labor if the mother has this procedure while pregnant
- Habitual abortion owing to incompetent cervical OS or uterine malformation
- **Placental factors**
- Antepartum hemorrhage due to placentae praevia and placenta abruptio

PREVENTION OF PREMATURE BIRTH

1. Early and continued prenatal care with stress on dietary and general hygiene education to the expectant mother.
   - This will also ensure immediate treatment of those complications of pregnancy likely to cause, or be associated with, premature labor.

PREVENTION CONT…

2. postpone or inhibit uterine contractions in some cases of premature labor through the use of certain pharmaceutical agents such as:
   - Those that act by preventing the release or synthesis of a known uterine stimulant, for example, prostaglandin inhibitors e.g. . non steroidal anti inflammatory drugs like aspirin, indomethacin, diclofenac
   - Those that act by the direct effect on the myometrial cells, for example, beta adrenergic receptor stimulants like retodrine, fenoterol, salbutamol

PREVENTION CONT…

3. Prolonged bed rest should be encouraged, especially where the mother has any of the conditions that predispose to preterm labor
4. Use of sedatives during preterm labor to ensure complete bed rest
5. Avoidance of strenuous exercise and calming the mother, because any strain or stress may aggravate preterm labor

PHYSICAL CHARACTERISTICS IN PREMATURE INFANTS

General:

- the baby may appear pink or dark red, hands and feet may be cyanosed and may be jaundiced early.
- Posture: flattened, abducted hips, flexed knees
- Swallowing and sucking reflexes absent or weak

Skin and Appendages

- The skin is covered by a small amount of **vernix caseosa.**
- **Lanugo** is present on the sides of the face and on the extremities and the back.
- There is scanty hair on the head and the eyebrows are usually absent.

- The nipples and areola are inconspicuous.
- The nails are soft.
- Generalized edema is apparent at birth and later the tissue fluid decreases, leaving the skin loose and wrinkled.
- Blood vessels can be easily seen under the skin because subcutaneous tissue is thin.

- **Head**
- Skull bones: soft with large fontalles, wide sutures
- Ear pinna: flat, soft and fold easily on pressure and uncoil slowly
- Eyes are closed most of the time
- Weak cry and there is no tears produce

Neck and Thorax

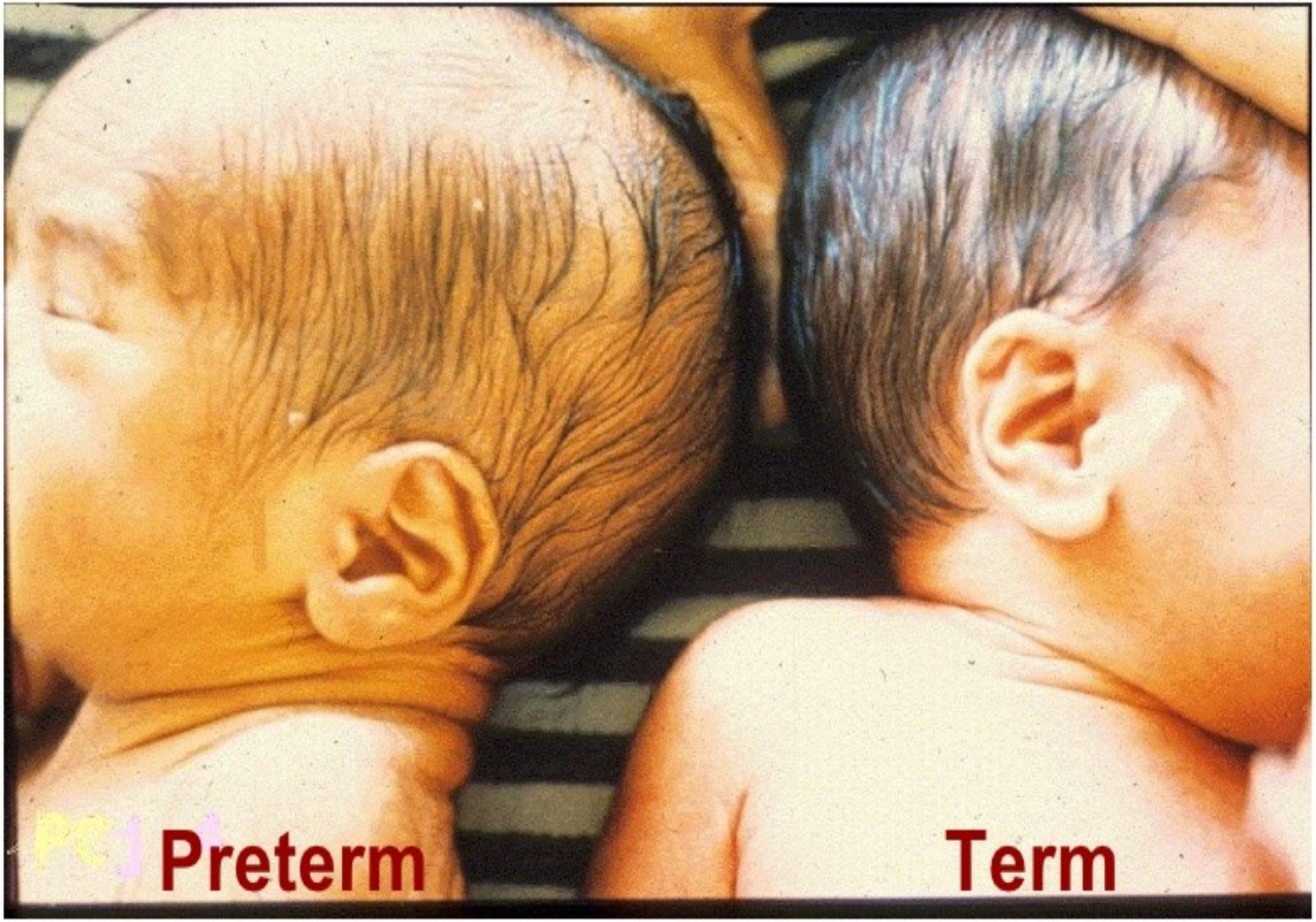
- The thorax is cone shaped
- sternal retraction,
- a rapid heart rate and respiratory noises.
- Occasionally, cardiac murmurs are heard.
- A change in position may cause periods of apnea.

- **Abdomen**
- Size: prominent, large liver and spleen
- Umbilicus: appears lower in the abdomen
- Cord: white, fleshy and glistening
- Plantar creases absent

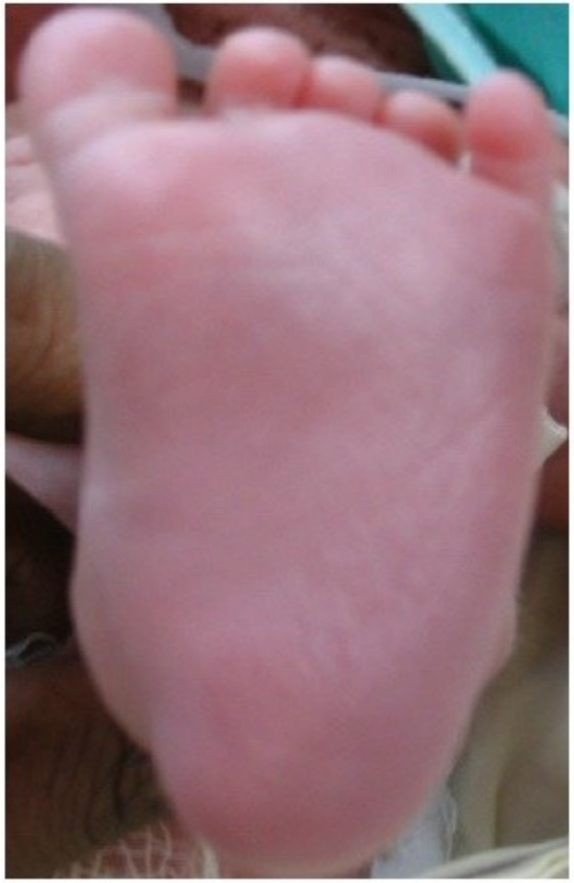
Genitalia

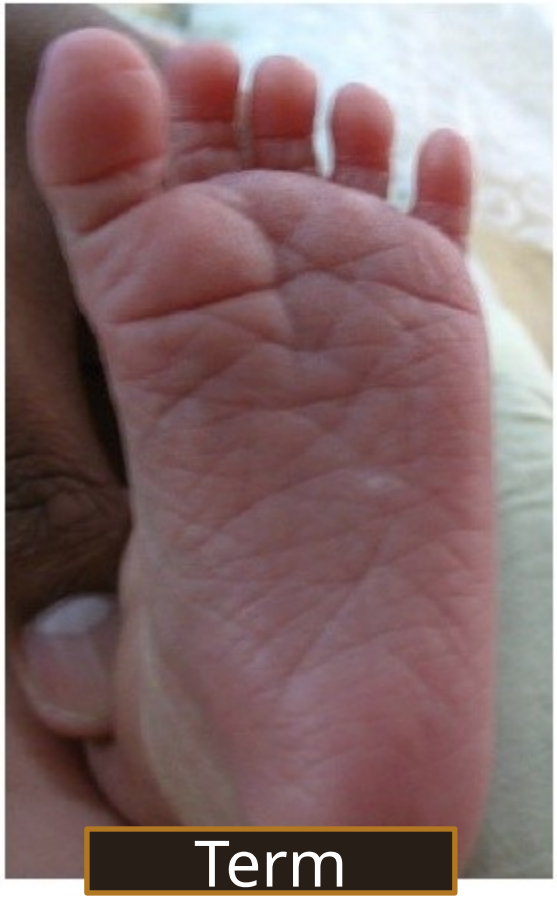
- Labia majora fail to cover labia minora in females
- Undescended testes in males

Ear Cartilage



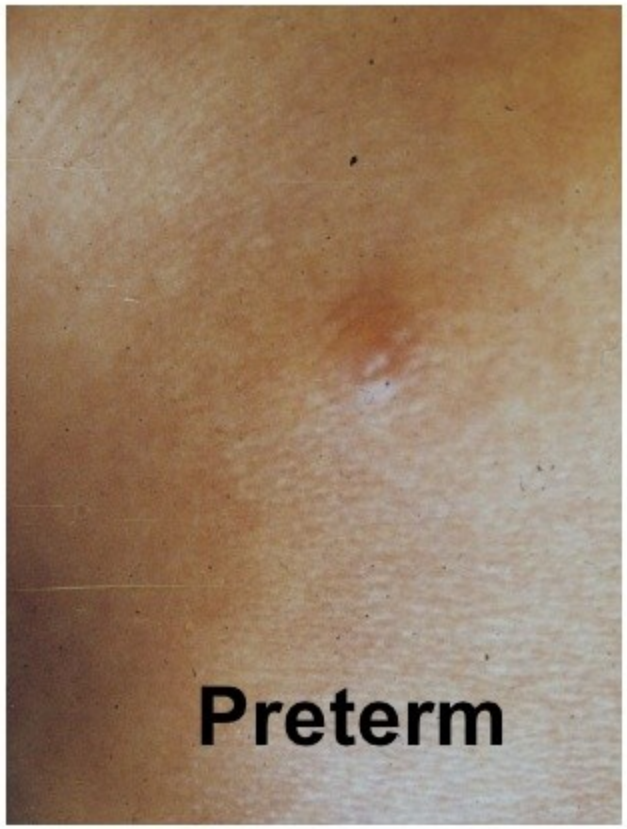
Preterm

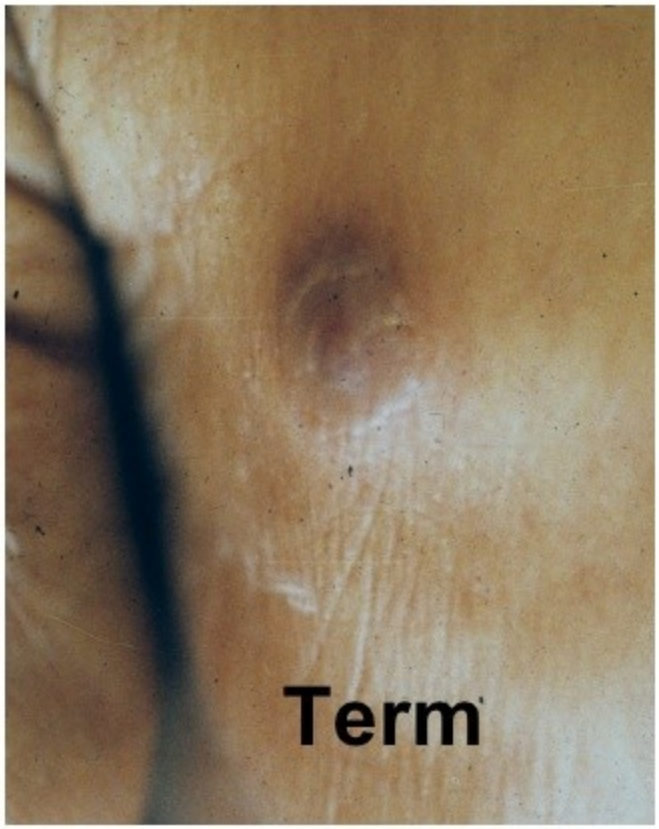




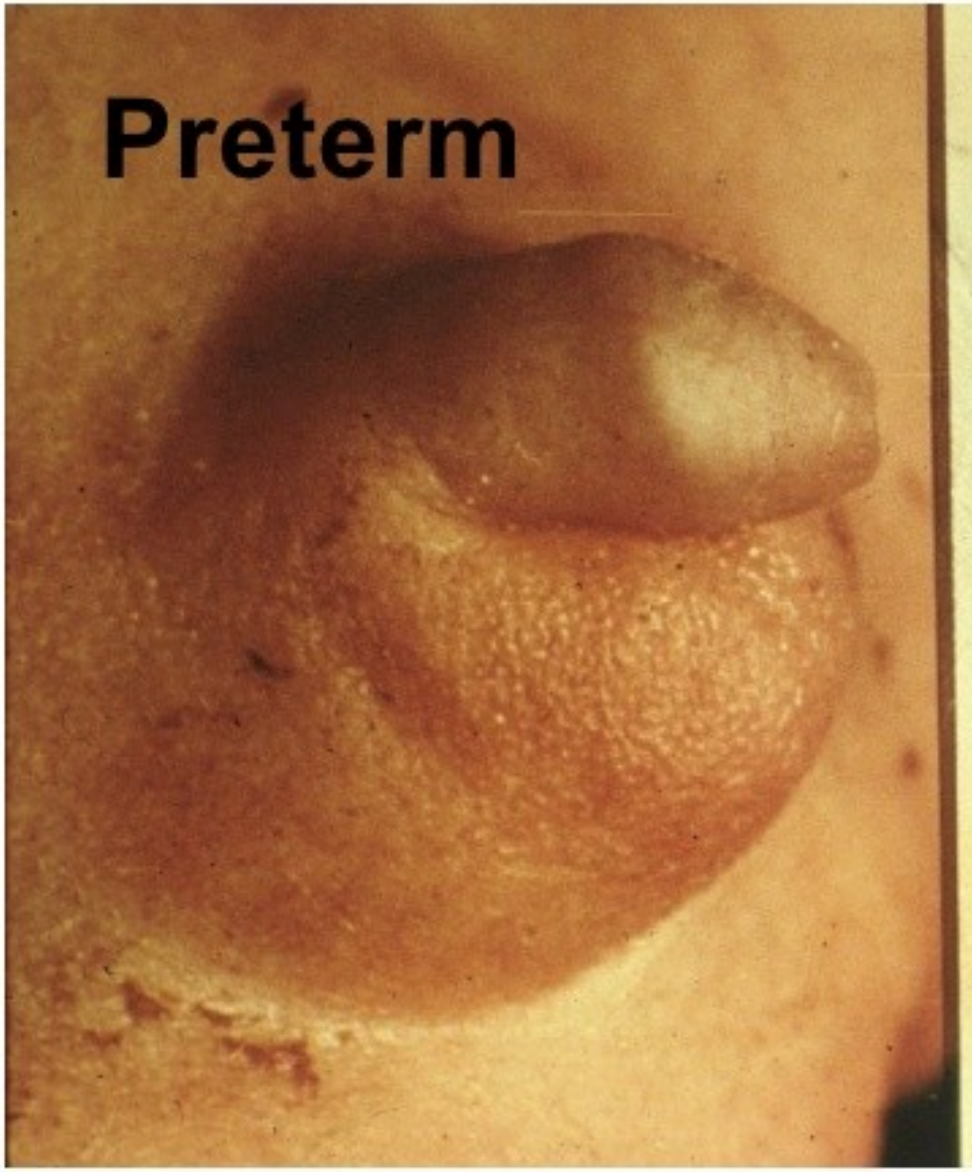
Term

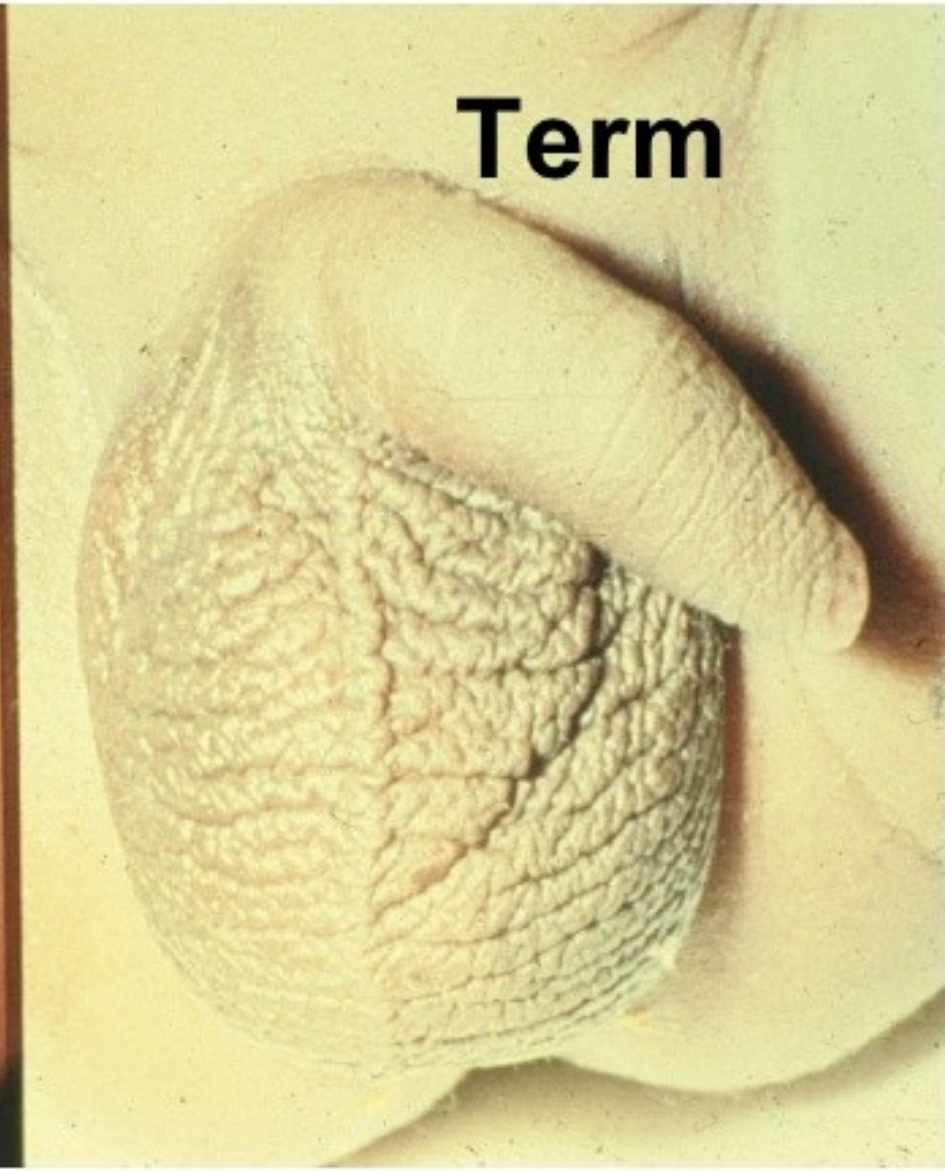
Breast nodule





Male genitalia





MANAGEMENT

- Sedatives of opium derivatives should not be used during premature labor
- Shorten the duration of second stage by performing an episiotomy. As this will reduce the possibility of intracranial injury
- Clear the airway using a fine mucus extractor/catheter
- Administer oxygen at 1 litre per minute till respiration is well established and color is satisfactory

- Put the infant in an incubator or cover with warm towels and transfer to a special baby care unit as soon as practicable
- Once immediate steps have been taken to ensure the well being of the premature infant, further management aims at:
  - Maintenance of respiration
  - Provision of warmth
  - Prevention of infection
  - Ensuring good progress and growth
  - Promotion of parent-infant attachment
  - Ensuring baby gets adequate nutrition

- If the baby cant tolerate feeds give iv fluids e.g. 10% dextrose
- Give nutritional supplements e.g. iron, folic acids, calcimax, multivitamin from 2 weeks, vitamin D at 1 month.
- Take weight on alternative days to monitor their progress
- Introduce cup feeding gradually as the baby gains weight

COMPLICATIONS OF PREMATURITY

- Cyanotic attacks
- Cerebral hemorrhage
- Heart failure and pulmonary edema
- Jaundice
- Anemia
- Infection
- Poor mental and intellectual development in later years
- Respiratory Distress Syndrome
- Asphyxia

- Rickets
- hypothermia

DISORDER OF GESTATION AGE AND BIRTH WEIGHT

LEARNING OBJECTIVES

By the end of the lesson the learner should be able to :

- Define SGA and light for dates infants
- List the characteristics of SGA infants
- State the factors that affect fetal growth/causes
- State the differentiation between SGA and a preterm infant

- Intra uterine growth standards are used to compare an infant's weight and gestational age with population averages
- Height may however be affected by sex, age, parity etc
- Weight serves to assess growth while gestational age assesses maturity
- The fetal growth chart presents average fetal weights for the 10^{th}, 25^{th}, 50^{th} and 75^{th} percentiles

- An infant born at 40 weeks gestation and weighing less 2500g would be mature but undergrown (intra-uterine growth retardation). This is classified as small for gestational age.
- An infant born at 36 weeks but weighing 3600g (above 90th percentile) would be immature but overgrown (large for gestational age)
- Premature infants are those born before 37 weeks gestation. An infant weighing 2500g or less is of low birth weight while infants weighing 1500g or less are of very low birth weight.

FACTORS AFFECTING FETAL GROWTH

1. **Maternal factors** – maternal nutritional factors will lead to low weight gain during pregnancy

- Women who are malnourished during pregnancy due to poverty, adolescence, women with frequent pregnancies are likely to deliver low birth weight babies
- pregnancy induced hypertension poses a threat to fetal growth since it decreases intra uterine blood flow
- advanced maternal diabetes and the accompanying vascular insufficiency promote intra uterine growth retardation

- Substance abuse – e.g. cigarette smoking has been correlated with decreased fetal growth. Birth weight is decreased on the average of 170g as a result of 10 cigarettes smoked per day
- Extremes of maternal age

- **2.Placental influences**
- Under normal circumstances the size of the placenta determines fetal size. When there is severe nutritional deprivation the maintenance needs of the placenta are met first leading to reduction in fetal growth. The diffussional capacity of the placenta increases with fetal growth. Any impairment of oxygen and nutrients transfer affects fetal .
- **3.fetal endocrine influences -????**

SMALL FOR GESTATIONAL AGE

- A term used to describe a baby who is smaller than the usual size for the number of weeks of pregnancy; considering parameters such as length head circumference

assesment of sga infants

- Neurological criteria tend to be more accurate than physical criteria since most of the physical characteristics tend to be abnormal.

LIGHT FOR DATES BABY

- A baby whose birth weight is below the 10th percentile for its estimated gestational age. One third of low birth weight babies are light for dates
- Both conditions reflect intrauterine malnutrition and growth retardation of the fetus.

MANAGEMENT

- There is no significant difference in management from that described for the normal infant except that the small for gestational age or light for dates infant would be more susceptible to hypothermia.
- Babies <2kg should be given incubator care if not on skin to skin care
- Initiate early feeding to prevent hypoglycemia using cup
- Labworks , I/O,

- Facilitate bonding
- Infection prevention
- Health education and support to parents and caregivers

COMPLICATIONS OF SGA

- Asphyxia
- Respiratory distress syndrome (rare)
- Hypoglycemia
- Hypothermia
- There is a high mortality risk

COMPARISON BTN PRETERMS AND SGAS

Preterm	Small for Gestational Age
Skin red and shiny, lanugo, plentiful	Skin red and shiny, lanugo, plentiful
Skull bones soft, sutures and fontanelles wide	Skull bones soft, sutures and fontanelles wide
Plantar creases not visible	Plantar creases visible
Most of the reflexes e.g. swallowing, moro absent	Moro and traction reflexes present
Pinna of ears soft and flat	Pinna of ear has cartilaginous ridges and firm
Born before 37 weeks gestation	Born at term
Eyes always closed	Eyes wide open (worried look)

NEONATAL JAUNDICE

OBJECTIVES

- Define jaundice
- Differentiate between physiological and pathological jaundice
- describe the management of jaundice
- Discuss nursing management of a baby on phototherapy
- Describe exchange transfusion
- Discuss the two main types of blood incompatibilities

DEFINITION

- Neonatal jaundice is the yellowish discoloration of the skin and/or sclera of newborn infants caused by tissue deposition of bilirubin.
  - There is Excessive levels and accumulation of bilirubin in blood
  - Normal levels are 0.2-1.4mg/dl.
  - Normally manifests when the bilirubin levels are more than 5mg/dl.

Bilirubin metabolism

- When RBCs are broken down by haemolysis, they produce haeme and globulin.
- The haem part produces bilirubin,carbon monoxide and iron (via biliverdin which is green)
- Unconjugated(indirect) bilirubin is fat soluble hence has to be converted to water soluble form(conjugated/direct bilirubin) for it to be excreted .
- Bilirubin is **transported** to the liver by binding to a protein(albumin)

Bilirubin metabolism cont…

- On arrival to the liver , bilirubin detaches itself from albumin
- Conjugation of bilirubin occurs in the liver
- **Conjugation** is done by glucuronyl transferase in which bilirubin is added to glucuronic acid to become bilirubin diglucuronide that is water soluble
- **Excretion** of the bilirubin is done through the biliary system into the intestines

Bilirubin metabolism cont…

- While in the intestines bilirubin is converted to stercobilinogen by the gut normal flora and excreted in stool
- Some of it is absorbed in the gut and becomes urobilinogen which is excreted in urine.
- If conjugation is interfered with there will be accumulation of unconjugated bilirubin leading to hyperbilirubinaemia and jaundice

Bilirubin metabolism cont…

- This bilirubin may cross the blood brain barrier and cause brain damage leading to a condition known as **kernicterus** (characterized by hypertonicity, seizures,lethargy,stiff neck with hyper extended head)

PHYSIOLOGICAL JAUNDICE

Characteristics:

Affects both term and preterm babies

It appears **between 24 to 72 hours** of life. It is apparent with signs on Day 3 of birth.

It is self limiting in term babies and fades away within 7 days.

the levels of bilirubin in term babies don't exceed 15 mg/dl

CAUSES OF PHYSIOLOGICAL JAUNDICE

1. **Excessive breakdown of RBCs**

While in the uterus the fetus relies on fetal hemoglobin which has a greater affinity for oxygen, than the adult Hb

Once the neonate is born it adapts to the pulmonary system of adult Hb and the large mass of RBCS being hemolyzed creates a bulk of bilirubin in the circulation.

2**. Glucuronyl transferase enzyme deficiency**

Enzyme responsible for conjugation of bilirubin. Deficiency of this enzyme results in increased levels of unconjugated bilirubin.

3. Increased enterohepatic reabsorption of bilirubin

Increases in newborns, because of lack of the normal enteric bacteria that breaks down bilirubin to urobilinogen/ stercobilinogen

Increased beta glucuronidase enzyme(hydrolyses bilirubin back to unconjugated bilirubin state). Accelerated by lack of feeding

4. Decreased albumin binding capacity:

albumin binds to unconjugated bilirubin and transports to liver for conjugation. In preterm babies, the albumin is in lower levels, hence bilirubin will bind to tissues with bilirubin affinity such as the skin and brain

PATHOLOGICAL JAUNDICE

- Clinical jaundice detected **before 24 hours** of life.
- Characterized by rapid rise in serum bilirubin (5 mg/dl) per day
- Serum bilirubin greater than 15mg/dl
- Clinical jaundice persisting beyond 14 days of life
- Clay or white colored stool and/dark urine staining the clothes yellow

CAUSES OF PATHOLOGICAL JAUNDICE

1. **Increased production of bilirubin**: due to increase in hemoglobin destruction

Causes of destruction:

- Blood type/group incompatibility
- Hemoglobinopathies- sickle cell disease
- Enzyme deficiency –G6PD (maintains the integrity of the cell membrane)
- Sepsis/ infections
- Polycythemia

2. **Interference with transportation**

due to:

- reduced albumin
- acidosis, hypothermia
- hypoxia,
- drugs that compete for albumin binding sites e.g. aspirin/ ampicillin

3. Conjugation interference:

- Immaturity of enzyme systems.
- Lack of/ inadequate glucuronyl transferase enzyme.

4. Excretion:

bilirubin excretion is interfered with e.g.:

- Hepatic obstruction due to anomalies (extrahepatic biliary atresia)
- Infection i.e. neonatal hepatitis can cause an excess of unconjugated bilirubin

MANAGEMENT OF PHYSIOLOGICAL JAUNDICE

- Admit the baby in the newborn unit and assess the general condition
- Start early and frequent feeds (breast-feeding) for glucose provision to the liver cells, and also to encourage bowel colonization with normal flora; important for formation of stercobilinogen for excretion in stool
- Closely monitor serum bilirubin levels at 12-24 hour interval
- If the bilirubin takes long to clear, phototherapy may be necessary

MANAGEMENT OF PATHOLOGICAL JAUNDICE

- Assessment of the newborn to determine the degree of jaundice
- To determine the cause of jaundice
- On physical examination, check for:
  - Skin color and sclera color
  - Signs of lethargy, stiff neck, hyperextended neck, seizures, (signs of kernicterus)
  - Presence of dehydration or starvation
  - Light stool or dark urine

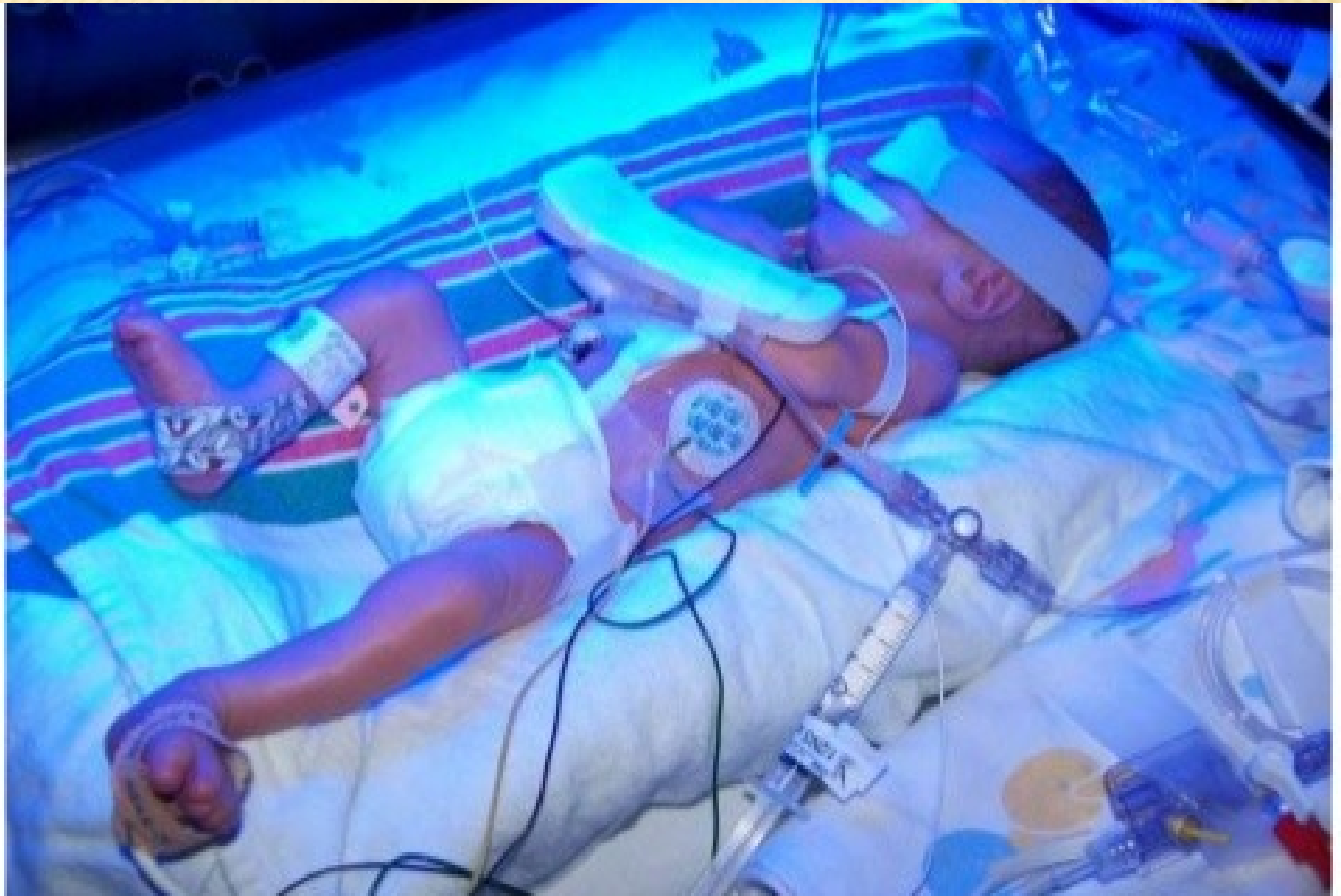
TREATMENT MODALITIES FOR PATHOLOGICAL JAUNDICE

- Phototherapy
- Exchange transfusion

PHOTOTHERAPY

High intensity light at a given wave length (white to blue) administered in order to convert unconjugated bilirubin to conjugated bilirubin, which is excreted through stool/urine.

PHOTOTHERAPY CONT…



NURSING CARE OF THE BABY ON PHOTOTHERAPY

- Clean the skin with warm water, avoid using creams and lotions on the skin of the baby
- Expose the whole body to ensure there is increased surface area of skin to light.
- Keep turning the baby 2 hourly to expose all parts to the light and monitor for rashes or dryness.
- Top-tail the baby for hygiene and change linens frequently

- Observe the skin color to monitor the progress.
- Monitor the temperature to prevent hyperthermia
- Cover the eyes with an eye pad to prevent retinal damage from the light. Uncover during breastfeeding to ensure eye contact
- For continuous phototherapy, give extra fluids for every ill neonate or dehydrate babies.
- Monitor neurological status of the newborn e.g. the sleep/wake patterns
- Explain and reassure the family

SIDE EFFECTS OF PHOTOTHERAPY

- Hyperthermia leading to fluid loss and dehydration
- Retinal damage due to high intensity lights
- Skin rashes and skin burns
- Visual deprivation and isolation

EXCHANGE TRANSFUSION

- The treatment in which the baby's blood is gradually removed and replaced by donor blood.
- Done when bilirubin levels are toxic (23-29)

Indications:

- Infants with a hemolytic disease
- Preterm babies less than 1500g
- Healthy term babies with bilirubin levels of 23 to 29

Complications:

- Circulatory collapse
- Incompatibility reactions
- Acquired infections
- Blood clots

COMPLICATIONS OF JAUNDICE

- Retinal damage due to light used in phototherapy
- Anemia due to increased RBC haemolysis
- Hyperthermia associated with phototherapy
- Hypocalcemia due to increased metabolism
- Kernicterus when bilirubin crosses BBB causing brain damage.

HEMOLYTIC JAUNDICE

OVERVIEW

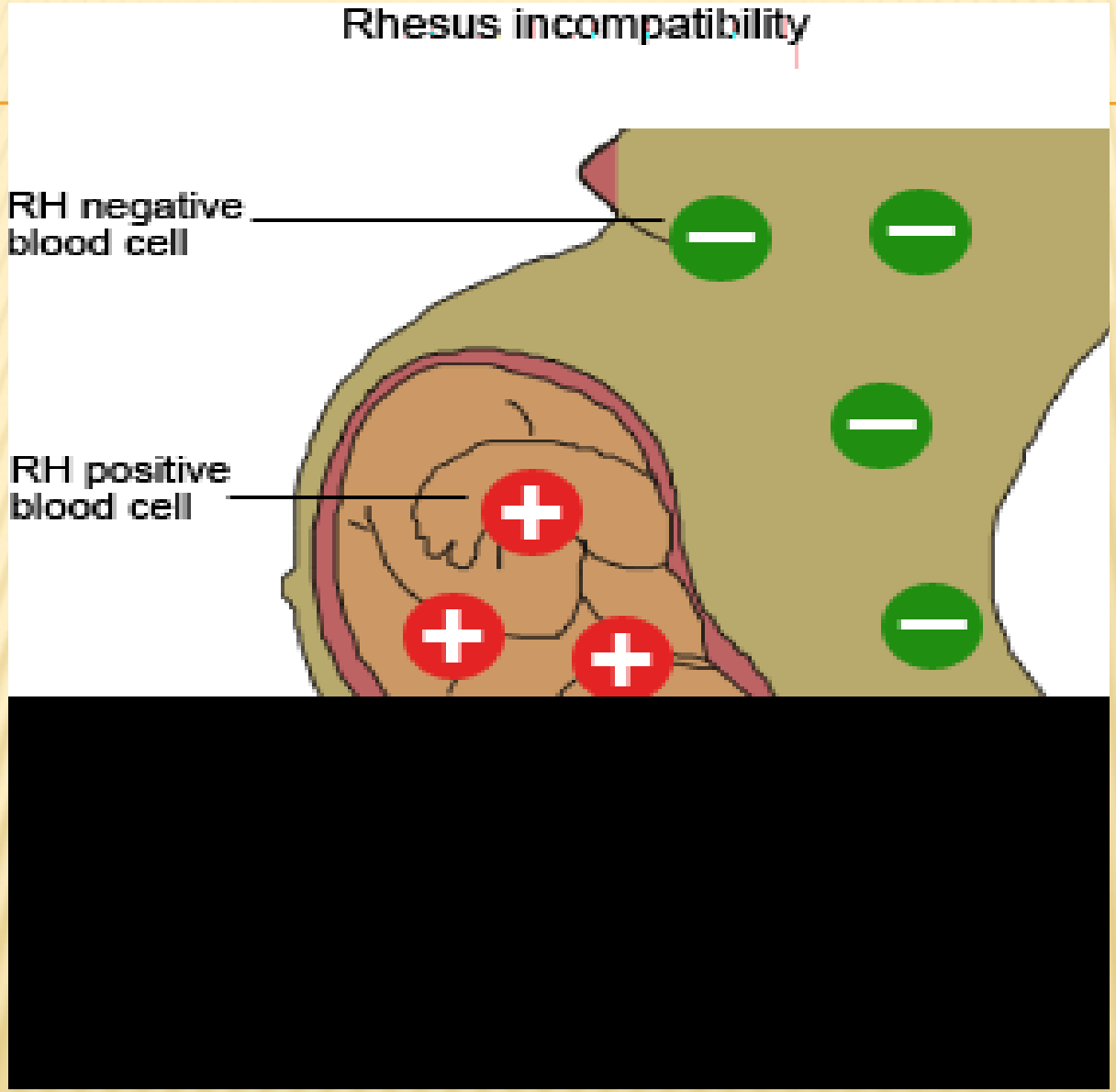
- Types of incompatibility
- Prevention
- Nursing management

TYPES OF INCOMPATIBILITY

- Rhesus incompatibility
- ABO incompatibility

RHD INCOMPATIBILITY

- Also referred to as **hemolytic disease of the newborn**
- The Rhesus complex (The D antigen) is an antigen found in the red blood cells of about **83%** of the population.
- Problems arise if a **Rhesus negative mother** and a **Rhesus positive father** have a child who inherits the Rhesus positive traits of the father.


Rhesus incompatibility
RH negative blood cell
RH positive blood cell

- The placenta usually acts as a barrier to fetal blood entering the maternal circulation.
- However, during pregnancy or birth, fetomaternal hemorrhage (FMH) can occur
- The woman’s immune system reacts by producing anti-D antibodies that cause sensitization.
- In subsequent pregnancies these maternal antibodies can cross the placenta and destroy fetal erythrocytes.

- This hemolytic disease of the fetus and newborn caused by Rh isoimmunization can **sometimes** occur during the first pregnancy
- Sensitization during the first pregnancy or birth leads to extensive destruction of fetal red blood cells during subsequent pregnancies
- Rh isoimmunization can result from any procedure or incident where maternal blood leaks across the placenta or from the transfusion of Rh positive blood to the woman

CELL DESTRUCTION

- Cells are destroyed, causing anemia and excess bilirubin in the blood of the baby.
- The baby will consequently be born with jaundice, which is normally seen 12 - 24 hours after delivery.

NB: Sometimes the neonate may not appear jaundiced, but the amniotic fluid will be golden and the cord is yellow.

PREVENTION OF RHD ISOIMMUNIZATION

- Anti D Ig:

Prevention is by administration of anti D immunoglobulin to mothers with non sensitized negative blood type who have a baby with Rh positive blood type within 72 hours of birth or any other sensitizing event

PROPHYLAXIS FOLLOWING OTHER SENSITIZING EVENTS

- Any threatened, complete or incomplete or missed abortion after 12 weeks of pregnancy; if bleeding persists, anti-D should be given at 6 week intervals
- Any spontaneous miscarriage before 12 weeks that requires surgical intervention to evacuate the uterus
- Therapeutic termination of pregnancy by surgical or medical methods regardless of gestational age
- Ectopic pregnancy

SENSITIZING EVENTS CONT.D

- Amniocentesis, cordocentesis, chorionic villus sampling, fetal blood sampling or any other invasive intrauterine procedure
- External cephalic version of the fetus
- Fetal death in utero or stillbirth
- Abdominal trauma and antepartum hemorrhage
- Transfusion of Rh positive blood or platelets

ANTENATAL MANAGEMENT

- A mother who is Rhesus negative should be closely observed and at 26 - 28 weeks gestation; blood is taken for Coomb's test, which is repeated at 34 - 36 weeks.
- Antibody titre is estimated. If it continues to rise, then labor is induced.
- However, Intravenous immunoglobulin (IVIG) has the potential to maintain the fetus until intrauterine fetal transfusion can be performed. (possible in more developed countries).

ABO INCOMPATIBILITY

- ABO isoimmunization usually occurs when the mother is blood group O and the baby is blood group A. or less often group B.
- Blood of types A and B has protein or antigen not present in type O blood.
- Individuals with type O blood develop antibodies against A and B
- By the first pregnancy the individuals may already have high serum anti-A and anti-B antibody titers.

- Some women produce IgG antibodies that can cross the placenta and attach to fetal red cells and destroy them
- First and subsequent babies are at risk; however, the destruction is usually much less severe than with Rh incompatibility.
- ABO incompatibility is also thought to protect the fetus from Rh incompatibility as the mother’s anti-A and anti-B antibodies destroy any fetal cells that leak into maternal circulation.

COMPLICATIONS.

- Hydrops fetalis due excessive RBC hemolysis (leading to anemia)It mainly presents as massive edema even to most organs and finally causes, multiple organ failure
- Anemia.
- Kernicterus

SEPSIS NEONATORUM

OVERVIEW

- Definition
- Risk factors
- Signs of sepsis
- Early onset and late onset syndromes
- Omphalitis

DEFINITION

- Neonatal sepsis is a clinical syndrome of systemic illness accompanied by bacteremia occurring in the first month of life

RISK FACTORS

- Prematurity
- Prolonged rapture of membranes
- Internal monitoring devices
- Twin pregnancy
- Maternal urinary tract infection
- Maternal fever
- Maternal bacteremia
- Chorioamnionitis

ROUTES OF INFECTION

- Transplacental/Hematogenous
- Ascending/Birth Canal
- Nosocomial

NON-SPECIFIC SIGNS OF NEONATAL SEPSIS

1. Temperature instability
2. Respiratory distress
3. Feeding intolerance
4. Vomiting
5. Abdominal distension
6. Diarrhea
7. Jaundice
8. Pallor
9. Skin rash/ petechiae
10. Hypotension
11. Tachycardia
12. Apnea and bradycardia
13. Irritability
14. High pitched cry
15. Lethargy
16. Weak suck
17. Convulsions
18. Bulging of fontanelle

EARLY ONSET SYNDROME

- Occurs mostly in first 5-7 days of life
- Usually multisystem fulminant illness with prominent respiratory symptoms (probably due to aspiration of infected amniotic fluid)
- Has high mortality rate (5-20%)
- Typically acquired during intrapartum period from maternal genital tract
  - Associated with maternal chorioamnionitis

LATE ONSET SYNDROME

- Mostly occurs in infants between 8 to 30 days of age, but may be seen as early as 5 days and as late as 12 to 16 weeks, especially in premature infants
- Acquired from maternal genital tract or human contact
- Onset is most often insidious and the affected infants present with non-specific signs of sepsis.
- Most common conditions are meningitis, osteomyelitis, septic arthritis, skin and soft tissue lesions, endocarditis, peritonitis, omphalitis and pericarditis

OMPHALITIS

OVERVIEW

- Definition
- Etiology
- Clinical features
- Collaborative management
- Nursing care

INTRODUCTION

- Omphalitis occurs predominantly in neonates
- Omphalitis is an **inflammation** of the umbilical stump
- It presents as superficial cellulitis (a red flare) around the umbilicus
- The cellulitis may progress rapidly with potentially serious consequences

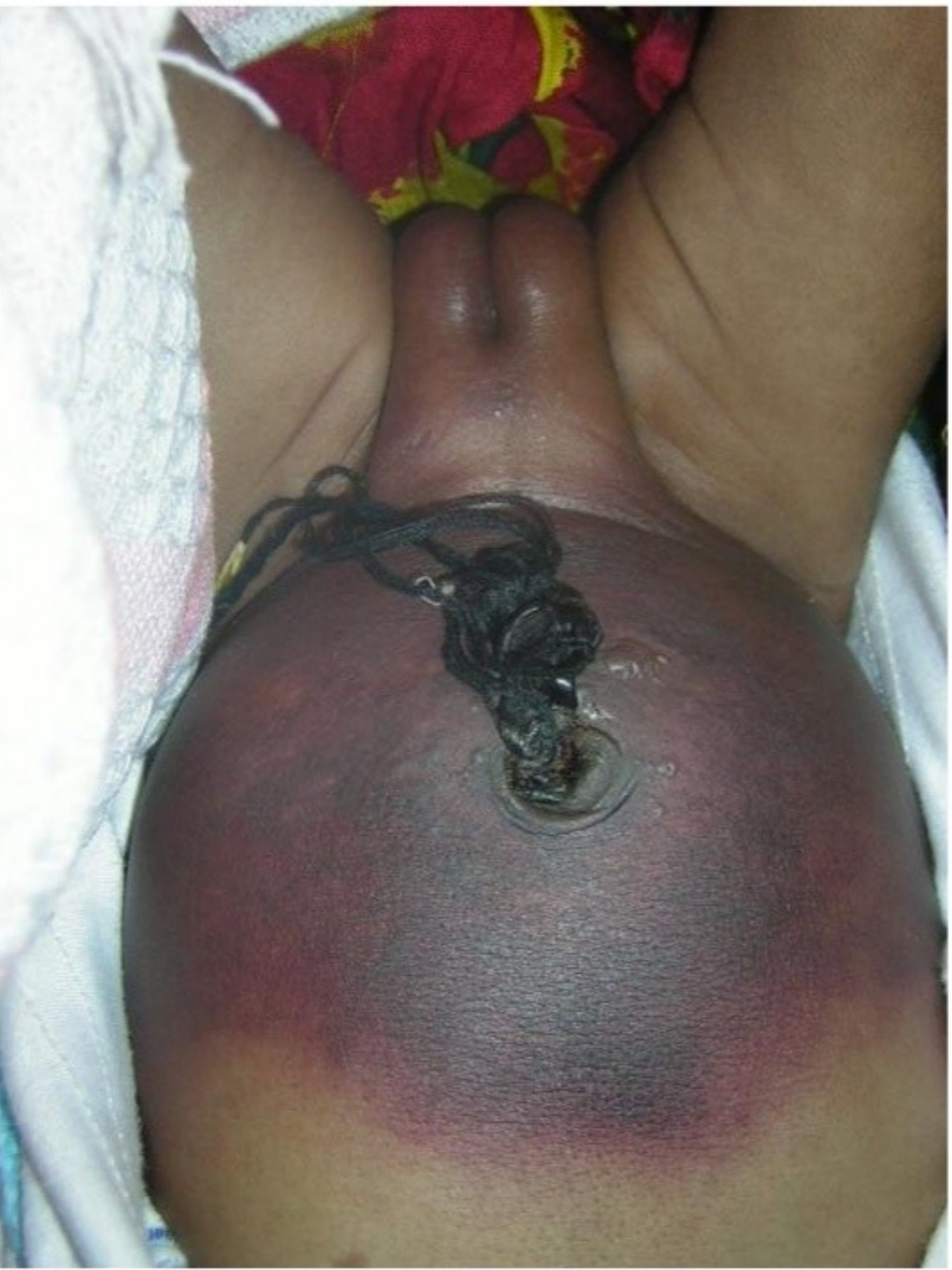
ETIOLOGY

- Aerobic bacteria in 85% of all cases e.g. staphylococcus aureus, Group A beta Hemolytic streptococcus, Escherichia coli, Klebsiella Pneumonia
- Pseudomonas species
- Occasionally from an underlying immunologic disorder (Leukocyte adhesion deficiency)

CLINICAL FEATURES

It occurs at around 5 to 9 days in term neonates

- Redness and swelling (cellulitis) around the umbilicus
- Purulent discharge from the umbilicus
- Tenderness
- Irritability
- Crepitus
- Bullae
- Necrotizing fascitis; characterized by abdominal distension, fever, tachycardia



MANAGEMENT

History taking:

Obtain a detailed history of pregnancy, labor, delivery and neonatal course

Physical examination:

Check for signs of local and systemic infection

INVESTIGATIONS

- Obtain specimen from umbilical discharge for gram stain and culture for aerobic and anaerobic organisms (umbilical swab culture)
- Blood culture for aerobic and anaerobic organisms
- Complete blood count (CBC)
- Random blood sugar

TREATMENT

1. Antimicrobial therapy:

Parenteral antimicrobial therapy for gram-positive and gram-negative organisms

Anaerobic coverage also recommended

The following combinations are preferred:

- **Cloxacillin + Gentamycin + Metronidazole**

  Or

- **Cephalosporin + Gentamicin + Metronidazole**

2. Surgical Care

It involves early and complete surgical debridement of the affected tissue and muscles

Excision of pre-peritoneal tissue (umbilicus and umbilical vessels)

NURSING CARE:

- Vital signs monitoring; especially temperature and respiration,
- Maintain oxygen saturations above 95%
- Obtain random blood sugars
- Feed as tolerated
- Obtain daily weights

ASSIGNMENT

- Discuss pemphigus in infants
  - Definition
  - Etiology
  - Management
  - complications

INFANT OF A DIABETIC MOTHER

INTRODUCTION

- Women with diabetes in pregnancy (Type 1, Type 2, and gestational) are all at increased risk for adverse pregnancy outcomes.
- Adequate glycemic control before and during pregnancy is crucial to improving outcome
- Diabetic mothers have a high incidence of polyhydramnios, preeclampsia, preterm labor, and chronic hypertension;
- Their fetal mortality rate, especially after 32 wks, is greater than that of non diabetic mothers.

- Most infants born to diabetic mothers are large for gestational age.
- If the diabetes is complicated by vascular disease, infants may be growth restricted, especially those born after 37 wk gestation
- The neonatal mortality rate is over 5 times that of infants of non diabetic mothers and is higher at all gestational ages

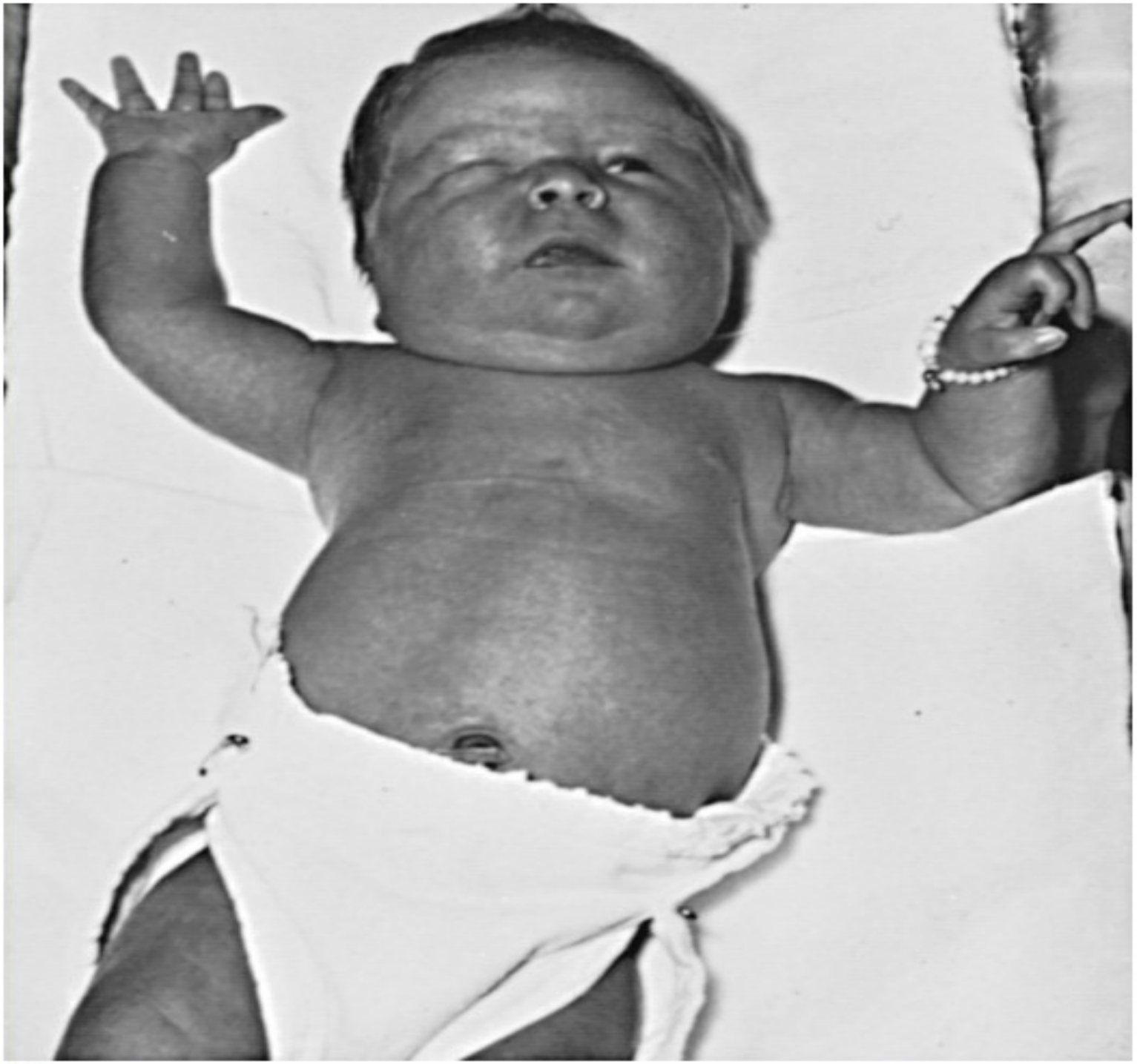
PATHOPHYSIOLOGY

- The probable pathogenic sequence is that maternal hyperglycemia causes fetal **hyperglycemia**
- The fetal pancreatic response leads to fetal **hyperinsulinemia**
- Fetal hyperinsulinemia and hyperglycemia then cause increased hepatic glucose uptake and glycogen synthesis, accelerated lipogenesis, and augmented protein synthesis

- Hyperinsulinism and hyperglycemia produce fetal acidosis, which may result in an increased rate of stillbirth.
- Separation of the placenta at birth suddenly interrupts glucose infusion into the neonate without a proportional effect on the hyperinsulinism
- Hypoglycemia and attenuated lipolysis develop during the 1st hr after birth.

CLINICAL MANIFESTATIONS

- They tend to be large and plump as a result of increased body fat and enlarged viscera, with puffy faces resembling that of patients who have been receiving corticosteroids



MANIFESTATIONS CONT…

- Hypoglycemia
- Tachypnea
- Cardiomegally

Increased incidence of: hyperbilirubinemia, polycythemia, and renal vein thrombosis

MANAGEMENT

- frequent prenatal evaluation of all pregnant women with overt or gestational diabetes, by evaluation of fetal maturity
- by planning the delivery of these infants in hospitals where expert obstetric and pediatric care is continuously available
- Periconception glucose control reduces the risk of anomalies and other adverse outcomes, and glucose control during labor reduces the incidence of neonatal hypoglycemia.

- Regardless of size, all infants of diabetic mothers should initially receive intensive observation and care.
- Asymptomatic infants should have a blood glucose determination within 1 hr of birth and then every hour for the next 6–8 hr;
- if clinically well and normoglycemic, feeding with breast milk or formula should be started as soon as possible and continued at 3 hr intervals.

- If any question arises about an infant's ability to tolerate oral feeding, the feeding should be discontinued and glucose given by peripheral intravenous infusion at a rate of 4–8 mg/kg/min
- Bolus injections of hypertonic glucose should be avoided because they may cause further hyperinsulinemia and potentially produce rebound hypoglycemia

COMPLICATIONS

- Macrosomia
- Hypoglycemia
- Polycythemia
- Respiratory distress syndrome
- cardiac failure
- cerebral edema from birth trauma
- asphyxia

BIRTH INJURIES

OVERVIEW

- Types of injuries (classification)
- Risk factors
- Caput succedaneum
- cephalhematoma
- Differences between caput and cephalhematoma
- Intracranial hemorrhage

INTRODUCTION

- A birth injury is an impairment of an infant’s body function or structure due to adverse influences that occur at birth

RISK FACTORS

- Primiparity
- Small maternal stature
- Maternal pelvic anomalies
- Prolonged or unusually rapid labor
- Oligohydramnios
- Malpresentation of the fetus
- Instrumental delivery (eg use of forceps, vacuum extraction)
- Very low birth weight or extreme prematurity
- Fetal macrosomia/ large fetal head
- Fetal anomalies

TYPES OF INJURY

- **Soft tissue** – lacerations, abrasions, fat necrosis
- **Nerve** – facial, brachial, spinal cord, phrenic
- **Eye** – hemorrhage
- **Viscera** – rupture of liver, adrenal gland, spleen
- **Scalp** – laceration, abscess, hemorrhage
- **Dislocation** – hip, shoulder, cervical vertebrae
- **Skull** – cephalhematoma, fractures
- **Intracranial** – hemorrhages – intraventricular, subdural, subarachnoid
- **Bone** fractures – clavicle, humerus, femur

HEAD INJURIES

1. Caput Succedaneum
2. Cephalhematoma
3. Intracranial hemorrhage

CAPUT SUCCEDANEUM

CAPUT…

- A caput succedaneum is a serosanguinous fluid collection above the periosteum
- It presents as a soft tissue swelling with purpura and ecchymosis over the presenting portion of the scalp
- It may extend across the midline and across the suture lines

CAPUT…

- The edema disappears within the first few days of life
- Molding of the head and overriding of the parietal bones disappear during the first weeks of life
- Analogous swelling, discoloration, and distortion of the face are seen in face presentations.
- Rarely, a hemorrhagic caput may result in shock and require blood transfusion

MANAGEMENT:

- No specific treatment is needed
- But if extensive ecchymoses are present, hyperbilirubinemia may develop
- Blood transfusion in case of shock

CEPHALHEMATOMA

CEPHALHEMATOMA

- A cephalhematoma is a subperiosteal blood collection caused by rapture of vessels beneath the periosteum

CLINICAL FEATURES CEPHAL..

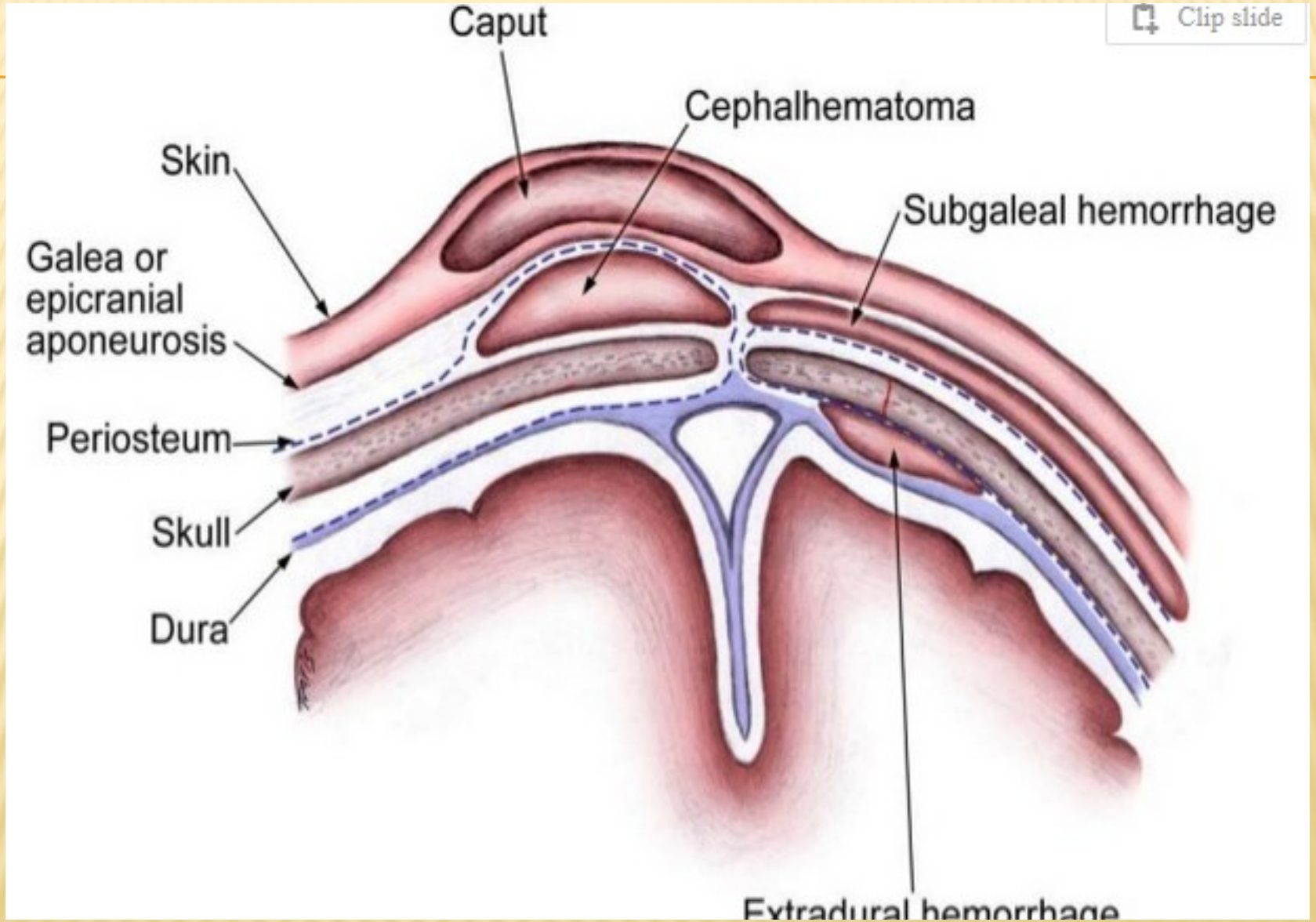
- swelling usually over a parietal or occipital bone
- Swelling does not cross a suture line and is often not associated with discoloration of the overlying scalp
- Limited to the surface of one cranial bone

DIAGNOSIS

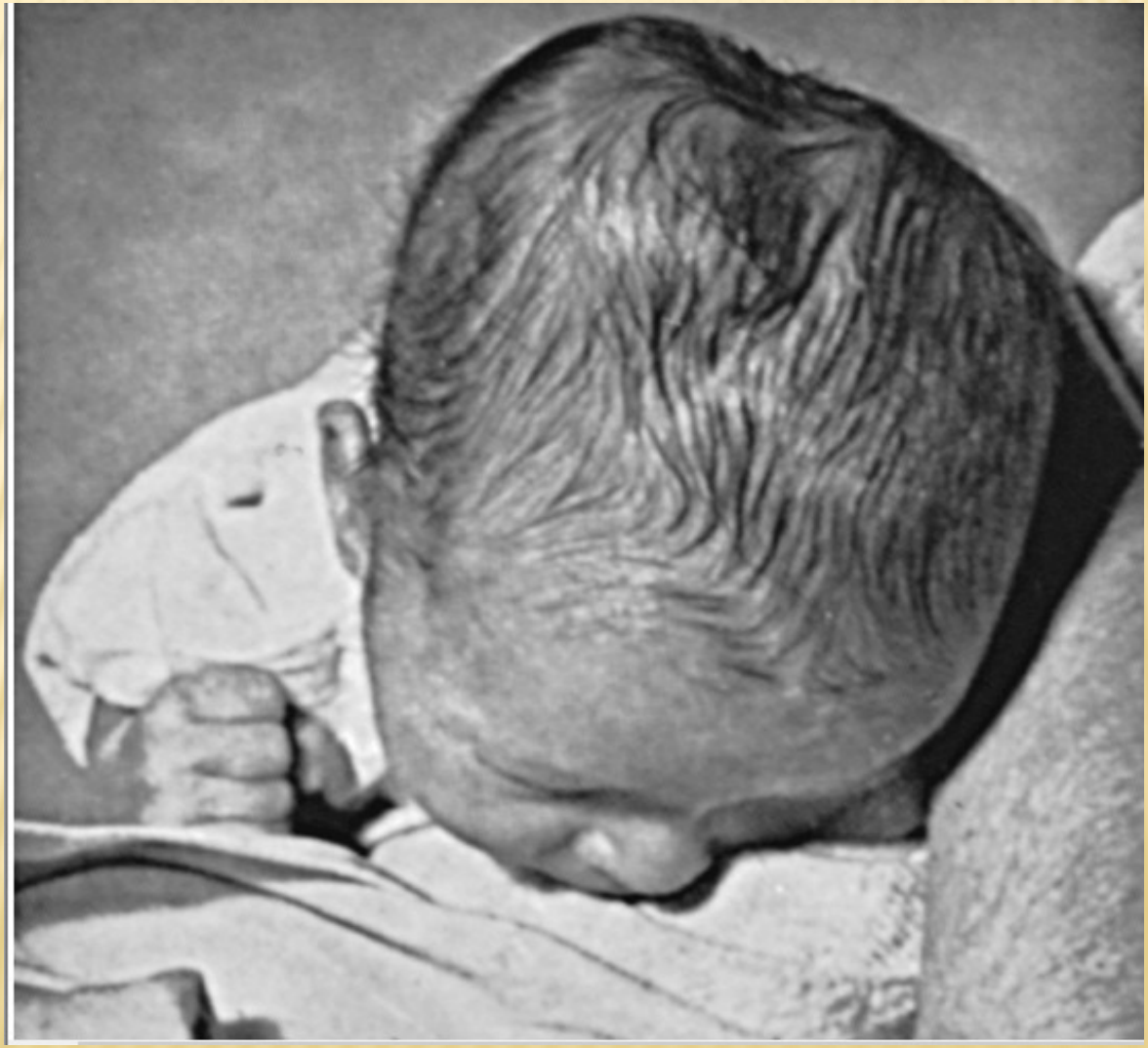
- Physical examination
- Skull radiograph
- Cranial computed tomography
- If infection is suspected, aspiration of the mass
- If sepsis, antibiotics should be started
- Phototherapy in case of hyperbilirubinemia

DIFFERENCES

INDICATORS	CAPUT SUCCEDANEUM	CEPHALHEMATOMA
Location	Presenting part of the head	Periosteum of the skull bone
Extent of involvement	Both hemispheres; crosses the suture lines	Does not cross the suture lines
Period of absorption	3 to 4 days	Few weeks to months


Caput
Cephalhematoma
Skin
Subgaleal hemorrhage
Galea or epicranial aponeurosis
Periosteum
Skull
Dura
Extradural hemorrhage

CEPHALHEMATOMA OF THE RIGHT PARIETAL BONE



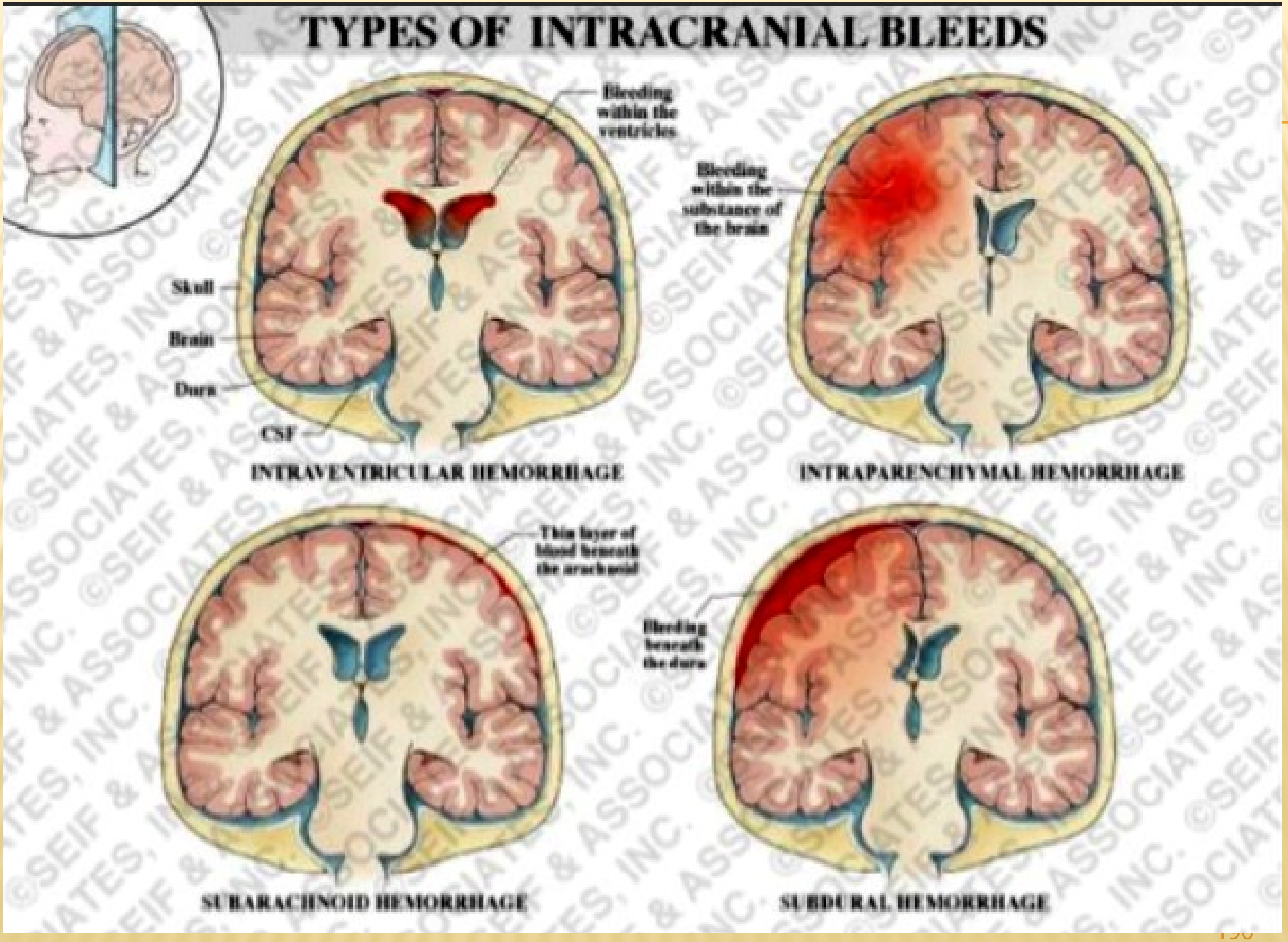
INTRACRANIAL HEMORRHAGE

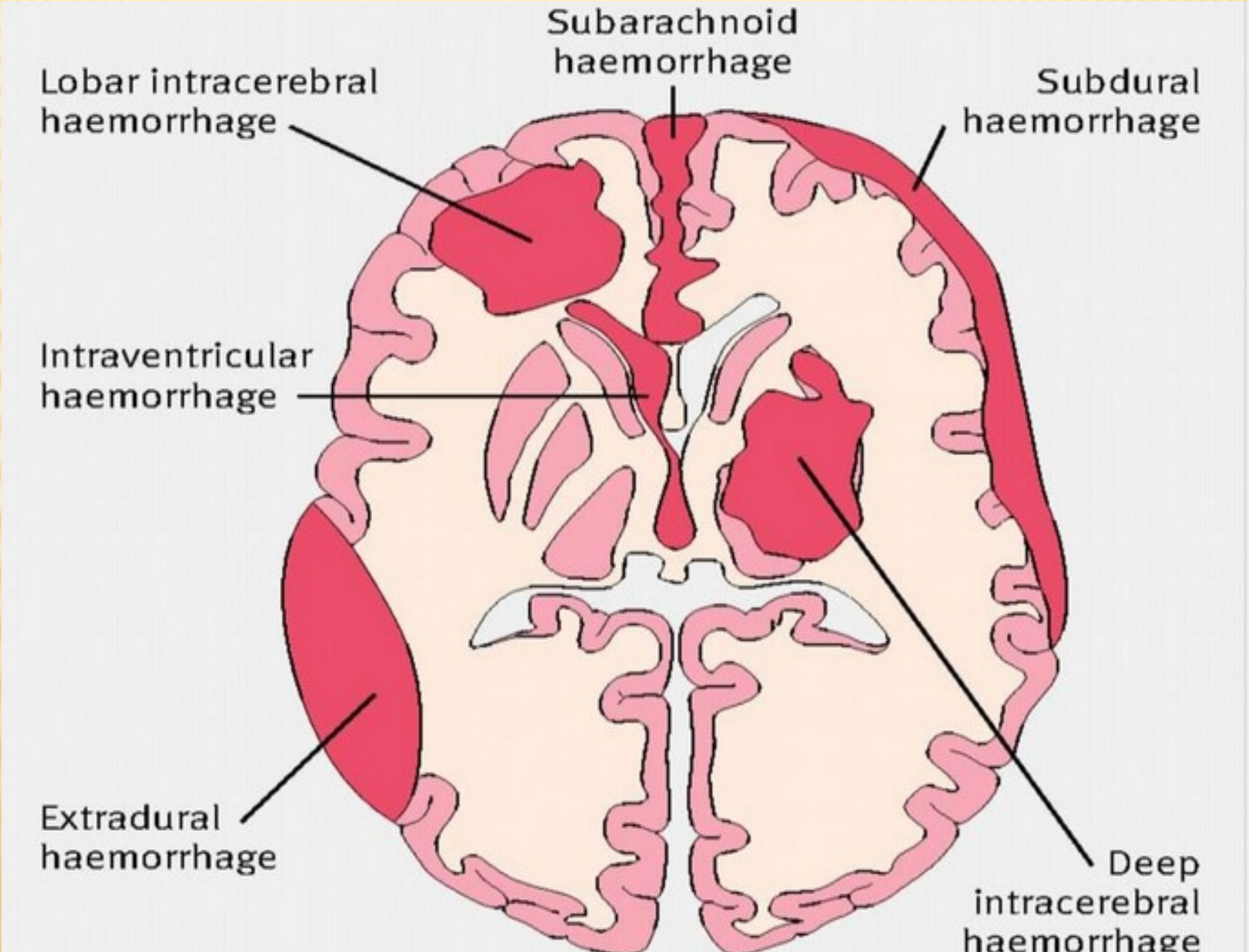
INTRODUCTION

- An intracranial hemorrhage is the pathologic accumulation of blood within the cranial vault.
- This condition can be categorized according to the site of origin of the hemorrhage (in some cases, intracranial hemorrhage may involve multiple compartments), as follows:

INTRO…

1. **Epidural hemorrhage** indicates blood between the skull and outside of dura
2. **Subdural hemorrhage** denotes blood between the dura and the arachnoid mater
3. **Subarachnoid hemorrhage** refers to blood between the arachnoid and the pia mater
4. **Intraventricular hemorrhage** refers to blood within the ventricles
5. **Intraparenchymal hemorrhage** refers to blood within the brain itself


TYPES OF INTRACRANIAL BLEEDS
Bleeding within the ventricles
Skull
Brain
Dura
CSF
INTRAVENTRICULAR HEMORRHAGE
Bleeding within the substance of the brain
INTRAPARENCHYMAL HEMORRHAGE
Thin layer of blood beneath the arachnoid
SUBARACHNOID HEMORRHAGE
Bleeding beneath the dura
SUBDURAL HEMORRHAGE


Subarachnoid haemorrhage
Lobar intracerebral haemorrhage
Subdural haemorrhage
Intraventricular haemorrhage
Extradural haemorrhage
Deep intracerebral haemorrhage

ETIOLOGY

- Large head size
- Rigid pelvis
- Nonvertex presentation (breech, face, etc.)
- Very rapid or prolonged labor or delivery
- Difficult instrumental delivery
- Coagulation disorders (e.g excess fibrinolytic activity, platelet dysfunction)

CLINICAL PRESENTATION

- A mass can be noted within 4 hours of birth
- Usually a clinically silent syndrome in preterms
- Term newborns present with seizures, apnea, irritability, lethargy, vomiting, bulging fontanelle
- Systemic signs like hypovolemia and anemia

MANAGEMENT IN PRETERMS

- Supportive care; watch for complications
- Maintaining normal BPs, electrolytes and blood gases
- Transfusions as necessary
- Correct thrombocytopenia and coagulation disturbances

MANAGEMENT IN TERM INFANTS

- Supportive care and treatment of seizures
- Serial lumbar punctures and eventual Venous pressure (VP) shunts
- In severe cases, surgery may be required to cauterize the bleeding vessels

DIAGNOSIS

- MRI/ Ct. scans
- Serial homoglobin and hematocrit monitoring
- Coagulation profile to investigate for presence of a coagulopathy
- Bilirubin levels also need to be monitored

FRACTURES

INTRODUCTION

- Most fractures noted at birth are caused by trauma at the time of delivery
- When more than one fracture is present, one should suspect underlying bone disease such as osteogenesis imperfecta
- Fractures associated with delivery are usually either midshaft fractures of the clavicle, humerus or femur or epiphiseal separations of the femur or humerus

FRACTURES OF THE CLAVICLE

- Fractures of the clavicle are often unrecognized
- Occur in less than 1% of newborns
- Common during vertex delivery
- Often of the “greenstick” type (bone bends but does not snap)
- Occasionally, crepitations may be felt
- An asymmetric moro reflex may be noted.
- Does not require treatment; other than care in handling

FRACTURES OF THE HUMERUS

- Epiphyseal separation of proximal humerus:
  - The fractured limb is limp
  - Can be confused with Erb palsy.
- Midshaft humeral fractures:
  - Motion is at the center of the shaft.
  - There may be associated radial nerve paralysis and resultant wrist drop, which resolves
  - Splinting of the limb to the chest is sufficient

FRACTURES OF FEMUR

- With a fracture of the proximal femoral epiphysis, the injured extremity appears shorter.
- Often associated with abnormal presentation, usually footling breech.
- Treatment consists of reduction and immobilization
- early professional interventions should be sought as serious vascular problems can occur during the management of this fracture

NB:

- Always rule out child abuse (battered child) in all types of trauma

NEONATAL TETANUS

OBJECTIVES

- Define neonatal tetanus
- state the etiology
- state the clinical features
- Discuss the management of a neonate with tetanus
- Outline the prevention of neonatal tetanus

INTRODUCTION

- Neonatal tetanus a form of generalized tetanus that occurs in newborn babies
- Occurs in infants born without protective passive immunity because the mother is not immune.
- Usually occurs through infection of the unhealed umbilical stump
- Estimated >270,000 deaths worldwide per year

CLINICAL FEATURES OF NEONATAL TETANUS

Overview:

- Muscle rigidity
- Irritability
- Dysphagia
- Restlessness
- Facial grimacing
- Muscle spasms
- Poor suck

CLINICAL FEATURES (CONT.D)

- Usually symptoms begin 3-10 days after birth
- Initial symptom is failure to suck and inability to open the mouth known as trismus or lockjaw
- Spasm of the facial muscles immobilizes the jaw and produces a fixed sardonic grin called risus sardonicus

CLINICAL FEATURES CONT.D

- Within 12-24 hours after the first symptom, generalized tonic muscular convulsions occur producing flexion and adduction o the arms, clenching of fists and extension of the lower extremities
- Initially spasms are mild but later become severe with spasms of the glottis and respiratory muscles

CLINICAL FEATURES….

- Abdominal muscles become rigid and spasms of the muscles of the back may result to opisthotonus
- Spasms may be precipitated by touch, noise or bright light
- Baby remains conscious and alert

MANAGEMENT OF NEONATAL TETANUS

The aims of treatment are:

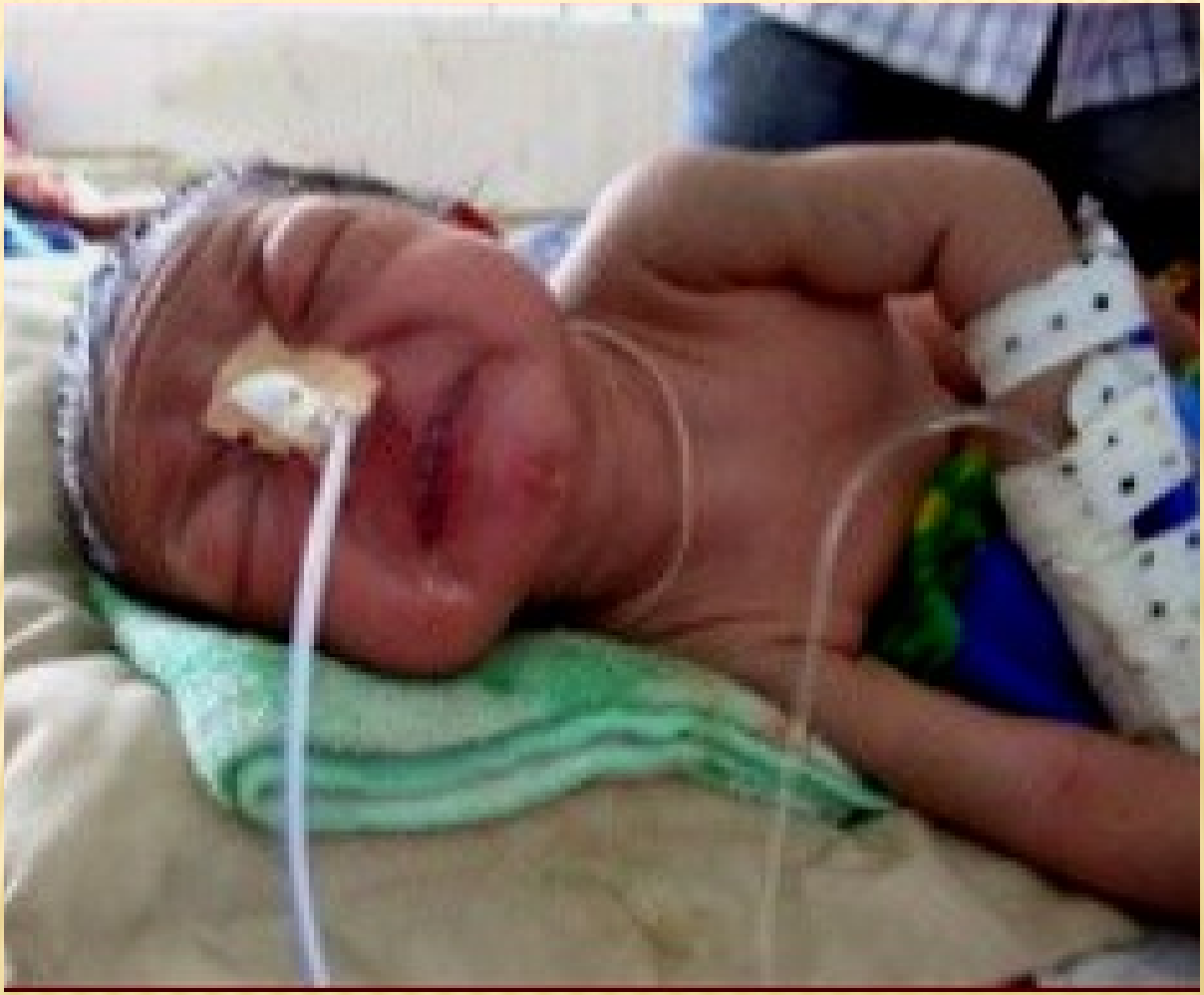
1) Remove the source of the exotoxin
2) Neutralize the remaining circulating toxins
3) Provide supportive care until toxin is metabolized

Medical management:

- Washing and debridement of the infected site, and administration of antibiotics such as benzyl penicillin or metronidazole
- Anti-toxin:
  - Anti-tetanus serum
  - Human tetanus immunoglobulin
- Sedation by:
  - Diazepam (0.1-0.2 mg/kg)
  - Phenobarbitone
  - Paraldehyde

NURSING CARE

- Cleaning the umbilicus/ wound
- Isolate the baby in a dark silent room
- ensure proper positioning; to avoid respiratory compromise
- Cardiorespiratory monitoring and support
- Feed via NG tube: daily milk requirement is 100-120ml/kg/day



COMPLICATIONS

- Aspiration pneumonia
- Lacerations of the mouth and tongue
- Intramuscular hematomas leading to hemoglobinuria and renal failure
- Vertebral fractures
- Decubitus ulcerations
- Autonomic disturbances

PREVENTION

- Immunize the mother during pregnancy
- Clean and safe delivery
- Care of umbilical cord
- Avoid early circumcision in male babies
- Immunize the baby after full recovery

FETAL ALCOHOL SYNDROME

OBJECTIVES

- Define FAS
- State the clinical presentation of babies with FAS
- Outline the physical characteristics of a baby with FAS
- Outline prevention of FAS

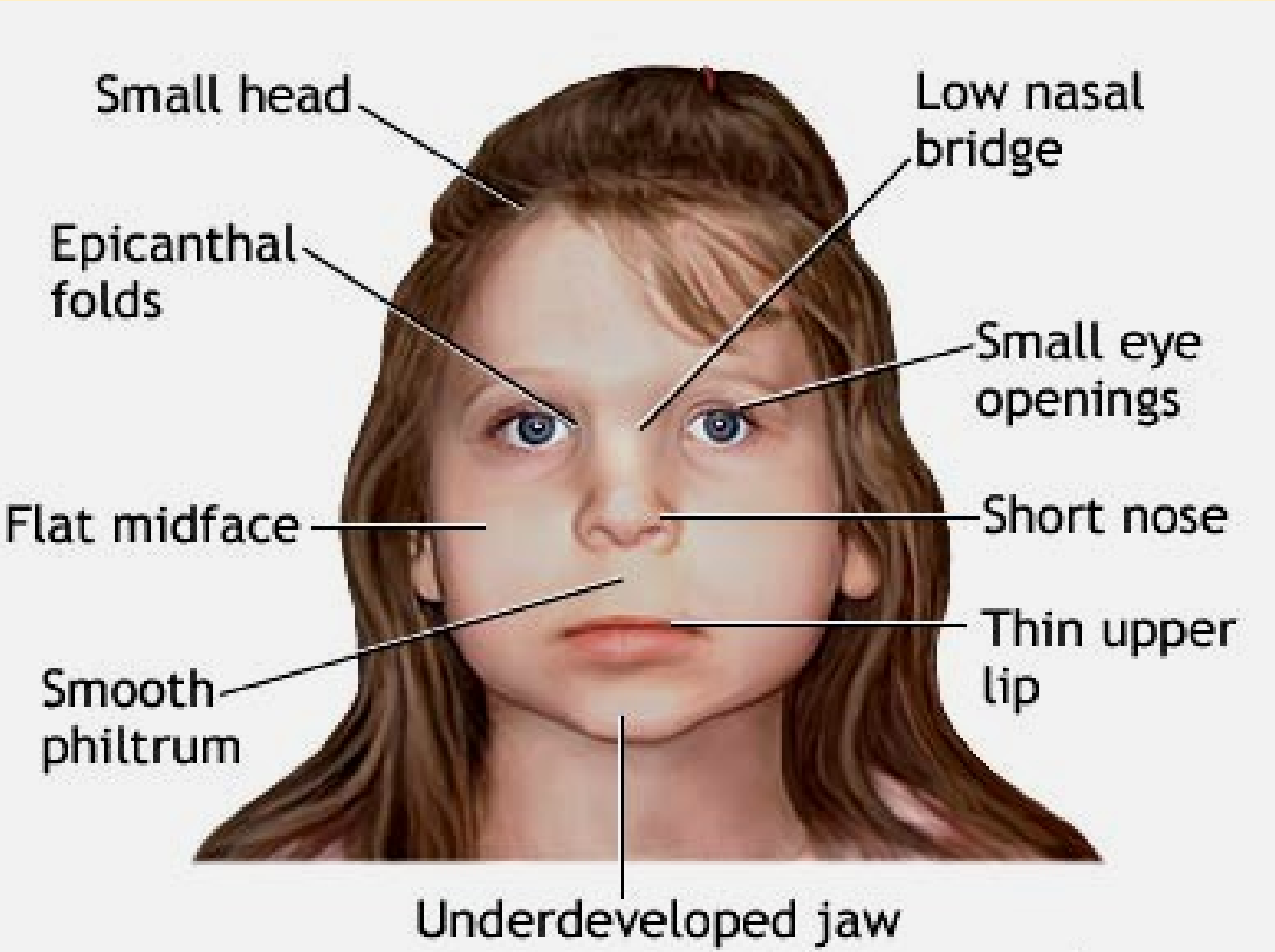
INTRODUCTION

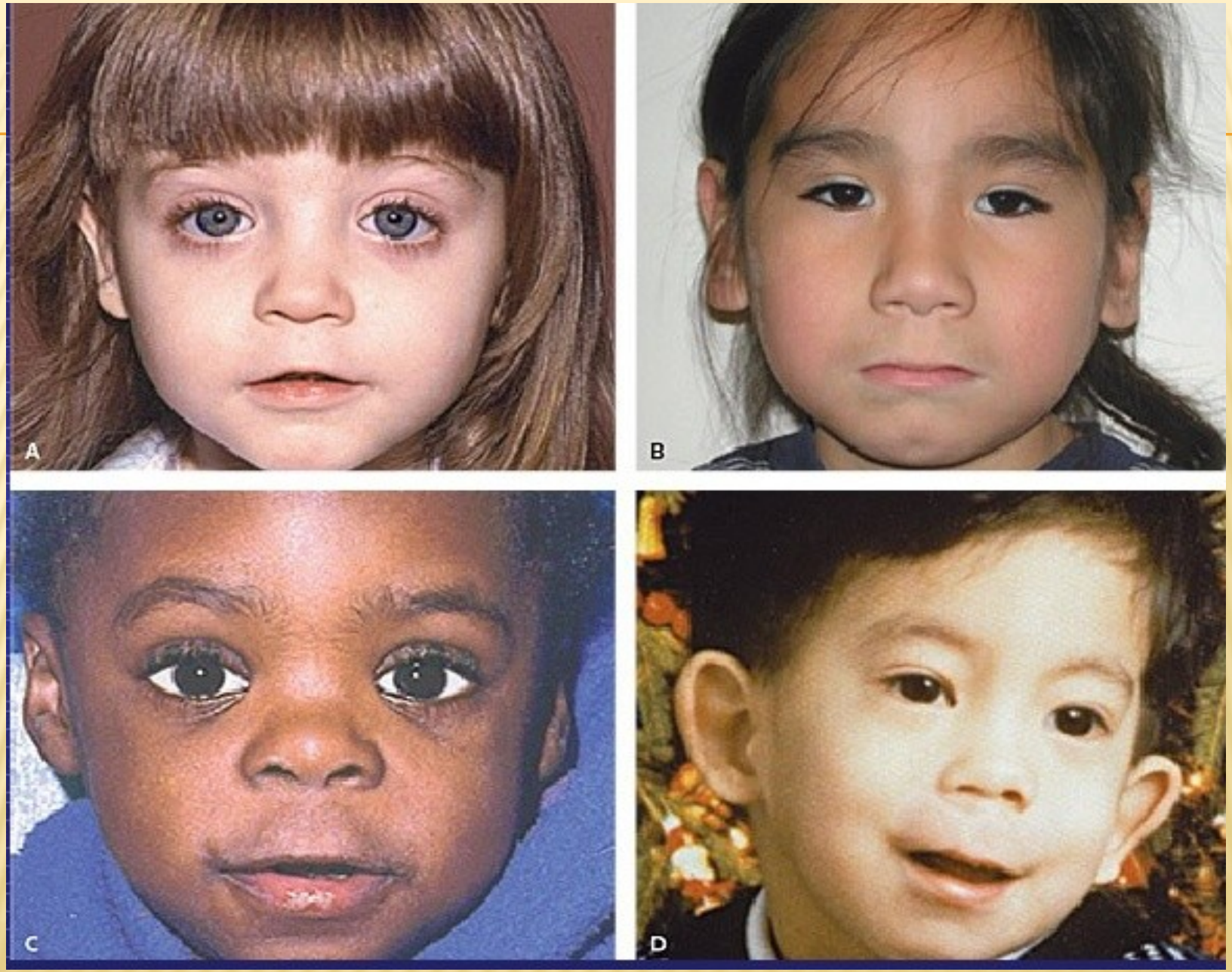
- Fetal alcohol syndrome (FAS) is a pattern of abnormalities with a child caused by exposure of alcohol to a fetus inutero
- It is not known how much alcohol is needed to cause problems with the baby, and so it is generally recommended that no alcohol be drunk during pregnancy

NB: permanent damage can be caused to the fetus when the mother is not aware that she is pregnant

PRESENTATION

- Small size and slow development
- Sleeping difficulties
- Feeding difficulties
- Easily over-stimulated, sensitive to noise and light
- Birth defects such as heart problems, kidney problems, tumors and skeletal anomalies
- Susceptibility to infection
- Mental retardation


Small head
Low nasal bridge
Epicanthal folds
Small eye openings
Flat midface
Short nose
Thin upper lip
Smooth philtrum
Underdeveloped jaw
A.D.A.M.


A
B
C
D

MANAGEMENT

- No specific management
- Create a supportive environment for the parents and children
- identify the specific needs: e.g. learning, safety

ASSIGNMENT

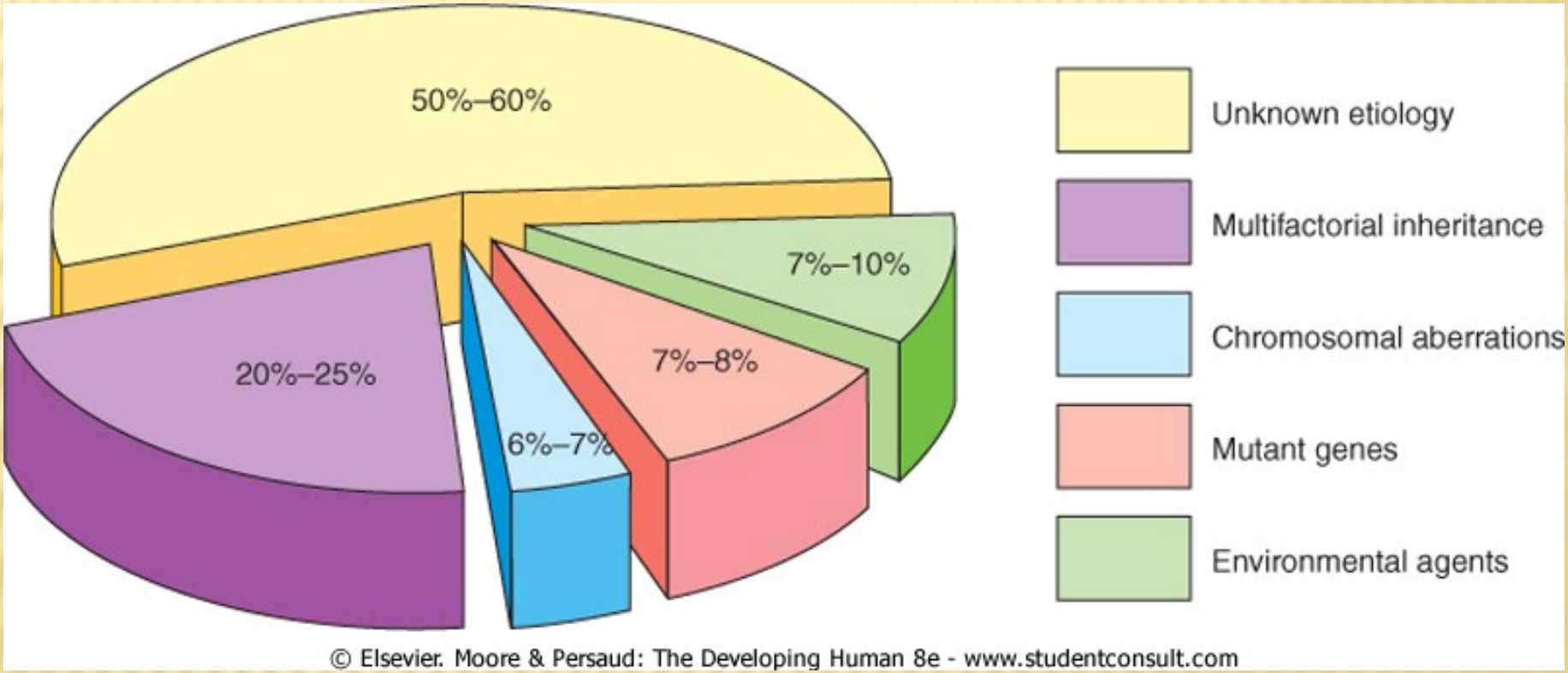
Neonatal Gastroenteritis:

- Definition
- Etiology: Common causative agents: from the most common
- Management: medical, nursing
- Prevention

CONGENITAL ABNORMALITIES

- Congenital malformation is a condition which is present at the time of birth which varies from the standard presentation.
- About 3% of newborns have a "major physical anomaly", meaning a physical anomaly that has cosmetic or functional significance

CAUSES OF CONGENITAL ANOMALIES



1.GENETICS

- Chromosomal defects – caused by too few or too many chromosomes, or problems in the structure of the chromosomes. Example includes Down syndrome (and extra copy of chromosome 21 and sex chromosome abnormalities (missing or extra copies of the sex chromosomes X or Y).
- Single gene defects – a mutation in one gene causes the defect

2. Environmental factors such as radiations, and drugs.

3.Infections: [2%]

- TORCH and Parvo viral infections
- TORCH, which includes Toxoplasmosis, Other (syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus (CMV), and Herpes infections,

4. Maternal Illnesses: [5%]- Diabetes, Epilepsy
5. Drugs: [1-2%]
   - Warfarin, Lithium, Phenytoin
6. Idiopathic – 60%
7. Advanced maternal age – above 40 years – Down’s syndrome or Mongolism
8. High Parity – at risk for malformations except Anencephaly and spinabifida

DIAGNOSIS

Genetic Counselling:

- Recurrence is 6 fold and 70% in second and third pregnancies
- Age, family history, history of past malformations
- Antenatal complications like oligo, poly hydramnios etc.,

COMMONLY-KNOWN BIRTH DEFECTS

- Congenital heart defects
- Cleft Lip and Cleft Palate
- Spina Bifida
- Club Foot Commonly-known Birth Defects
- Down Syndrome

CONGENITAL HEART DISEASES

SIGNS OF CVS DISEASE IN NEONATES

- Cyanosis,
- signs of heart failure other signs include tachypnea, retractions, nasal flaring, shallow respirations, enlarged liver, arrhythmias, murmurs, cardiomegaly, abnormal ECG.

CARDIAC DEFECTS

- **Cyanotic**
- Transposition of the great vessels
- Pulmonary atresia
- TOF
- Tricuspid atresia

- **Acyanotic**
- Patent ductus arteriosus
- Ventricular/atrial septal defect
- Coarctation of the aorta
- Hypoplastic left heart syndrome

DEFECTS

TRANSPOSITION OF THE GREAT VESSELS – c

the aorta originates in the right ventricle rather than the left while the pulmonary artery originates from the left ventricle rather than the right.

ATRIAL SEPTAL DEFECT -a

- An abnormal opening between the right and left atria persists after birth with left to right shunting of blood. This may result from failure of the foramen ovale to close properly.

PATENT DUCTUS ARTERIOSUS-A

- The vascular connection between the pulmonary artery and the aorta which is functional during fetal life persists after birth.
- When it remains open the direction of blood flow is reversed because of higher pressure in the aorta shunting oxygenated from the aortic blood into the pulmonary vasculature (during foetal life the shunt is from the pulmonary artery to the aorta)

VENTRICULAR SEPTAL DEFECT-A

- There is an abnormal left opening between the right and left ventricles. Shunting of blood between the right and left ventricle occurs during systole because of higher left ventricular pressure.
- If pulmonary resistance occurs the shunt is reversed and occurs right to left with resultant cyanosis.

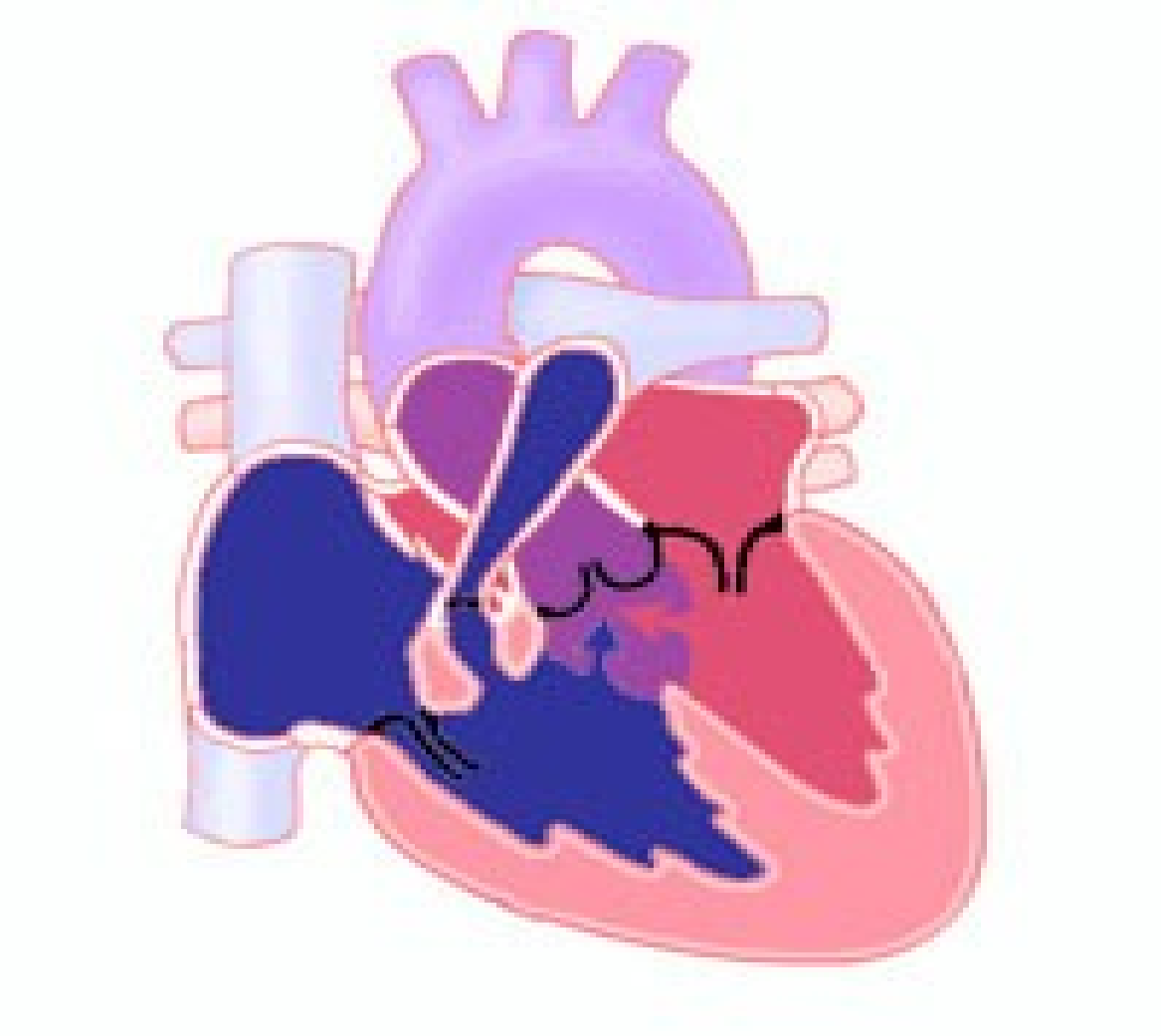
COARCTATION OF THE AORTA-A

- There is a constriction of aorta causing narrowing of the lumen. This partially obstructs blood flow, creating increased left ventricular pressure and work load. The coarctation may occur before or after the ductus arteriosus

TETRALOGY OF FALLOT-C

- Four defects are combined in TOF. There is:
  - pulmonary stenosis,
  - ventricular septal defect,
  - overriding aorta and
  - hypertrophy of the right ventricle.
- Severities of the symptoms depend on the size ventricular septal defect, degree of pulmonary stenosis and the degree to which the aorta overrides the septal defect.

TOF



MEDICAL MANAGEMENT

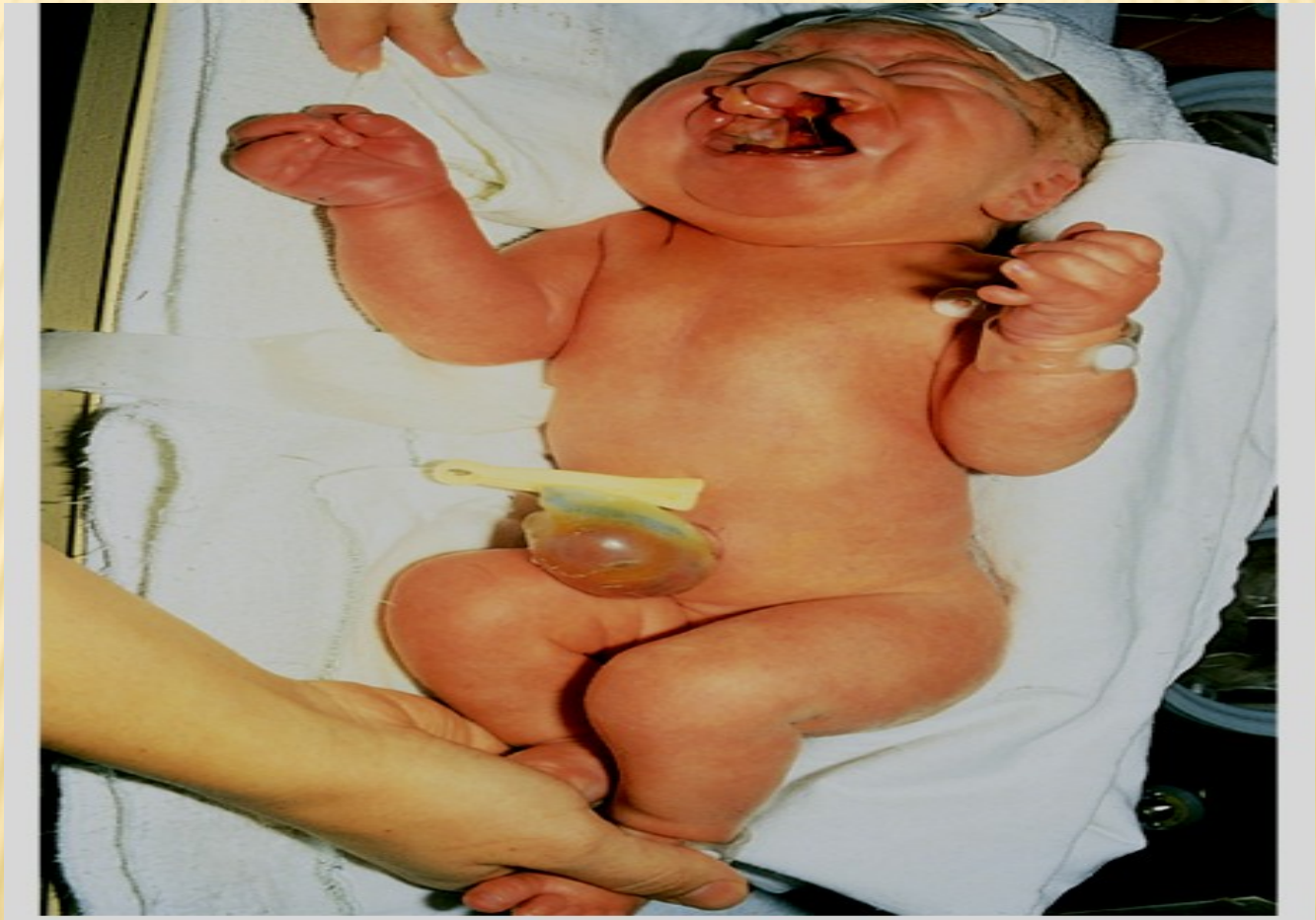
- Diagnosis is done through cardiac catheterization as well as by angiography
- Once the lesion has been identified it can be corrected surgically or by use of medication such as: Digoxin, morphine, prostaglanding infusion, diuretics, and use of supplementary oxygen
- Monitoring of fluid and electrolyte balance.

NURSING CARE

- Preparing the infant and assisting in diagnostic procedures
- Provide small frequent feeds
- Maintain input and output fluid records
- Administering oxygen and monitoring blood gas status
- Ensure safe administering of digoxin by checking dosage and route with another nurse
- Monitor apical pulse and withhold digoxin if less than 90.

GASTROINTESTINAL MALFORMATIONS

CLEFT LIP AND CLEFT PALATE



INTRODUCTION

- This results from failure of the soft or bony tissue of the palate and the upper jaw to unite during the fifth to tenth weeks gestation. It may be unilateral or bilateral. Factors associated with these conditions include: genetic factors, hypoxia, maternal viral infections and nutritional factors.

SURGICAL MANAGEMENT

- When only the lip is involved, surgery can be done within the first day of life. When the Palate is involved, the repair is usually postponed to 6 to 24 months. The infant should be fit nutritionally and free of infections, complications of this surgery include speech problems and respiratory tract infections.

NURSING CARE

- Reassure the parents and ensure that the surgical team meets with the parents to explain the procedure.
- Using a feeding bottle would be appropriate as sucking of the nipple strengthens the muscles.
- Place the infant in an upright position and direct the flow of milk against the side of the mouth to decrease the possibility of chocking.
- Mother needs to be told that feeding is important to ensure adequate growth until surgery can be performed

- Thickened formulas are often used.
- Show mother how to feed the baby with breast milk
- Note that babies with minor clefts can breastfeed
- However those with bilateral clefts must be fed by cup and spoon
- Take care to prevent aspiration

POST OPERATIVE NURSING CARE

- Cleft lip repair- position the child on one side to avoid injury to the incision site
- After feeding cleanse the suture line with a cotton tipped swab dipped in normal saline. Administer antibiotics as prescribed.
- Cleft palate repair- Infant can be positioned on the abdomen.
- Feedings are resumed by bottle, breast or cup.
- Encourage parents to hold the infant.

OTHERS INCLUDE:

- Gastroschisis
- Ompholocele
- Atresias- Esophageal and rectal
- Hirchsprung’s disease

- Define the above

CENTRAL NERVOUS SYSTEM ABNORMALITIES

SPINA BIFIDA


Spina Bifida
Myelomeningocele
Meningocele
Spina Bifida Occulta

- It is a midline defect of vertebral discs without protrusion of spinal cord and meninges.
- This is a common malformation of the spinal column and is due to congenital lack of one or more vertebral discs, usually at the lower part of the spine.(usually involving L5 and S1)
- Patches of hair and discoloration of the skin in the midline of lower back signifies malformation of the spinal cord.
- When the membranes covering the spinal cord bulge through the opening the condition is known as **meningocele** (a soft tumor filled with CSF fluid is formed). The tumor can be influenced by pressure.
- Surgical closure should be done as soon as possible to prevent further deterioration of the spinal cord

- **Myelomeningocele**-both the spinal cord and the meninges herniate through the defect in the vertebral column. Most cases involve the lumbo-sacral region but may exist anywhere in the neural axis.
- **Encephalocele** – abnormality that affects the skull and result in protrusion of tissue through a bony midline defect called **cranium bifida.**
- The two forms are:
- cranial meningocele-herniation of CSF filled meningocele sac through the cranium bifida.
- Cranial encephalocele- contains a sac plus cerebral cortex, Cerebellum and a portion of the brain stem herniating out.

AETIOLOGY AND RISK FACTORS

- Genetic determinants
- Malnutrition- folic acid deficiency
- Drugs e.g. anticonvulsants
- Radiation

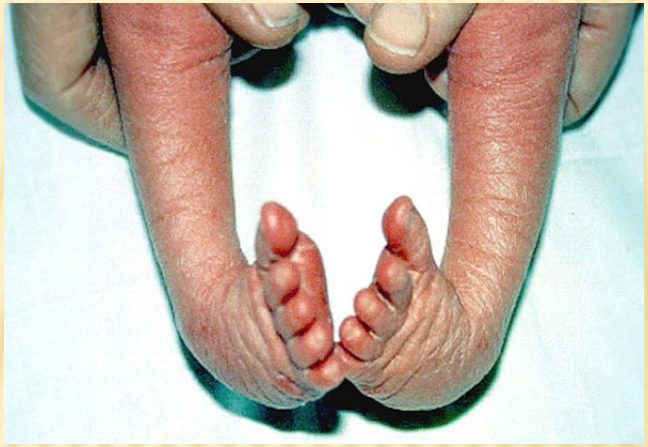
NURSING CARE

- Ensure the infant is nursed in a prone position
- Ensure the area remains clean and free from contamination by urine or feces.
- Ensure infection prevention to the defect site
- Provide support to the parents.

- If the defect is not covered by skin:
- – Cover with sterile gauze soaked in sterile normal saline
- – Keep gauze moist at all times and ensure that the baby is kept warm
- – If ruptured give Benzyl Penicillin 50,000units/kg 12hrly and Gentamycin 5mg/kg daily for 5 days

MUSCULOSKELETAL DEFORMITIES

TALIPES EQUINOVARUS



INTRODUCTION

- Also known as club foot, a general term used to describe a range of unusual positions of the foot.
- Each of the following characteristics may be present, and each may vary from mild to severe:
- The foot (especially the heel) is usually smaller than normal.
- The foot may point downward.
- The front of the foot may be rotated toward the other foot.
- The foot may turn in, and in extreme cases, the bottom of the foot can point up.

RISK FACTORS

- Previous history
- Multiple pregnancy
- Macrosomic fetus
- Oligohydramnios

- More common in boys than girls
- May be unilateral or bilateral

MANAGEMENT

- Apply plaster of Paris
- Revise every 1-2 weeks
- Refer if no response after 3 months

- Talipes calcaneovalgus- foot is dorsiflexed and everted.

DOWNS SYNDROME

- Also known as trisomy 21, is a genetic disorder caused by the presence of all or part of a third copy of chromosome 21. It is typically associated with physical growth delays, characteristic facial features, and mild to moderate intellectual disability.
- A child with trisomy 21 has three copies of chromosome 21 instead of the usual two copies in all of his or her cells.


RESORT

SIGNS AND SYMPTOMS
1.PHYSICAL CHARACTERISTICS

Down syndrome can vary from child to child.

Some of the most common characteristics are as follows:-

- Flat face,
- Broad forehead,
- Short neck,
- Narrow slit to the eyes,
- White spots in the iris of the eyes (known as Brushfield spots)
- Small, abnormally shaped ears,
- Small depression near the nose with a somewhat flattened bridge,
- Small mouth,

SIGNS AND SYMPTOMS CONT.

- Slightly protruding tongue(the tongue may be abnormally big),
- Short arms and legs,
- Short fingers and toes with the little fingers curving inwards,
- a single horizontal, deep crease in the palms,
- Large space between the first and second toe on each foot,
- weak reflexes,
- muscle hypotonia
- hyperflexibility of joints

2.MENTAL CHARACTERISTICS

- Most individuals with Down syndrome have mild (IQ: 50–70) or moderate (IQ: 35–50) Intellectual disability with some cases having severe (IQ: 20–35) difficulties.
- Slow rate of learning and information processing
- They also have trouble expressing what they have learned or understood via the conventional outlets of speech and writing

ASSOCIATED MEDICAL CONDITIONS

- Congenital heart defects- Atrial and ventricular septal defects are the most common congenital heart defect.
- Gastrointestinal disorders- including the anatomical abnormalities such as imperforate anus, and functional disorders such as gastroesophageal reflux, and malabsorption.
- Respiratory problems such as frequent cold, cough and flu.

- Skin problems -Infants with Down syndrome have very soft skin. As they grow older, their skin becomes coarse and dry. Atopic dermatitis or atopic eczema is the main skin problem found in children with Down syndrome.
- They may suffer from epilepsy, hypothyroidism, hyperthyroidism and a shortage of growth hormones.

NURSING MANAGEMENT

- **Physical Therapy**
- focuses on motor development. Since most children with Down syndrome have hypotonia or low muscle tone.
- Early intervention is a program of therapies, exercises and activities designed to specifically help children with Down syndrome or other disabilities.

NURSING MANAGEMENT CONT..

- Encourage use of other means of expressions such as pictures, colors, sounds, or any other media, to express themselves, for those babies who cant express themselves convectionally on what they have learnt or understood.

NURSING MANAGEMENT CONT..

- **Surgical Treatments**
- Incases of congenital heart defects and intestinal defects
- **Medications**
  Depending on the presenting problem in downs syndrome ie anticonvulsants etc

NERVE INJURIES

OBJECTIVES

By the end of the lesson, students should be able to:

- Discuss different types of nerve injuries
- Differentiate brachial plexus injuries: Erb palsy and Klumpke’s palsy
- Discuss the management of nerve injuries
- State the prevention measures for birth injuries

TYPES OF NERVE INJURIES

1.Cranial nerve injuries

- facial nerve injuries ,recurrent laryngeal nerve injury

2.spinal cord injuries

3.Cranial nerve root injuries

phrenic nerve palsy , injuries to brachial plexus

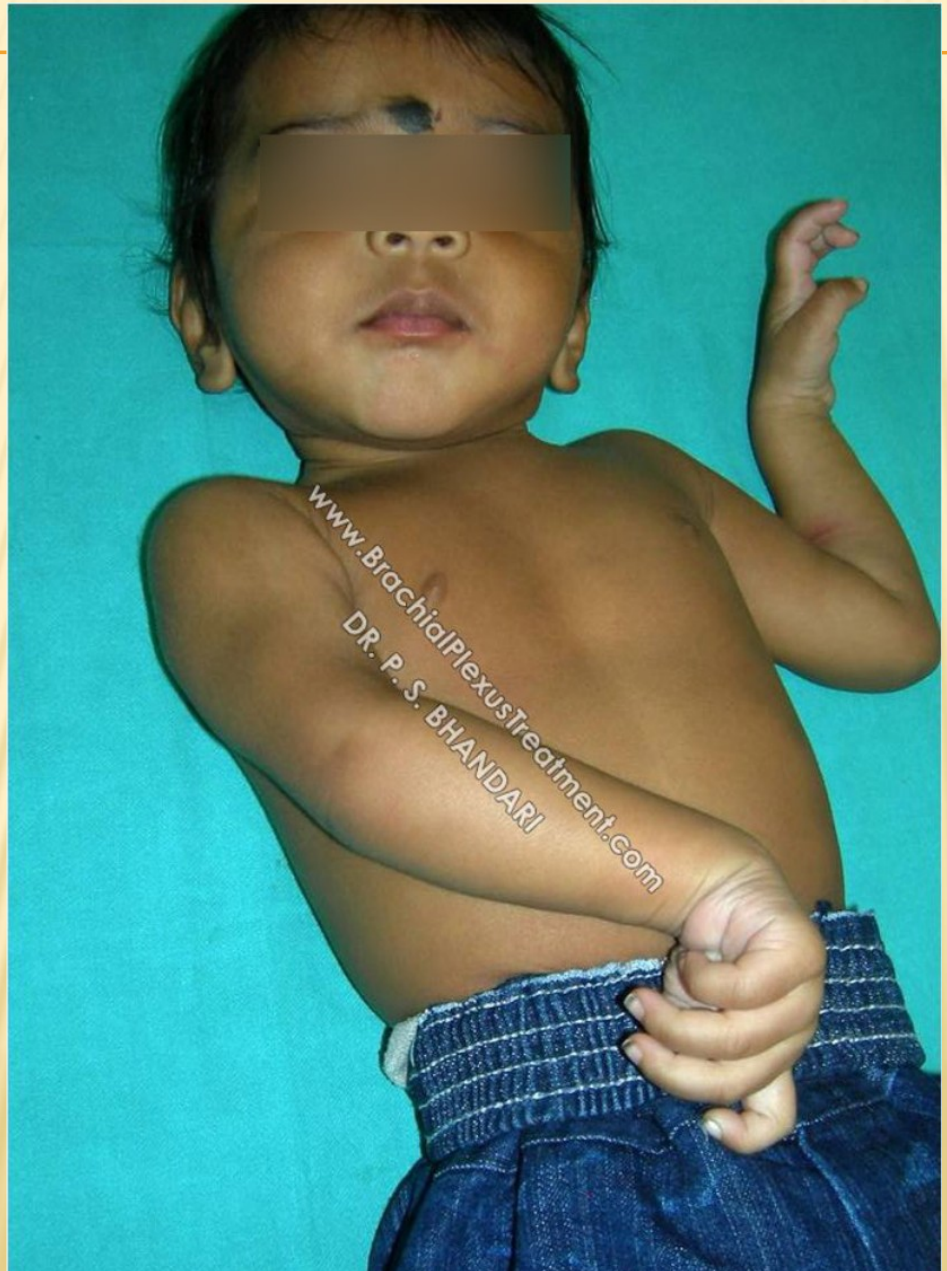
BRACHIAL PLEXUS INJURIES

- Brachial plexus comprises of nerve roots at the level of 5^{th}, 6^{th}, 7^{th} and 8^{th} cervical vertebrae and the 1^{st} thoracic vertebrae.
- Injuries are mainly 3:
  - Erb's palsy
  - Klumpke's palsy
  - Total brachial plexus palsy

DUCHENNE-ERB PARALYSIS (C5-C6)

- Affected arm is adducted and internally rotated with elbow extended (Waiter’s tip position)
- Forearm is prone and wrist is flexed
- The limb falls limply to the side of the body when passively adducted
- Moro’s, biceps, radial reflexes absent on the affected side
- Grasp reflex intact

INFANT WITH ERB PALSY



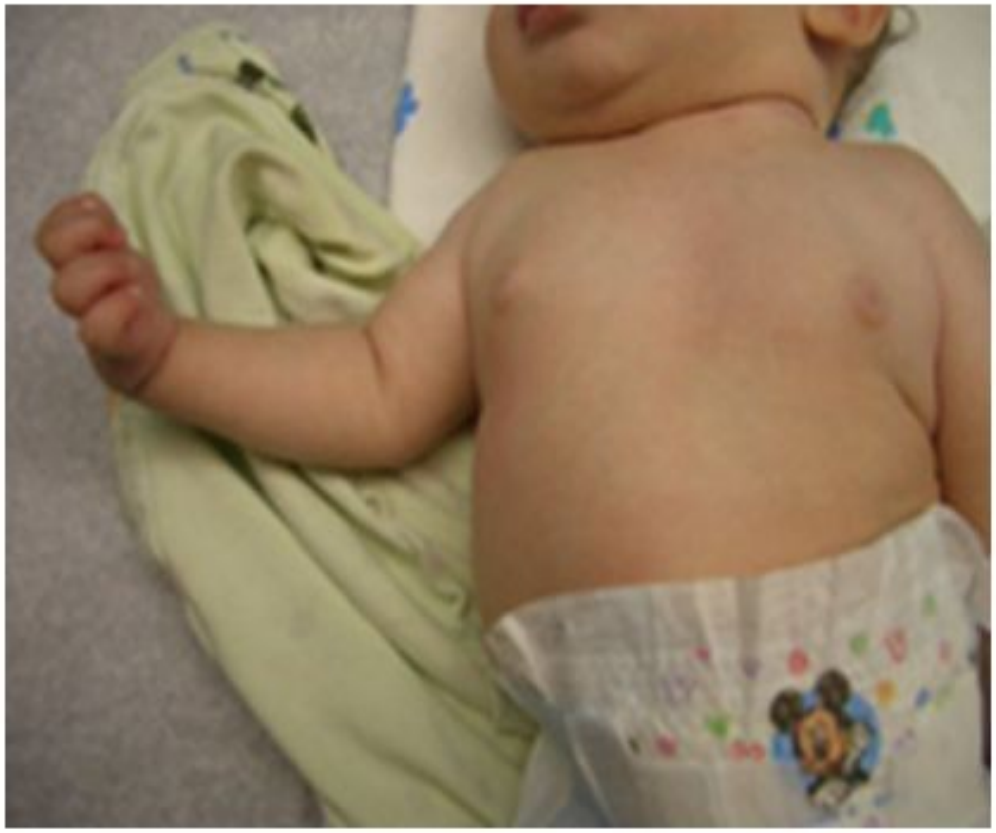
Klumpke’s paralysis (C7, C8 & T1)

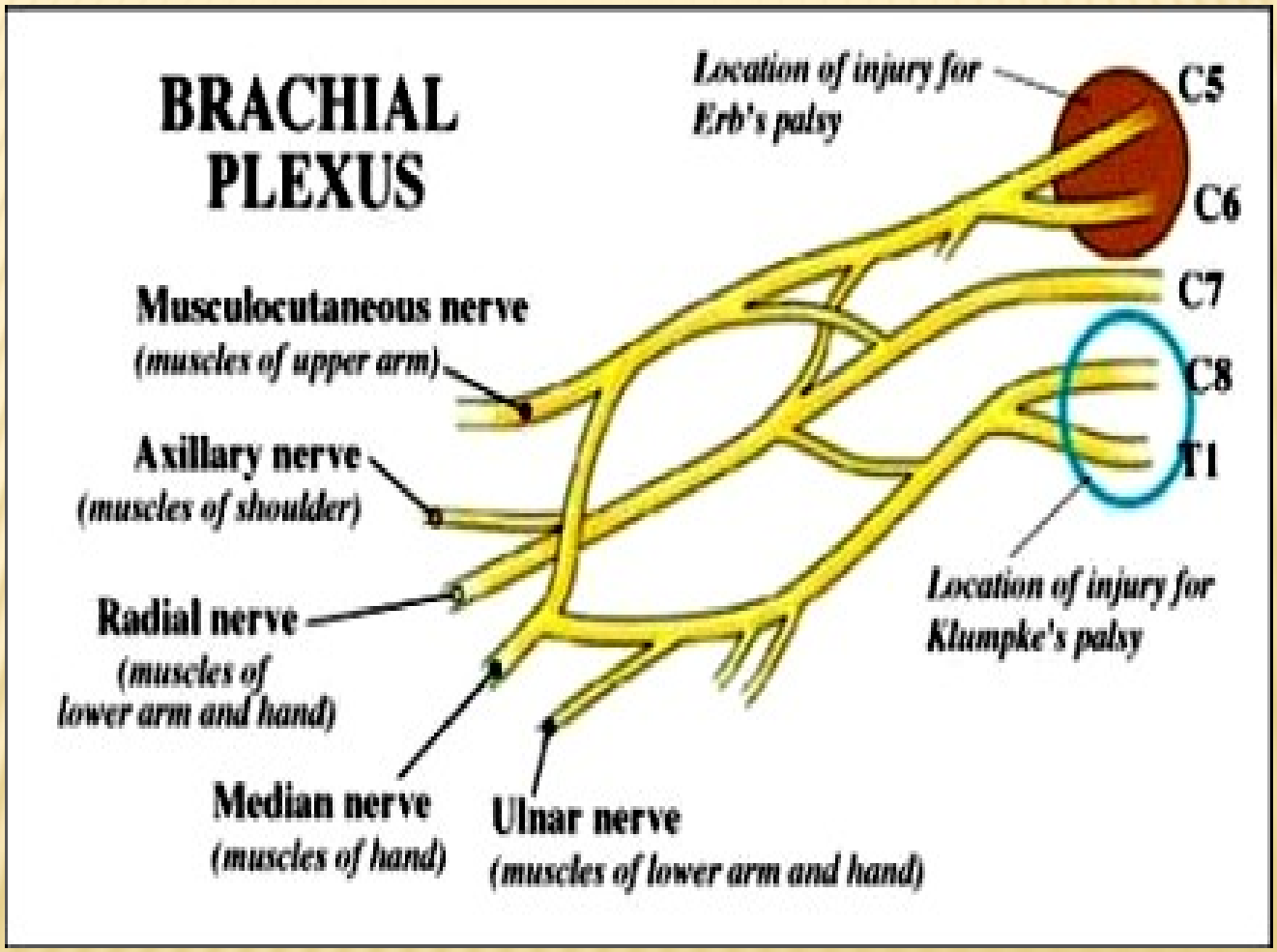
- Intrinsic muscles of the hand are affected and grasp is absent (claw hand)
- Biceps and radial reflex are present

TOTAL BRACHIAL PLEXUS PALSY

- Injury to the entire brachial plexus- the entire arm is flaccid, all reflexes are absent
- In cases of bilateral paralysis, spinal cord injury should be suspected

KLUMPKE’S PARALYSIS (IMAGE)




BRACHIAL PLEXUS
Location of injury for Erb's palsy
C5
C6
C7
C8
T1
Musculocutaneous nerve
(muscles of upper arm)
Axillary nerve
(muscles of shoulder)
Radial nerve
(muscles of lower arm and hand)
Median nerve
(muscles of hand)
Ulnar nerve
(muscles of lower arm and hand)
Location of injury for Klumpke's palsy

MANAGEMENT OF NERVE INJURIES

- X-ray studies to rule out bony injuries
- u/s and assessment of joints
- Chest examination to rule out diaphragmatic involvement
- Passive movements started after 7-10 days (after resolution of the nerve edema)
- Splints to prevent wrist and digit contractures

Nursing management:

- The goal of care is to prevent contractures of the paralyzed muscle
- The arm should be partially immobilized in a position of maximum relaxation so that the non-paralyzed muscles cannot exert pull on the affected muscles
- By the use of splint or brace when the arm is paralyzed, the arm is abducted 90 degrees and rotated internally at the shoulder with the elbow flexed so that the palm of the hand is turned towards the head

NURSING MANAGEMENT CONT..

- When the lower arms and hand are paralyzed, the lower arm and the wrist are kept in a neutral position and the hand is placed over a small pad
- The infant is immobilized for 6 months during part of the day and night
- A longer period of immobilization may be necessary for some infants
- After 7-10 days, a complete range of motion (ROM) exercises may be given gently several times each day in order to maintain muscle tone and prevent contraction deformity

Nursing management cont..

- When any form of immobilization is used, the fingers and hands must be observed for any coldness or discoloration and the skin for signs of irritation
- When a splint is used, the parents must be taught how to apply it properly and how to provide skin care

NURSING MANAGEMENT CONT..

- They should be taught the proper dressing technique-affected hand first and on removing the unaffected hand first
- More physical contact and affection should be encouraged than a normal child

COMPLICATIONS

- Severe hypotension and bradycardia
- Nerve damage or neuritis
- Horners syndrome with dyspnea and hoarseness of voice
- Puncture of the pleura may cause pneumothorax
- Hemothorax
- Hematoma and infection.

WHAT WE HAVE LEARNT

- types of nerve injuries
- Differences between : Erb palsy and Klumpke’s palsy
- The management of nerve injuries
- prevention measures for birth injuries
- Complications of nerve injuries

ASSIGNMENT

- Discuss the care of the mother with a baby admitted to a Special care baby unit

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