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# INTRODUCTION

## 1.1 ADMISSION CRETERIA INTO THE NEWBORN UNIT

The new born unit does not only admit babies at risk but also offers accommodation to normal neonates due to unstable condition or death of the mother.

Reasons for admitting a baby into the nursery include the following:

* Pre-maturity
* Asphyxia neonatorum
* Haemorrhagic disease of the new born
* Ophalmianeonatorum
* Birth injuries
* Congenital abnormalities e.g hydrocephalus
* Respiratory distress syndrome
* Infants of diabetic mothers(risk of hypoglycaemia)
* Maternal death
* Unstable maternal condition

## 1.2 INFECTION CONTROL IN THE NEW BORN UNIT

Due to low immunity of the babies in the NBU, infection control is critical to protect the babies from infection during their stay in the unit. This is necessitates high infection control measures within the unit.

The following are some ways of ensuring infection control in the nursery.

* Keep the unit clean, free from dust. The windows should remain closed at all times to prevent flowing in of dusty air.
* Daily dump dusting and cleaning of the incubator and cots
* Isolation of infected babies for barrier nursing.
* Restriction of visitors to ensure adequate control of human traffic into the nursery. Visitors should see the babies through the window glass.
* Washing hand before and after handling the baby for any procedure.
* Strictly observing aseptic technique while performing procedures.
* Feeding utensils should be rinsed, decontaminated, cleaned thoroughly in soapy water and kept in presept till the next feed.
* Staff working in isolation room should not move into other nurseries
* Cleaning of incubators upon discharge or death of a baby, before the next baby is put.
* Mothers changing clothes whenever they come to feed the babies.
* Health educating the mothers on the importance of personal hygiene and care of the baby.(Myles; 2nd Edition by Diane M. Fraser ,Margaret A. Cooper , and Anna G. w. Nolte ).

## 1.3 FIRST EXAMINATION OF THE BABY

This is a routine procedure done after third stage of labour in labour ward but is also done in the nursery as part of admission procedure. The aims are;

* To rule out congenital abnormalities
* To rule out birth injuries
* To assess maturity of the baby

**Head**

* measure the head circumference (average is 35 cm
* Check for the moulding of the foetal skull
* Width of the fontannelles and sutures, bilging of fontanel and wide sutures may indicate hydrocephalus
* Depression on the skull may imply a fracture
* Injuries e.g caput succadenium and cephalohaematoma

**Eyes**

* absence of eyebrows
* Conjuctivalhaemorrage / bleeding
* Any discharge and squint

**Nose**

* Check for any deformities e.g.Well formed septum
* Bleeding from the nose
* Check for nasal flaring which is a sign of respiratory distress
* Check for blocked nostrils

**Mouth**

* Bleeding from the mouth
* Check for tongue tie
* Abnormalities e.g cleft palate or cleft lip
* Frothing of the mouth

**Ears**

* Bleeding from the ears
* Leakage of CSF through the ears
* Shape of the lobes to rule out malformations
* Extra lobe of the ears

**Neck**

* Check out for abnormalities e.g. congenital goiter
* Check for meningocele

**Chest**

* Shape of the chest for symmetry
* Chest movement during respiration
* Take apex beat
* Check breast for swelling and discharge

**Abdomen**

* Check for skin colour and presence of rashes
* Check whether the cord is well ligated
* Bleeding from the umbilical cord
* Abdominal abnormalities e.g. hernia

**Genitalia**

* For males check for the testis to rule out undescended testis
* Female check for vaginal discharge, labia should be well formed size of a clitoris

**Hip joint**

* Rule out congenital hip dislocation

**Limbs**

* Check if arms and hands are moving freely
* Rule out dislocation, fractures and Erb’s paralysis
* Check for equality of the arms and to rule out abnormalities
* Fingers for webbed and extra digits
* Legs for equality, abnormalities and movement
* Rule out tallipes and club foot

**Back**

* Abnormalities of the backe.g.spina bifida, myelomeningocele
* Check for skin colour and septic spots

**Anus**

* While taking rectal temperature, check for imperforate anus
* Bruises on the skin or rashes

**Check for the following reflexes:**

* Sucking reflexes – full term infant sucks the small finger
* Moro reflex – tested by gently lifting the baby up by its fingers from a flat surface and suddenly releasing it. It will respond byspreading its hands then move them together as though hugging.
* Rooting reflex – the baby turns in search of the nipple
* Grasping reflex – it will grasp your finger if you put it in its palm.
* Stepping reflex – when held on a flat surface in standing position, it makes stepping movement.

# 2.0 NORMAL NEONATE

This refers to a baby born at term or as near term as possible after 37 weeks of gestation and has no complications.

Upon birth the infant has to undergo physiological changes in order to adapt to life outside the uterus to have independent existence.

## 2.1PHYSIOLOGICAL CHANGES AT BIRTH

1. **Respiration** occurs due to:

* Low oxygen and high carbon- dioxide stimulates respiratory center and respiration begins
* Compression of the chest wall during second stage creates a vaccum and aid respiration
* External stimuli e.g handling the baby, cold extra uterine environment makes the baby gasp and respiration starts
* Baby is encourage to cry initially by flicking the sole of the foot for it allows complete aeration of the lungs
* Presence of surfactant factor aids expansion of the lungs (lecithin:sphingomyelin =2:1 and is an indicator of lung maturity detectable on amniocentesis)

The normal respiration rate at birth is 40-50/ min

Irregular breathing may be due to the following factors:

* Prematurity (inadequate surfactant factor)
* Depression of the respiratory centre by drugs e.gpethedine or strong uterine contractions
* Excessive carbondioxide (hyperpnoea)
* Lack of oxygen (hypoxia)

**2. Circulatory system**

* Extrauterine circulation is established and the baby is able to divert deoxygenated blood to the lung for deoxygenation. This accounts for the pink colour of an infant.
* In utero the Hb is high 18-20g/dl and high RBC to transport sufficient oxygen to the foetus. After birth the Hb drops to 14g/dl and some of the RBC are broken down by the liver cells to bilirubin and may lead to physiological jaundice.
* Normal heart rate inutero is 120 -160 beats per minute but upon birth it drops to 100 -120 beats /min

**3. Temperature regulation**

* Temperature in utero is 38\*C but the baby’s rectal temperature is 37\*C. The temperature drops due to evaporation, conduction, convection and radiation.
* Temperature is not adequately regulated due to low metabolic rate and insufficient heat regulating center in the hypothalamus. They are at risk of overheating as well as chilling.
* They have thin subcutaneous layer which provides poor insulation and heat is lost. They have brown adipose tissue to mobilize heat resources.

**4. Digestive system**

* Sucking and swallowing reflexes are present and they feed on colostrums and pass meconium (initially green then turns yellow)
* They open bowels 3-4 times a day.

**5. Liver**

* Stars functioning in utero although negligible. Its function remains depressed for a few days yet it has to handle excess Hb thus there is accumulation of bilirubin leading to physiological jaundice.

**6. Urinary system**

* A kidney start functioning in utero and the foetus passes urine but has no ability to concentrate urine thus excretes chlorides and phosphates.
* The baby should pass urine within the first 48 hours of birth.

**7. Weight**

* Average birth weight is 2.5 -3.5 kgs but is affected by factors such as period of gestation, placental function, nutritional status of the mother, size of the parents, type of pregnancy i.e. single or multiple and sex of the baby.
* In the first three days the baby looses 1/10 of its weight (physiological weight loss) due to limited intake, loss of meconium and loss of tissue fluid. The weight is gained within 7-10 days then it gains 250-500g weekly.

## 2.2 CHARACTERISTIC OF NORMAL NEONATE

Weight is 2.5 -3.5 kgs.

Length from vertex to heel is 45-52 cm

Head circumference is 35 cm and increases by 1-2 cm during the first month

Fontannelles and sutures are patent. Anterior fontanel closes at 18 -24 months while the posterior closes at 6-8 weeks.

Skin is covered by vernixcaseosa, a secretion of the sebaceous gland that helps in heat retention and acts as a lubricant during delivery.

Umbilical cord shrivels by necrosis and falls off in 7 days. The remaining part forms abdominal ligaments. Hernia may develop but usually disappears spontaneously.

Reflexes are fully developed.

Senses are developing.

# 3.0 PRE TERM BABY

 This refers to a baby born before 37 complete weeks of pregnancy. Some of them may have growth retardation and therefore be small while others may be excessively large for gestational age (macrosomia)

Low birth weight baby is one with less than 2500g

## 3.1 predisposing factors

1. Maternal factors – maternal ageeg. Primigravida below 17 years or above 35 years
2. Maternal disease in pregnancy such as anaemia, hypertension, pre-eclampsia.
3. Foetal factors –congenital abnormalities; multiple pregnancy and polyhydromnous due to over distension of the uterus; rhesus incompatibility interfering with foetal viability
4. Placental factors –APHdue to placenta praevia and placenta abruption
5. Social factors –strenuous exercises, excessive drinking of alcohol and smoking, previous history of miscarriage, physiological stress.

## 3.2 clinical features

* Small stature with low birth weighs less than 2500g
* Thin and sparsely distributed hair on the head.
* Skin is reddish with plenty of lanugo
* Widely open sutures
* Eyes closed most of the time
* Pinnae of the ears are soft and fold easily on pressure and slow to uncoil
* Narrow sinuses and the nose a bit flat
* Swallowing and sucking reflexes absent or very weak
* Weak cry and there are no tears
* Chest is small, soft with underdeveloped breast tissue
* Poor muscle tone and the baby lies inactive most of the time
* In females, labia majora are widely separated and labia minora is protruding in between
* In males, scrotal muscles are smooth and testis are undescended
* Palmer and planter creases are absent
* Grasp reflex are absent

## 3.3 physiology of the pre term baby

1. Immunity is low due to:

* Low gamma globulins responsible for immunity.
* Delicate skin that is vulnerable to injuries and infection
* Lack of passive immunity which usually develops around 38 weeks gestation

2. Blood system

* Has poor peripheral circulation with high tendency to hemorrhage because of weak vascular walls.
* Prone to hemorrhage due to lack of clotting factors(vitamin A is administered to promote clotting)
* Unable to store iron hence at risk of iron deficiency anemia.
* They have very few blood cells and may develop non pitting anemia

3. Weight

* Initially they lose up to 10% of their birth weight and start gaining and reach birth weight 2-3 weeks post delivery.

4. Temperature regulation is poordue to:

* Immature heat regulatory centre
* Limited food intake and low metabolic rate
* Inability to shiver and generate heat
* Excessive heat loss due to little or nosubcutaneous fat. The brown fat is usually in baby’s body by 36 weeks gestation.

5. Respiratory system

* Under developed respiratory centre leading to difficulty in initiation of respiration.
	+ Frequent apnoeic attacks with irregular respiration.
* Abdominal movements more than chest movements.

6. Renal system

* Immature kidneys are unable to concentrate urine hence they excrete chlorides and phosphates.

7. Digestive system

* Absence of swallowing and sucking reflexes lead to poor feeding
* Regurgitation after feeds due to underdeveloped cardiac sphincter

8. Nervous system

* All regulatory centres are under developed.

## 3.3 NURSING MANAGEMENT

* Delivery of a preterm baby should be conducted in a warm room and subsequently nursed in a preterm incubator.
* Temperatures of the incubator should be maintained within normal range of about36 – 37 \*c
* Perform first examination of the baby to assess maturity.
* Fix NGtube and the baby with breast milk and substitute only where breast milk is not available.
* Feed the baby using the oral feeding regime:
* Baby is given 60- 65 mls per kg of body weight in 24 hrs in 8 divided doses e.g. 2.5 kg baby will have *2.5 x60/8 =18.99 mls* per feeding thus should be fed 3 hourly.
* If the baby tolerates, the feed can be increased
* If the baby can’t tolerate the oral feeds, give IV fluids e.g. 10% dextrose
* Introduce cup and spoon feeding gradually as the baby gains weight
* Aspirate the gastric content to rule out indigestion.
* Close observation to include:

-vital signs TPR

- Respiratory rhythm to note apnoeic attack

- Umbilical stump for signs of infection

-vomitting or retaining food

- general activity and emotional status

* Provide care of IV line i.e. securing, cleaning and dressing.
* Give nutritional supplements e.g. iron , folic acids, vitamin from the second week.
* Administer broad spectrum antibiotic prophylactically for prevention of infection
* Take weight on alternate days to monitor the progress.
* Discharge the baby at 2000 – 2500g
* Give BCG vaccine on discharge or advice the mother to go for it.
* Advice mother on family planning so that she gets another baby by choice and not by chance.

## 3.5 COMPLICATIONS

1. Hypothermia neonaterum
2. Haemorrhagic disease of the newborn
3. Respiratory distress syndrome
4. Retrolental fibroplasias
5. Failure to thrive
6. Jaundice
7. Infections
8. Anaemia
9. Rickets

# 4.0 SMALL FOR GESTATIONAL AGE

This term refers to a baby whose birth weight is below 10th percentile of his gestational age commonly referred to as low birth weight but this includes preterm babies.

They are susceptible to various problems including:

* Congenital abnormalities
* Foetal hypoxia that may lead to intrapartal death
* Birth asphyxia due to inadequate perfusion, meconium aspiration leading to airway obstruction.
* Hypothermia due to little subcutaneous tissues
* Apnoeic attacks hypoglycemia

## 4.1 SIGNS AND SYMPTOMS

* Mostly they are born after 37 weeks.
* Pale, dry loose skin with wrinkles and have little or no lanugo
* Subcutaneous fat is minimal
* Shows features of retarded growth
* The abdomen appears sunken
* Sutures and fontanel appear normal
* Eyes are alert and has mature facial expression
* Skull bones are hard and allow little mobility
* Have strong cry
* Umbilical cord is thin
* Swallowing and sucking reflexes are present so they feed well
* Normal muscle tone are active

## 4.2NURSING MANAGEMENT

* The baby is predisposed to the risks similar to those of preterm baby thus the management principles are the same.
* Management should start in labour by closely monitoring foetal condition for signs of foetal distress.
* In case of foetal distress in the first stage, administer oxygen to the mother and start IV drip of 10% dextrose as you prepare the mother for emergency caesarian section. If in second stage, the delivery is hastened by giving generous episiotomy.
* Since the baby is prone to hypoglycaemia, it should be stared on breastfeeding as soon as possible.
* Gastric lavage should be done with warm dextrose before breastfeeding.
* Substitutes are given if there is no breast milk. The feed is calculated at 90 mls/kg of body weight in 24 hrs in 8 divided doses i.e. 3 hourly feeding.
* Closely observe vital signs TPR and signs of infection.
* The baby should be nursed in a warm environment to prevent hypothermia although it has temperature regulating mechanism.
* Closely monitor blood sugar to rule out hypoglycaemia.
* Weigh the baby on alternate days to monitor the progress. Usually weight loss is minimal and it gains weight more rapidly and steadily than preterm.
* Teach the mother how to take care of the delicate skin that may be dry, cracked or peeling.

## 4.3COMPLICATIONS

* Hypoglycaemia
* Respiratory distress syndrome
* Aspiration pneumonia
* Brain damage

# ASPHYXIA NEONATORUM

This is a term which refers to a condition in which the baby fails to breath at birth.

## 5.1TYPES OF ASPHYXIA

 The degree of asphyxia is determined by Apgarscorein which the following features are observed and score 0-2

* Appearance (colour of the body)
* Pulse (heart rate)
* Grimace (response to stimuli)
* Activity (muscle tone)
* Respiration /respiratory effort

A score between 8- 10 does not show asphyxia. There are three types of asphyxia namely:

1. Mild asphyxia – Apgar score is 6-7. It requires clearing of the airway and application of external stimuli to in initiate breathing

2. Moderate asphyxia – Apgar score is 4-5. It requires resuscitation, administration of oxygen and drugs to initiate breathing.

3. Severe asphyxia – Apgar score is 0-3. It requires intensive resuscitative measures and intubation to survive.

## 5.2PREDISPOSING FACTORS

* Any condition causing foetal distress e.g. cord prolapse, prolonged labour,APH, intrauterine hypoxia due to placental insufficiency, post maturity, placenta abruption.
* Anaemia, Pre-eclampsia
* Pre- maturity due to under development of the respiratory centre.
* Blockage of the airway by mucus or liquor amnii at birth.
* Birth injuries e.g. intracranial injury
* Severe maternal disease inpregnancy e.g. sickle cell anaemia, cardiac disease
* Depression of respiratory center due to drugs e.g. GA and narcotics

## 5.3SIGNS AND SYMPTOMS

**MILD AND MODERATE ASPHYXIA**

1. Apex beat (pulse rate) 100/min or less

2. Skin colour is pink with blue extremities

3. Response to stimuli may be present

4. Cry may be weak or strong

5. Makes effort to breath and may gasp with irregular respiration

**SEVERE ASPHYXIA**

1. No attempt to breath and may gasp periodically
2. it does not cry
3. Entire body skin is blue i.e. cyanosed-central.
4. No response to stimuli
5. Pulse rate very low or absent
6. Poor muscle tone

## 5.4NURSING MANAGEMENT

* Clear the airway as soon as possible.
* Nurse the baby in an incubator for at least 48 hrs to keep it warm at body temperature.
* Resuscitation may be needed to promote ventilation and ensure effective circulation to prevent acidosis, hypoglycaemia and intracranial hemorrhage
* Do suctioningwhenever necessary
* Closely observe the baby for skincolour, TPR.
* Administer oxygen by mask, ambu bag or nasal catheter whenever there is an apnoeic attack
* Give IV fluids for rehydration.
* Aspirate mucus to unblock the airway or may intubate the baby.
* Give fluids with electrolytes to maintain fluid – electrolyte balance.
* If the mother was given narcotics during labour, administer its antidote *naloxone*thro the umbilical vein.
* Administer the following drugs:
* *Sodium bi- carbonate 1-2 mls* to combat acidosis.
* *Vitamin K 0.5 -1 mg i.m*to prevent haemorrhagic disorders.
* *Aminophylline to improve* respiration.
* *Calcium gluconate to* strengthen heart muscles.
* Maintain accurate input output chart to prevent over hydration and under hydration
* When the baby is stable pass NG tube and start feeding.
* Observe aseptic technique to prevent cross infection.
* Administer broad spectrum antibiotic prophylactically.

## 5.4 PREVENTION OF ASPHYXIA

* Proper screening of mothers to detect those mothers at risk and advice on hospital delivery for proper management.
* Pelvic assessment should be done at 36 weeks gestation to rule out pelvic inadequacy e.g. CPD.
* Proper management of maternal diseases in pregnancy.
* Drugs that depress respiratory center e.g. sedatives, GA and narcotics should be avoided in late first stage.
* Early detection and management of foetal distress.
* Clearing baby’s airway as soon as the head is born.
* Avoiding instrumental deliveries but rather prepare for caeserian section.

## 5.5 COMPLICATIONS

1. Brain damage
2. Cardiac arrest
3. Respiratory distress syndrome
4. Respiratory acidosis.

# RESPIRATORY DISTRESS SYNDROME

This is a condition that occurs due to lack of or inadequate surfactant in the lung tissue. Mature lungs have adequate surfactant factor that lower the surface tension in the alveoli, stabilizes the alveoli and prevents them from adhering together and collapse. This leads to breathing with ease. Surfactant is produced slowly from 20 weeks gestation and reaches a surge at 30- 34 weeks gestation and another surge at onset of labour.

The premature infant lack this function thus the alveola walls pressure rises as he breaths out and alveoli collapse leading to severe difficulty in breathing.

Other namesare:

* Hyaline membrane disease
* Pulmonary syndrome of the newborn
* Developmental respiratory distress

It is a disease of prematurity and self limiting with recovery phase or death.

## 6.1PREDISPOSING FACTORS

* RDS may be a complication of asphyxia and develops within 48 hrs of birth
* Prematurity due to inadequate surfactant factor
* Perinatal hypoxia e.g due to APH which reduces surfactant synthesis
* Perinatal hypoxia
* Profound hypothermia –leads to injury of cells that produces surfactant
* Congenital heart disease

## 6.2CLINICAL FEATURES

* difficulty in breathing- dyspnoea
* flaring of the alaenasi
* tachypnoea with respiration of above 60/min
* hypothermia
* generalized cyanosis
* costal and sterna retraction
* grunting expiration ( prevent atelectasis)
* reduced or increased heart rate
* chest X-ray shows collapsed alveoli
* the baby has poor muscle tone and is motionless
* poor digestion due to diminished bowel movement
* resolves or death occurs within 3-5 days

## 6.3 NURSING MANAGEMENT

Management is symptomatic until the disease resolves.

If RDS is anticipated, inform the paediatrician to resuscitate the baby.

Nurse the baby in an incubator to prevent hypothermia by controlling the body temperature.

 Administer oxygen or do artificial ventilation to prevent hypoxia.

Closely monitor the blood PH to prevent acidosis and support pulmonary circulation because high carbon dioxide level leads to constriction of pulmonary arterioles leading to poor pulmonary blood flow.

In case there is acidosis, sodium bicarbonate is added to 10 % dextrose drip.

 Keep the baby nil per oral till the distress resolves.

Administer IV fluids eg.10% dextrose and add calcium gluconate to strengthen heart muscles; sodium bicarbonate to ensure fluid electrolyte balance.

Check heamocrit (PCV) and if less than 40% transfuse with blood.

Maintain the normal BP with volume expanders eg. n/saline.

Position the baby to provide greatest air entry(prone position with extended head)

Suction and do postural drainage to remove secretion and keep the airway patent.

 Close observation to monitor the progress whether improving or deteriorating i.e. the heart rate, respiration, chest in- drawing, grunting respiration, and cyanosis.

When the condition resolves, introduce oral feeds. Incase the baby develops abdominal distention due to ingestion, stop the oral feeds and start IV fluids.

NB: principles followed during care of babies with respiratory problems are observation, oxygenation, positioning, nutrition and hydration.

## 6.4 PREVENTION

* Early detection and management of high risk pregnancies to prevent premature delivery
* Conditions such as diabetic mellitus should be properly managed so that delivery can be prolonged to 36 -38 weeks. The mother is given *Dexamethasone 4mg tds 48 hrs* before c/s to stimulate lung maturity.
* Prevent prenatal hypoxia by ensuring there is no intracranial injury at birth.
* Effective resuscitation at birth of high-risk babies.
* Assessment of gestational age and lungs maturity through amniocentesis so that elective c/s or delivery can be delayed if lungs are not mature enough

## 6.5 COMPLICATIONS

* Retrolentalfibriplasia
* Hypothermia
* Hypoglycaemia
* Patent ductusarteriuosus
* Abdominal distension
* Hypocalcaemia
* Intracranial
* Infection

# 7.0 HYPOGLYCAEMIA

This is a metabolic disorder in which the blood glucose level falls below 2.6 mmol/L. At term, the baby’s glucose level is almost equal to that of the mother but gradually drops within 3-4 hrs after birth. This is why the baby has to be fed within four hours of life. The baby’s maintain their energy requirements as long as they are kept warm.

This condition is common in infants of diabetic mothers. Due to excess glucose, the large babies (macrosomia). At birth the glucose level falls rapidly while insulin levels remain relatively high so the baby is at risk of hypoglycemia .this is why such babies are admitted to the NBU.

Prolonged hypoglycaemia can lead to mental retardation, permanent neurological damage anddeath due to respiratory and metabolic acidosis.

## 7.1 PREDISPOSING FACTORS

* Low birth weight
* Prematurity
* Birth injuries
* Maternal diabetes mellitus
* Asphyxia
* Septicaemia
* Respiratory distress syndrome

## 7.2 CLINICAL FEATURES

* Low blood glucose less than 2.6 mmol/L
* Poor feeding
* High pitched cry
* Lethargy
* Irritability
* Hypotonic muscle activity
* Hypothermia
* Apnoea

## 7.3 NURSING MAMAGEMENT

* Give 10% dextrose infusion until normal glucose levels are achieved.
* Encourage the mother to breastfeed the baby
* Feed through NGtube or cup and spoon expressed breast milk.
* If the hypoglycemia is severe, put up 10% dextrose infusion and give 65-85 mls/kg of body weight in 24hrs.
* Give bolus dose of 25% dextrose 2wmls/kg body weightiv slowly for 30 min.
* Closely monitor the glucose levels 1 hourly until the general condition is stable or normal levels have been achieved.
* Once the normal levels have been achieved, wean off the dextrose and observe closely for changes in the condition.

## 7.4 PREVENTION

* Taking blood glucose levels at birth and introducing glucose feeds e.g. dextrose or breastfeeding within 1hr of life.
* Prevent hypothermia.
* Monitoring glucose level 2hrly for the first 6-8 hours.
* Infants of diabetic mothers should be admitted into NBU and blood glucose level regularly checked.

## 7.5 COMPLICATION

* Hypothermia
* Convulsions
* Brain damage
* Neonatal death as an outcome.

# 8.0 NEONATAL HYPOTHERMIA

 This is a condition in which the neonates body temperature falls below 36\*C .the baby losses heat through radiation,conduction, convection and evaporation.

## 8.1 PREDISPOSING FACTORS

* Prematurity
* Asphyxia neonatorum
* Maternal diabetes mellitus
* Respiratory distress syndrome
* Cold environment

## 8.2 CLINICAL FEATURES

* Rectal temperatures is below 36\*C
* Baby feels cold on touch
* Paleness of extremeties and face
* Very weak cry
* Low respiration rate
* Baby not eager to feed (poor feeding)

## 8.3NURSING MANAGEMENT

* Nurse the baby in a warm environment in a resuscitaire or wrap it in warm clothings
* Feed the baby with expressed breast milk via NG tube
* Give the baby extra glucose e.g. dextrose
* Closely observe the baby for signs of hypoglycaemia and if present, give 10% dextrose
* Check for and treat convulsions with anticonvulsants

## 8.4 PREVENTION

* Delivery should be conducted in a room temperature
* Put the baby on resuscitaire or in incubator to compensate heat loss to the environment.
* Baby should not be bathed within 1hr of life but top-tailing can be done after one hour.
* Encourage skin to skin contact (kangaroo method) when carrying the baby.

## 8.5COMPLICATIONS

* Convulsions
* Hypoglycaemia
* Brain damage

# 9.0 OPTHALMIA NEONATORUM

This is a condition that occurs in neonates within 21 days of life and is characterized by purulent discharge from the eyes. It is common in infants of mothers who had vaginal discharge e.g. gonnorrhoea during pregnancy. Syphilis does not predispose an infant to opthalmianeonatorum but it causes congenital syphilis that is characterized by gross congenital malformation.

## 9.1CAUSATIVE ORGANISMS

* *neisseriagonorrhoeae*
* *chalmydiatrachomatis*
* *staphylococcus aureus*
* *Escherichia coli*
* *Haemophilus influenza*
* *Streptococcus pneumonia*
* *Pseudomonas .spp*
* *Klebsiella*

## 9.2 CLINICAL FEATURES

* Eyes have sticky watery discharge
* Eyes are slightly red
* Oedematus eyelids
* Yellow purulent discharge if the infection is by *N.gonorrhoeae*
* Inflamed conjunctiva

## 9.3 NURSING MANAGEMENT

* All perinatal mothers presenting with vaginal discharge suggestive of gonnorrhoae should be treated before delivery.
* Correctly swab the baby’s eye at birth.
* Instill 1% tetracycline ointment (TEO) to all babies prophylactically.
* All infected babies should be isolated
* Take eye swab for culture and sensitivity
* Administer drugs such as;
* Gentamycin eye drops
* TEO but not systemic tetracycline
* Penicillin eye drops
* Kanamycin eye drops
* Swab the eyes with warm saline 3 times a day from inside outwards
* Administer some broad –spectrum systemic antibiotic but not tetracycline because it deposits in bone leading to depressed bone growth.

## 9.4 COMPLICATIONS

Partial or permanent blindness

# 10.0 NEONATAL JAUNDICE

This is condition in neonates characterized by yellow discoloration of the skin, sclera and mucous membrane. It develops when there is an excessive bilirubin level in the blood stream. When there is increased rate of haemolysis of RBC or decreased conjugation, there are high amounts of free bilirubin in circulation leading to jaundice.

## 10.1BILIRUBIN METABOLISM

When RBC’s are broken down by haemolysis, they produce heme and globulin. The heme part produces bilirubin and iron. Unconjugated (indirect) bilirubin is fat soluble hence has to be converted to water soluble form (conjugated/ direct bilirubin) by process of conjugation for it to be excreted. Conjugation of bilirubin occurs in the liver and thus it has to be transported to the liver by binding to transport protein, albumin. On arrival to the liver, bilirubin detaches itself from the albumin.

Conjugation is done by glucoronlytransfares in which bilirubin is added to glucoronic acid to become bilirubinDiglucoronide that is water soluble. Excretion of the bilirubin is done through the biliary system into the intestine. While in the intestine, it is converted to stercobilinogen by the gut normal flora and excreted in stool. Some of it is absorbed from the gut and becomes urobilinogen which is excreted in urine.

If conjugation process is interfered with, there will be accumulation of unconjugatedbilirubin leading to hyper bilirubinaemia and jaundice. This bilirubin may cross the BBB and cause brain damage, a condition known as *kernicterus*that is characterized by seizure, hyper-tonicity, lethargy, and stiff neck with hyper extended head.

## 10.2TYPES OF JAUNDICE

### 10.2.1 PHYSIOLOGICAL JAUNDICE

This type of jaundice affects both preterm and term babies in the first few days of life. It is apparent with the signs on the third day when the unconjugatedbilirubinlevels in serum is 25-125 mmol/L. In term babies , it never appears before 24 hrs of life but it can be in pre terms and the serum levels never exceeds 200mmol/L. it is also self limiting in term babies.

## Causes

* Excessive haemolysis of RBCs greater than conjugation rate.
* Glucoronyltransferase enzyme deficiency
* Increased enterohepaticreabsorption
* Decreased albumin binding capacity thus less bilirubin is transported to the liver for conjugation.

## Nursing management

* Admit the baby into NBU and assess the general condition.
* Start early and frequent breastfeeding for it provides glucose to the liver cells and also encourages bowel colonization with normal flora which is important in formation of stercobilinogen for excretion in stool. It also leads to increased gut motility leading to faster excretion of bilirubin. Feeding also enhances enzyme production and conjugation.
* Closely monitor serum bilirubin levels at 12 -24 hrs interval.
* If bilirubin levels takes time to clear, put the baby on phototherapy.

## 10.2.2 PATHOLOGICAL JAUNDICE

This type of jaundice appears within 24 hrs of life and is not self- limiting thus may persist for long. There is rapid rise in serum bilirubin. It includes both obstructive and haemolytic jaundice.

## Causes

They include pathological disorders that increasebilirubin production, reduces transportation to and fro the liver or reduced rate of conjugation.

1. Increased haemolysis –Rhesus and ABO incompatibility, G6PD enzyme deficiency, bacterial septicaemia.
2. Non- haemolytic causes of increased unconjugatedbilirubin –CNS hemorrhage, cephalohaematoma, polycythaemia, exaggerated enterohepatic circulation of bilirubin due to fuctionalileus.
3. Decreased rate of conjugation –Criggler Nagar syndrome, Gilbert’s syndrome
4. Hepatotoxic drugs
5. Billiary obstruction that prevents transport of conjugated bilirubin to GIT for excretion
6. Reduced bilirubin binding sites to the albumin.
7. Malnutrition
8. Increased reconversion of conjugated to unconjugatedbilirubin if it stays in the GIT for long.

## Nursing management

* Assess the baby to determine the degree of jaundice.
* Do investigation on serum bilirubin levels and Hb.
* Start the baby on phototherapy.
* Order for blood exchange transfusion if necessary.

## 10.3Complication of neonatal jaundice

* Retinal damage due to lights used in treatment
* Anemia
* Hyperthermia associated with phototherapy.
* Hypocalcaemia
* Kernicterus

NB:read more on obstructive and haemolytic jaundice

## 10.4 TREATMENT MODALITIES OF NEONATAL JAUNDICE

 There are three main modalities namely;

* Phototherapy
* Blood exchange transfusion
* Protoporphyrins

## 10.4.1 phototherapy

Phototherapy prevents bilirubin levels from going high enough to cross BBB and cause kernicterus

### Mechanism of action

Blue florescent light at a given wave length is absorbed by the unconjugatedbilirubin in the skin and superficial capillary and is converted into conjugated bilirubin which is water soluble and can be excreted in stool and urine.

### Indications

* Pre term with jaundice appearing after 48 hrs and bilirubin levels are 260 -265 mmol/L
* Pre term with weight less than 1500g and bilirubin levels are 85 -114 mmol/L
* Pre term with weight more than 1500g and bilirubin levels are 114-165 mmol/L

### Care of the baby on phototherapy

* Expose the whole body of the baby to increase surface area exposed to light
* Keep turning the baby 2hrly to expose all parts to the fluorescent light.
* Ensure the airway of the baby is patent by extending the head.
* Cover the eyes of the babyto prevent damage by direct ray of lights.
* When breastfeeding the eyes are unpadded to encourage eye contact with the mother.
* Provide intermittent phototherapy i.e. 6 hrs on and 6 hrs off but may be continous.
* Give phototherapy for 2-3days and assess the serum bilirubin levels twice or three times a day NB. Greatest reduction in bilirubin levels will be in the first 24 hrs of phototherapy.
* Observe the eyes for weeping or discharge.
* If phototherapy is continous, give extra fluids to prevent dehydration and maintain accurate input output charts.
* Change linen frequently because opening of bowels is increased(loose stool)
* Observe the feeding and sleeping behavior of the baby.
* Observation e.g. temperature to rule out hyperthermia and skin colour to monitor the progress.
* Top tail the baby to maintain hygiene.

## Side effects

* Loose stool due to rapid instinal transit
* Dehydration
* Hyperthermia
* Visual deprivation
* Poor feeding
* Fragility
* Lethargy
* Irritability
* Hypocalcaemia

## 10.4.2 BLOOD EXCHANGE TRANSFUSION

This is a treatment in which the baby’s blood is gradually removed and replaced by donor’s blood. It is used as a definitive treatment when bilirubin concentrations are approaching toxic levels. The baby has haemolyticdisease or low Hb. The transfusion has the following benefits

-it helps in increasing the baby’s Hb

-excessive bilirubin and unwanted antibodies are washed from the babys circulation.

The donor’s blood used for the transfusion should be rhesus negative so that it does not alter the babys blood group and to ensure that no antigen ios introduced into the baby’s circulation that may lead to antibodies production. It should also be fresh and ABO compactible.

## Indications

* Infants with haemolytic disease.
* Preterms with bilirubin levels of 300 -400 mol/l
* Babies whose birth weight was less than 1500g and have bilirubin levels of 255mol/l
* Term babies with bilirubin levels above 100 mol/l at birth or later 400 -500 mol/l

## Care of the baby post transfusion

* Put the baby back to phototherapy to continue with it.
* Closely observe the baby for bleeding from the umbilical cord.
* If the baby was on infusion, continue for some time.
* Reassure the mother and involve her in the care of the baby.

## Complications

* Circulatory collapse
* Incompatibility reactions
* Acquired infections e.g. HIV, hepatitis B.

## 10.4.3PROTOPORPHYRINS

These are hemeoxygenase inhibitors which are administered to inhibit the breakdown of heme thus reduce bilirubin production.

They are usually used in combination with phototherapy and/or blood exchange transfusion.

## 10.4.4 Nursing diagnosis of children undergoing phototherapy

* Deficient fluid volume
* Imbalanced nutrition less than body requirements
* Impaired skin integrity
* Risk for injury
* Ineffective thermoregulation

# 11.0 HAEMORRHAGIC DISEASE OF THE NEWBORN

This bleeding occurs during the first fews days of life due tovitasmin K deficiency. Vitamin K is synthesized by the bowel normal flora and its role is to convert clotting factors such as prothombin, thrombokinase, thromboplastin. To prevent HDN the neonates are given *vitamin K 0.5-1 mgi.m.*

## 11.1 predisposing factors

* Hereditary factors- clotting factor defect e.g. haemophilia
* Prematurity
* Birth trauma
* Treatment with antibiotics
* Respiratory distress syndrome
* Disseminated intravascular coagulopathy (DIC)
* Birth asphyxia
* Mothers who are on drug such as warfarin, heparin and Phenobarbital

## 11.2 Clinical features

* Continuous oozing of blood from the umbilical cord
* There is a spontaneous bleeding from various parts of the body
* Bleeding in the mucous membrane of GIT and may present with maleana stool or haematemesis
* Continuous bleeding from any punctured blood vessel or injection site thus when looking for venous access avoid puncturing femoral or jugular veins which are the largest veins in the body
* Haematuria or omphalorrhagia

## 11.3 Nursing management

* Upon admission into NBU, administer vitamin K 0.5-1 mg i.m
* Preserve all linen soiled by blood for estimination of blood loss
* Administer vitamin K 1-2 mg to arrest bleeding immediately
* Observe vital signs TPR ¼ hrly
* If bleeding is severe, transfuse fresh blood or frozen plasma at 20mls/kg of body weight
* Obnserve for signs of shock and if present transfuse with packed cells and fresh whole blood at 75 -100mls/kg of body weight if the baby is term
* General management is like any other baby in the unit

## 11.4 Complications

* Anaemia
* Hypovolaemic shock
* Brain damage

# 12.0 BIRTH INJURIES

Birth injuries refer to trauma that a foetus sustains during birth. The structures commonly involved are muscles, nerves, bones, visceral organs and skin.

## 12.1types of birth injuries

1. Internal organ injuries – spleen, liver, adrenal glands
2. Nerve injury –mostly brachial plexus leading to Erb’s palsy
3. Soft tissue injury –intracranial haemorrage, skull fractures
4. Extracranial injuries –cephalohaematoma,caputsuccadenium.

## 12.2 predisposing factors

* Prematurity
* Large for dates
* Cephalo pelvic disproportion
* Malpresentation
* Congenital malformation e.g. hydrocephalus

**CAPUT SUCCADENIUM AND CEPHALOHAEMATOMA**

*Caput succadenium –* is an oedematous swelling due to accumulation of serum fluid under the foetal scalp. It results from pressure between the foetal skull and pelvic bones during delivery that leads to reduced venous blood and lymphatic drainage and part of the serum escapes into the tissues. The swelling is self – limiting and disappears within 36hours of life.

*Cephalohaematoma –*is accumulation of blood between the periosternum and the skull bone. It is caused by friction between the foetal skull bones and the pelvic bones e.g. in CPD

***Caput succadeniumCephalohaematoma***

Present at birth Appears after 12 hrs of life

Disappears within 36 hrs May persist for weeks

Diffuse and pits with pressure Circumscribed; doesn’t pit on pressure

May cross a suture line Never crosses a suture line

Double suture line is unilateral Double cephalohaematoma is bilateral

Tends to grow less with time Tends to grow larger with time

INTRACRANIAL INJURIES AND HAEMORRHAGE

This refer to the damage of structures within the cerebral hemispheres of the brain. Various structures may be injured leading to different types of haemorrhage:

* Cerebral tissue – injury to cerebrum leading to cerebral haemorrhage
* Cerebral hemisphere and basal ganglia –supra tentorialhaemorrhage
* Veins of gallen and tentorium – subarachnoid haemorrhage
* Falxcerebri (fold of dura mater and tentoriumcerebelli) –subdural haemorrhage

PREDISPOSING FACTORS

* Prematurity
* Excessive moulding
* Instrumental delivery
* Hypoxia that leads to engorgement of blood vessels
* Precipitate labour
* Prolonged labour
* Large babies

CLINICAL FEATURES

* Dyspnoea
* Asphyxia
* Rolling of the eyes
* Pallor of the skin and mucous membranes
* Bulging of the anterior fontanelle due to increased intracranial pressure
* Shock due to circulatory collapse
* Twitching of the facial muscles if facial nerve is affected
* Cyanosis
* Grunting respirations
* High pitched cry
* Rigidity of limbs

## 12.3 GENERAL MANAGEMENT OF BIRTH INJURIES

* Intraparetally, predisposing factors should be diagnosed and managed early e.g. preterm labour, malpresentation, prolonged labour.
* Observe the baby closely for skin colour, twitching, rolling of the eyes, convulsions
* Keep the baby warm
* Administer Vitamin K 0.5 -1 mg i.m for they are predisposed to haemorrhage
* Maintain 2 hrly turning of the baby
* Provide intermittent oxygen therapy PRN
* Give IV fluids e.g. 10% dextrose for the first 24 hrs then introduce oral feeds if the condituion improves
* Give symptomatic management
* Have resuscitative equipment ready in case of an emergency
* Administer anticonvulsants e.g. Phenobarbital prophylactically

## 12.4 COMPLICATIONS

* Musculoskeletal deformities
* Brain damage
* Respiratory distress
* Hyperbilirubineamia
* Hypoglycaemia

# 13.0 HYDROCEPHALUS

This is a condition where ther is accumulation of CSF within the ventricles of the brain with the resultant increased ICP and enlargement of cerebral ventricles. It can be detected prenatally by ultrasound and in labour they may present by breech presentation, fontanel and sutures are very wide on VE.