**PAEDIATRIC NURSING**

**Main objective**

The student will acquire knowledge, skills, and attitudes to promote health, prevent illness, diagnose, manage and rehabilitate infants and children suffering from common childhood conditions/diseases

**COURSE OUTLINE**

Identify children with normal growth and development.

* Recognize and manage deviation from normal growth patterns
* Triage and provide emergency care to infant and children
* Diagnose, manage and rehabilitate children with various paediatric conditions
* Apply I.M.C.I. knowledge in managing children under five years

**Teaching methods**

* Lectures, demonstration, group discussions, audiovisual aids, peer teaching and handouts

**Assessment method**

* Cat
* Final examination

**Table of contents**

**Growth and development 3**

**Milestones 3**

**Erikson’s theory of psychosocial development 5**

**Psychosexual Stages of Development 7**

## Factors influencing growth & development: 7

**Deviations from normal growth and development and their management 8**

**Disorders of physical growth 9**

**Paediatric conditions 11**

**Impact of sickness and admission to child, parents and family 11**

**Immediate and long term 11**

**Respiratory conditions 12**

**Streptococcal sore throat (Pharyngitis 12**

**Tonsillitis 13**

**Otitis media 14**

**Acute laryngotracheobronchitis 15**

**Pneumonia 15**

**Asthma 17**

**Cardiovascular conditions 19**

**Rheumatic fever 19**

**Rheumatic heart disease 20**

**Anaemia 20**

**Diarrhea 21**

**Urinary tract conditions 24**

**Urinary tract infection 24**

**Acute glomerulonephritis 26**

**Nephrotic syndrome 27**

**Meningococcal meningitis 28**

**Malnutrition 30**

**Malignancies 34**

**Wilm’s tumour 34**

**Burkitt’s lymphoma 35**

**Tetanus 36**

**Congenital Anomalies 38**

**Cleft palate and cleft lip 38**

**Esophageal atresia and Tracheoesophageal fistula. 40**

**Hypertrophic pyloric stenosis 42**

**Ompalocele 44**

**Gastroschisis 45**

**Hirschsprung’s disease 46**

**Anal rectal malformations 48**

**Hydrocephalus 49**

**Spinal Bifida 51**

**Ambiguous genitalia 52**

**Convulsive disorders 55**

**Poisoning in children 56**

**Acetaminophen poisoning (paracetamol) 57**

**Carbon Monoxide Poisoning 58**

**Aspirin and Other Salicylate Poisoning 58**

**Organophosphate Poisoning and Carbamate Poisoning 58**

**Hydrocarbons 59**

**Paediatric HIV 59**

**Growth and development**

All body systems undergo progressive maturation; concurrent development of skills allows infants to increasingly respond to the environment. Acquisition of these fine and gross motor skills occurs in an orderly head to toe and center to periphery sequence.

**Proportional changes**

Growth is rapid during the first year, especially the initial 6 months. Height increases by 2.5 per month during the first 6 months. Increase in length occur, sudden spurts rather than in a slow, gradual pattern. Average height is 65cm at 6 months and 74cm at 12 months. By 1 year the length has increased by almost 50%. This occurs mainly in the trunk rather than the legs and contributes to the characteristic physique of the older infants.

Head growth is rapid and an important determinant of brain growth. Head circumference increases approximately 2 cm per month from birth to 3 months, 1 cm per month from 4 to 6 months. The average head size is 43 cm at 6 months and 46 cm at 12 months. By1 year, the head size has increased by almost 33%. Closure of cranial sutures occurs, with the posterior fontanelle fusing at 6 to 8 weeks and the anterior fontanelle closing by 12 to 18 months of age.

It is important to note that genetic, metabolic, environments and nutritional factors strongly influence infant growth. Use appropriate growth charts reflecting weight for length and head circumference in each case to determine appropriate growth parameters. Expanding head size reflects the growth and differentiation of the nervous system.

The chest assumes a more adult contour, with the lateral diameter becoming larger than the anterioposterior diameter. The heart grows less rapidly than the rest of the body does; its weight is usually doubled by 1 year of age, where as the body weight triples over the same period.

**Milestones**

A child’s development represents the interaction of heredity and the environment on the developing brain. Heredity determines the potential of the child, while the environment has to meet the child’s physical and psychological needs. These vary with age and stage of development;

* An infant is totally physically dependent on his parents and requires a limited number of cares to meet his psychological needs.
* A primary school-age child can usually meet some of his physical needs and cope many social relationships.
* Teenagers are able to meet most of their physical of their physical needs while experiencing increasing increasingly complex emotional needs.

**Four areas of development**

1. Gross motor
2. Fine motor and vision
3. Speech, language and hearing
4. Social, emotional, behavior
5. **Gross motor**

In the first 18 months of life there is rapid motor development from an immobile newborn infant to one who is able to adopt an upright posture.

* Acquisition of tone and head control

By 6 weeks an infant can lift his head and move it from side to side. By 3-4 months of age the infant can hold his head upright when held sitting.

* Primitive reflexes e.g. Moro, grasping etc. need to disappear by to allow motor development to progress, and almost all do so by 4-6 months of age.
* By 6 months an infant will sit without support
* Locomotor pattern

He becomes mobile, with crawling at 8-9 months of age, walks around furniture at10months, and take his first steps unsupported at 12 months, some can walk unsteadily, with broad gait and hands apart. At 20 months can kick a ball, hop on one leg, go up and down stairs, one leg at a time, jump and pedal a bicycle.

**2. Fine motor and vision**

* Fixing, following and visual alertness

By six weeks the infant will be visually alert and when supine will move his head from side to side to follow an object.

* Hand regard

At 3-4 months, infants show hand regard, spending a lot of time looking at their hands.

* Grasp reflex, voluntary grasping, pincer grasp, pointing:

A new born will grasp an object and at six months the infant is able to grasp actively with the whole hand (palmar grasp). Has a good pincer grip (thumb and first or second finger) of a small object at 10 months of age. Soon afterwards the infant points at objects with the index finger.

* Handling objects with both hands and transferring: At 6 month’s infants should be able to handle objects with both hands and transfer objects from one hand to another. There should be no hand dominance during the first year.
* Manipulation of small objects, writing, building bricks, dressing. At 14 months children are able to grip when they hold the pencil and their ability with building blocks, and they are able to scribble. Subsequent skills are dressing, writing, and key board skills.

**3. Speech and language and learning**

* Sound recognition and vocalization: The newborn infant stills to voice and startles to loud noises. He responds to his mother’s voice at 6 weeks of age even when she cannot be seen. Vocalization emerges and by 4 months the infant is making vowel sounds.
* Cools and babes: At 6 months babies start to use monosyllables e.g ’ba’ ‘da’. At 7-8 months they turn to voice and acquire two syllables like ‘dada’ ‘mama’ none specifically.
* Single words and understanding simple requests: Appropriate use of single words such as ‘dada’ or ‘mama’ and one or two words occurs at 13 months and also understanding simple commands such as ‘no’ or ‘give’ me and respond to their names. By 18 months children can say can say about ten words and can demonstrate six parts of the body
* Joining words, phrases: At 18 months he can combine two words and at 24 months he can combine three words.
* Simple and complex conversation: Between 2-3 years the conversation becomes increasingly complex with phrases followed by sentences. At 3 years knows his age and a few colours.

1. **Social and emotional behavior**

* Smiling, social responsiveness: Smiling at 6 weeks is an important and remarkable constant milestone. It means that the baby smiles in response to someone smiling directly at him. At 10 months they will wave ‘bye bye’ if they wish to.
* Separation anxiety: At about 18 months of age, many infants become anxious and unhappy when separated from their mother or care taker and become aware of strangers.
* Self help skills, feeding, and dressing: infants will put solid food into their mouth at about 6 months of age, drink from a cup by 12 months and hold a spoon and feed themselves safely at 18 months. At 24 months infants are able to remove garments.
* Symbolic play: At 24 month’s children symbolic play where they play with toys such as doll, brush, chair, spoon and they play as if they were life size equivalents.
* Social behavior: Once mobile children explore the surrounding and require constant attention and supervision. Problems may arise with food refusal, reluctance to go to sleep, waking up at night, temper tantrums and antisocial behavior. During the second year of life children play on their own or alongside others (parallel play) and only subsequently learn interactive play at three years plus.

**Erikson’s theory of psychosocial development**

* Erikson’s theory of psychosocial development has eight distinct stages and he assumes that a crisis occurs at each stage of development. Successful completion of each stage results in a healthy personality and the acquisition of basic virtue whereas failure to successfully complete a stage can result in a reduced ability to complete further stages and therefore a more unhealthy personality and sense of self.

**The different ages and stages relevant to paediatric nursing (Erik Erickson)**

|  |  |  |
| --- | --- | --- |
| **Stage** | **Psychosocial crisis** | **Age** |
| 1 | Trust vs. mistrust | Infancy(0 to 1 1/2) |
| 2 | Autonomy vs. shame | Early childhood (1 ½ to 3) |
| 3 | Initiative vs. guilt | Play age (3 to 5) |
| 4 | Industry vs. Inferiority | School age (5 to 12) |
| 5 | Ego identity vs. Role confusion | Adolescence (12 to 18) |
|  |  |  |

1. **Trust vs. Mistrust**

During this stage the infant is uncertain about the world in which they live and infant looks towards their primary caregiver for stability and consistency of care. Infants will develop a sense of trust if they receive consistent, predictable and reliable care. Success in this stage will lead to the virtue of **hope** Failing to acquire the virtue of hope will lead to the development of fear.

**2. Autonomy vs. Shame and Doubt**

At this stage the child is mobile and developing physically children begin to assert their independence by walking away from their mother, picking which toy to play with discovering that he or she has many skills and abilities, Such skills illustrate the child's growing sense of independence and autonomy. Children should be allowed to explore the limits of their abilities within an encouraging environment which is tolerant of failure. Parents must try not to do everything for the child but if the child fails at a particular task they must not criticize the child for failures and accidents (particularly when toilet training). The aim has to be “self-control without a loss of self-esteem”. Success in this stage will lead to the virtue of **will**.

**3. Initiative vs. Guilt**

During this period the primary feature involves the child regularly interacting with other children at school. Central to this stage is play, as it provides children with the opportunity to explore their interpersonal skills through initiating activities.

Children begin to plan activities, make up games, and initiate activities with others. If given this opportunity, children develop a sense of initiative, and feel secure in their ability to lead others and make decisions.

Conversely, if this tendency is marred, either through criticism or control, children develop a sense of guilt. They may feel like a nuisance to others and will therefore remain followers, lacking in self-initiative.

The child takes initiatives which the parents will often try to stop in order to protect the child. The child will often overstep the mark in his forcefulness and the danger is that the parents will tend to punish the child and restrict his initiatives too much.

It is at this stage that the child will begin to ask many questions as his thirst for knowledge grows. If the parents treat the child’s questions as trivial, a nuisance or embarrassing or other aspects of their behavior as threatening then the child may have feelings of guilt for “being a nuisance”.

Too much guilt can make the child slow to interact with others and may inhibit their creativity. Some guilt is, of course, necessary, otherwise the child would not know how to exercise self control or have a conscience.

A healthy balance between initiative and guilt is important. Success in this stage will lead to the virtue of **purpose.**

**4. Industry (competence) vs. Inferiority**

Children are at this stage will be learning to read and write, to do sums, to do things on their own.

The child’s peer group will gain greater significance and will become a major source of the child’s self-esteem and now feels the need to win approval by demonstrating specific competencies that are valued by society, and begin to develop a sense of pride in their accomplishments.

If children are encouraged and reinforced for their initiative, they begin to feel industrious and feel confident in their ability to achieve goals. If this initiative is not encouraged, if it is restricted by parents or teacher, then the child begins to feel inferior, doubting his own abilities and therefore may not reach his or her potential. Yet again, a balance between competence and modesty is necessary. Success in this stage will lead to the virtue of competence.

**5. Identity vs. Role Confusion**

This is the transition period from childhood to adulthood. Children are becoming more independent, and begin to look at the future in terms of career, relationships, families, housing, etc. The individual wants to belong to a society and fit in. They learn the roles they will occupy as an adult. It is during this stage that the adolescent will re-examine his identity and try to find out exactly who he or she is.

Erikson claims that the adolescent may feel uncomfortable about their body for a while until they can adapt and “grow into” the changes. Success in this stage will lead to the virtue of fidelity.

Fidelity involves being able to commit one's self to others on the basis of accepting others, even when there may be ideological differences.

During this period, they explore possibilities and begin to form their own identity based upon the outcome of their explorations. Failure to establish a sense of identity within society ("I don’t know what I want to be when I grow up") can lead to role confusion. Role confusion involves the individual not being sure about themselves or their place in society.

**Psychosexual Stages of Development**

According to Freud, childhood experiences shape our personalities and behavior as adults.

 Oral *(0-1 years of age):* During this stage, the mouth is the pleasure center for development. Freud believed this is why infants are born with a sucking reflex and desire their mother's breast. If a child's oral needs are not met during infancy, he or she may develop negative habits such as nail biting or thumb sucking to meet this basic need.

 Anal *(1-3 years of age)*: During this stage, toddlers and preschool experiment the control they learn to exert over their bodily functions is manifested in toilet-training. Improper resolution of this stage, such as parents toilet training their children too early, can result in a child who is uptight and overly obsessed with order.

 Phallic *(3-6 years of age):* During this stage, preschoolers take pleasure in their genitals and, according to Freud, begin to struggle with sexual desires toward the opposite sex parent (boys to mothers and girls to fathers). For boys, this is called the *Oedipus complex,* involving a boy's desire for his mother and his urge to replace his father who is seen as a rival for the mother’s attention. At the same time, the boy is afraid his father will punish him for his feelings, so he experiences castration anxiety. The *Electra complex,* later proposed by Freud involves a girl's desire for her father's attention and wish to take her mother’s place.

 Latency *(6-12 years of age):* During this stage, sexual in stint subside, and children begin to further develop the superego, or conscience. Children begin to behave in morally acceptable ways and adopt the values of their parents and other important adults.

 Genital *(12+ years of age):* During this stage, sexual impulses reemerge. If other stages have been successfully met, adolescents engage in appropriate sexual behavior, which may lead to marriage and childbirth.

## Factors influencing growth & development:

**1. Genetic factors.**

* Heredity: Hereditary traits are passed to the offsprings.eg tall parents tend to have tall children. Parents with high intelligence quotient are more likely to have children with high intelligence quotient.
* Racial: Different racial groups have varying growth potentials
* Gender: Pubertal growth spurt occurs earlier in girls, mean height and weight of girls are usually less as compared to boys of the same age.
* Chromosomes: Some chromosomal abnormalities can affect growth and developmwnt.eg Turner or Downs’s syndrome.

**2. Nutritional**

Nutritional deficiency considerably retards physical growth. Malnourished mother produce babies with IUGR. Over nutrition may cause obesity. So to control the nutritional requirements of mother is necessary to have a health child. Lack of proper nutrition can interfere with the maturation of your child’s brain and body.

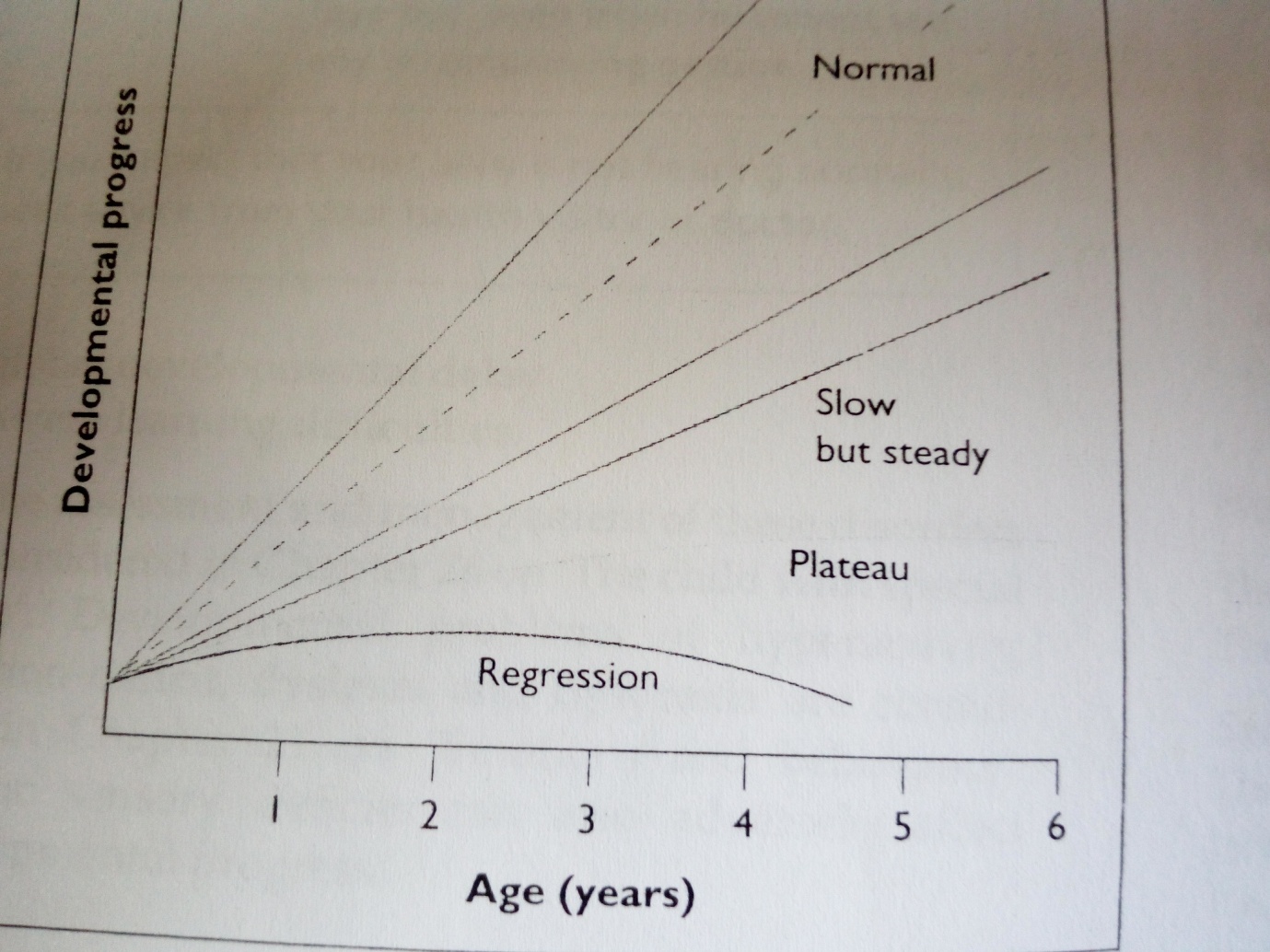
**3. Environmental factors.**

* **Maternal infections**: Some diseases passed from mother to fetus can lead to arrest or retard the fetal devrlopment.eg syphilis, viral hepatitis, and toxoplasmosis. Maternal rubella can cause congenital malformations in the infant if it occurs during the first trimester.
* **Exposure to teratogens**: abnormal fetal growth and development can be caused by intrauterine exposure to smoking, alcoholism, drug intake and radiation
* **Infections and infestations**: Systemic infections and parasitic infestations can decrease the rate of growth.
* **Systemic disease**: chronic conditions whether communicable or non communicable can have adverse effects on growth. Chronic untreated diseases of heart, lungs, liver etc. impair growth and development seriously. Growth Hormone Deficiency, Hypothyroidism, Cushing’s syndrome etc. medical condition that needs early attention
* **Hormones and drug:** Androgenic hormones and steroids accelerate skeletal growth, however ultimately epiphyses of bones close prematurely and the bone growth ceases relatively earlier.

1. **High social economic;**

* High social economic level families have a superior nutritional state; neglect has an adverse affect on development.
* Emotional factors: Children from broken homes and orphanages do not grow and develop at an optimal rate. Anxiety, insecurity, lack of emotional support and love, poverty, disturbed parent-child relationship, child abuse among others may adversely affect development.
* Cultural factors: Religious taboos and cultural beliefs plus myths of a community can affect the nutritional state and growth performance of children.

**Deviations from normal growth and development and their management**



**Variations in normal development**

* Average
* Slow starters, later coming to average.
* Advanced in general or in a specific field.
* Persistently retarded in all fields, becoming average or advanced
* Intermittent gaps in development

**Slow starters**

Such children are often late in acquiring certain skills such as walking, sphincter control. Sometimes this may be familial tendency. Some children may show retardation in all fields and go on to be normal

In case a child shows gross slowness in a particular field while normal in others, look for the cause. Some causes of isolated motor development variations include familial factors, environmental factors, and personality traits eg. Children of apprehensive mothers who are worried and frightened that something might happen to their children are late learners. A one year old child may refuse to walk due to ill fitting shoes and children with hypotonia (Down’s syndrome, malnutrition, rickets, and hypothyroidism) or hypertonic (cerebral palsy) walk late.

Visual impairment or neuromuscular disease results in delayed motor development. NB At times no cause can be found for a transient delay in a particular field and such children grow up to be normal.

Children with visual handicap, diminished parent child interaction, institutional care, and facial muscle atropy may smile late or never smile.

Usually a child is dry at 2 years and by night at 3 years; however some might continue to wet their bed up to 5-6 years. This may be due to delayed maturation of CNS or familial. Delay in speech is not always abnormal as it may be attributed to familial factors, poor environment or even mental abnormality and so deafness and autism should be ruled out.

**Children with temporary developmental gaps**

Temporary developmental gaps may be witnessed in normal children who have suffered illness eg. Encephalitis

**Progressive slowing and retardation in all fields**

Progressive slowing and retardation in all fields is almost always pathological, children who manifest with a continuous deterioration in their development have one or other degenerative disorders

**Advanced development**

Children who grow up to have higher than average IQ may show indication of this in childhood.

**Disorders of physical growth**

Physical growth is measured in terms of weight and height.

**Failure to thrive**

Def; Failure to thrive has no universal definition. Failure to thrive is a term assigned to a child whose weight for age is below 3rd percentile or a downward change in growth that has crossed two major growth percentiles.

**Causes**

Inadequate calorie intake e.g. incorrect formula, poverty

Inadequate absorption e.g. cystic fibrosis, celiac diseases

Increased metabolism e.g. hyperthyroidism, congenital heart disease, chronic immunodeficiency

Defective utilization e.g.in genetic anomaly such as Trisomy 21 or 18, congenital infection

**Clinical features**

Weight of the child is less than 2SD. Child interacts poorly with his peers and family members, motor activity is slow, speech is delayed and child remains preoccupied in his own.

**Management**

Goal is to provide sufficient calories to support ’catch up’ growth, a rate of growth greater can be expected rate for age. In addition to adding calorie density to feeds add multivitamin, dietary supplementation with high calorie foods and drinks. Any co existing medical condition should be treated.

Provide a positive feeding environment whether in hospital, at the clinic or home Teach parents’ successful feeding strategies, and supporting the child and family are essential components of care. Take daily weight and record all foods taken and record the child’s feeding behavior.

**Obesity**

A child is considered obese when there is excess accumulation of fat in the subcutaneous tissue and other parts of the body.

**Causes**

Dietary factors, consuming large infrequent meals

Habits, children speeding more time with in door activities e.g. computer games

Decreased energy expenditure which could be genetically predetermined

Genetic factors, obesity in children has a strong correlation with parental obesity

**Management**

Diet; Reduced calorie intake. These children should be encouraged to reduce sweets and junk food and embrace food rich in fibre and low calorie content.

Encourage physical activity outdoor games and exercise.

Behavior modification; the will of the child to reduce his weight should be strengthened by constant encouragement. Childs weight should be kept under surveillance, restrict television viewing and give psychological support.

Attempts should be made to reduce the weight gradually and a psychologist may be sought to uncover psychogenic causes.

**Short stature**

Short stature is defined as height less than 2 SD (standard deviation) below the mean or and there are two types, proportionate and disproportionate. Proportionate short stature is more common may occur, secondary to constitutional delay in growth, chronic infections, malabsorption, congenital heart disease, congenital heart disease, renal failure or endocrine malfunctioning, whereas disproportionate short stature occurs as a result of abnormal skeletal growth either the limbs or the trunk.

**Management**

Short stature children should be reviewed by the pediatrician

**Tall stature**

Child with a height of more than 2SD above the mean Short stature are said to be abnormally tall

**Causes**

Hereditary factors, cerebral gigantism, acromegaly and chromosomal abnormality e.g.Klinefelter syndrome. NB; most of the tall children are normal and their tallness is based on an optimal environment and genetic predisposition.

**Microcephaly**

A child is labeled as having microcephaly if his head circumference remains less than 3SD the mean for that age

**Causes**

Genetic or uterine disturbances, which include chromosomal defects, intra uterine infections and exposure to teratogens, once born, insult to the growing brain can be caused by meningitis, malnutrition, hypothermia and asphyxia.

**Management**

There is no specific treatment and nursing care should be directed toward helping parents to adjust to rearing a child with cognitive impairment when this condition is present.

**Macrocephaly**

Macrocephaly is defined as a head circumference that is more than 2SD above the mean for that age.

**Causes**

Increased brain size is increased ventricular size (hydrocephalus)

**Management**

There is no specific treatment for macrocephaly, medical care for children with macrocephaly focuses on management of specific symptoms, such as developmental delays and mental retardation and the primary diagnosis responsible for the macrocephaly.

**Paediatric conditions**

**Impact of sickness and admission to child, parents and family**

**Immediate and long term**

**Child**

Immediate and long term emotional distress to the child, Children below the age of 5 years are more vulnerable to emotional distress form hospitalization.

Anxiety due to lack of understanding and minimal experience with illness, hospitalization and hospital procedures.

A lack of sensory stimulation in the hospital environment produces listlessness, indifference, unhappiness and even appetite changes. When a child’s motor is restricted, anger and hyperactivity may result.

**Parents**

Parents have anxiety and concern about their child’s health and this contributes to stress to the child. Parents watching children in pain is difficult and some may feel guilty of not bringing these children earlier to hospital.

Parents may exhibit other feelings such as denial, anger, depression, and confusion and parents may deny that their child is ill. They may exhibit anger due to lack of control of caring for their child and may be directed to the nursing staff, family members or higher power Depression may occur due to exhaustion, psychological and physiological requirements of spending long hours in hospital caring for the child. Confusion may arise due to dealing with an unfamiliar environment or the loss of parental roles, long separation and increased stress. At times parents feel helpless when they play a passive role in their child’s care, such as when a medical procedure is required that hurts or traumatizes the child.

**Siblings**

Sibling may experience jealousy, insecurity, resentment, confusion and anxiety. They may have, understanding why there sibling is ill or getting all the attention and leaving little for them. Due to changes in roles in the family children may feel insecure or anxious and they develop change in behavior or school performance, during this time siblings feel that they caused the illness.

**Family**

Whether planned or unplanned hospitalization increases the family’s stress and anxiety level and this affects all the members of the family, family’s normal routines are disrupted and may alter family roles. Parents and siblings have their own reactions to this experience.

**Management**

Liberal visitation

Rooming-in in hospital policy

Shorter hospital stays minimizes separation anxiety

Play, recreation and educational opportunities provide an outlet to distract children from illness

Parents attempt to understand by becoming informed and understanding the procedures. They deal with fear of uncertainty and attempt to promote a sense of comfort by interacting with hospital staff. They seek reassurance from the care givers.

**Respiratory conditions**

**Common cold (coryza)**

Commonest infection in children

**Causative organisms**

Viruses-Rhinovirus, corona virus and RSV (respiratory synytial virus)

**Clinical manifestations**

Clear or mucopululent nasal discharge and nasal blockage

**Management**

Health education to advice parents that colds are self limiting and have no specific curative treatment. Pain and fever may be treated with paracetamol or Ibuprofen. Antibiotics are not of any benefit and secondary bacterial infection is rare. Saline nasal drops every 3-4 hours.

**Streptococcal sore throat (Pharyngitis)**

Sore throat is inflammation of the pharynx and soft palate.

Etiology

Virus about 90%

Bacterial-Streptococcal infections, Group A Beta hemolytic streptococcal

**Pathophysiology**

The pharynx and soft palate are inflamed and local lymph nodes are enlarged and tender.

**Clinical manifestations**

Sore throat, cough, fever, irritability, malaise, nausea, vomiting, and poor appetite

**Management**

Treat with oral penicillin-Amoxicillin, Amoxicillin with clavunic acid or IM penicillin and dose should be completed, prescribe Erythromycin for children allergic to penicillin.

Analgesics-paracetamol

Cold or warm compresses may bring relief

Warm saline gargles for bigger children offer throat relief

Do not force to eat, however encourage cool liquids

**Tonsillitis**

Tonsillitis is extensive inflammation of the tonsils, often with a purulent inflammation

**Causative organism**

Viral infection

Bacterial infection-group A Beta hemolytic streptococci

**Clinical manifestations**

Sore throat, difficulty in swallowing, fever, nasal congestion, mouth breathing and drying of mucous membranes aggravates pain

**Pathophysiology**

Several pairs of tonsils are part of a mass of lymphoid tissue in oropharynx. Tonsillitis inflammation of the palatine tonsils

**Diagnosis**

Diagnosis is by visual inspection and throat culture

**Medical management.**

Viral infection will resolve, institute warm saline gargles, antipyretic.

Bacterial infections-Give penicillin/cefuroxime

Tonsillectomy with or without adenoidectomy can be done under the following conditions

* Recurrent streptococcal infection
* Enlarged tonsils affecting breathing/eating
* The child should be 3 years or older

**Nursing care**

Nursing care for children with tonsillitis includes proving comfort; provide a soft diet, warm salt water gargles, throat lozenges, and analgesics/antipyretics.

If surgery is needed- provide psychological preparation, take history which include; bleeding tendency because the operation site is highly vascular, take baseline vitals, blood test for clotting time and check on loose teeth.

**Post operative**

Post surgery- until they are fully awake, children should be placed on the abdomen or side to facilitate drainage of secretions, suctioning is performed carefully to avoid trauma on the operated site.

When alert, children may prefer sitting up, however they should remain in bed for the rest of the day.

Discourage from coughing frequently; cleaning their nose or any activity which would injure the operated site, inspect all secretions and vomitus for evidence of fresh bleeding.

Apply ice collar

Give analgesics

Give antiemetic.

Food and fluids are restricted until children are fully alert and with no signs of hemorrhage-cool water, crushed ice and diluted juice is given, however avoid giving fluids with a red or brown color so as to distinguish fresh and old blood in emesis from ingested liquid.

Straws should be avoided since they can damage the surgical site and cause bleeding

Citrus juice may cause discomfort and is usually poorly tolerated.

Milk, ice cream or pudding is not usually offered until clear fluids are retained because milk products coat the mouth and throat, causing the child clear the throat which may initiate bleeding

Children often begin with soft foods like gelatin, soup and mashed potatoes on the first or the second day post operatively as the child tolerates feeding

The nurse observes the throat directly for evidence of bleeding using a good source of light and if necessary, carefully inserting a tongue depressor.

Other signs of bleeding include tachycardia and pallor, swallowing and vomiting bright red blood, decreasing blood pressure is a late sign.

Check for a cream colored membrane which is normal and often visible on the tonsillar bed post operatively.

Check for airway obstruction which may occur due to edema or accumulated secretions and is indicated by signs of respiratory distress, such as a stridor, drooling, restlessness, agitation, increasing respiratory rate, and progressive cyanosis. Suction equipment and oxygen should be available post tonsillectomy.

**Advice on discharge**

Advice irritating and highly seasoned food

Avoid use of gargles and vigorous tooth brushing

Discourage the child from coughing or clearing the throat or putting objects in the mouth

Use analgesics or ice collar for pain

Limiting activity to decrease the potential for bleeding, hemorrhage may occur up to 10 days after surgery due to tissue sloughing from the healing process

Report any bleeding

Mouth odor and slight ear pain with low grade fever are common a few days after postoperatively

**Otitis media**

Def; Otitis media is an inflammation of the mucous membrane lining the middle ear space.

Three types

* Acute otitis media
* Chronic otitis media- has lasted >3 months
* Otitis media with effusion

**Etiology**

AOM is frequently caused by streptococcus pneumonia, Hemophilus influenza and moraxell catarrhalis, viruses, RSV, and influenza

**Pathophysiology**

Most vulnerable group is children below 5 years. Otitis media is primarily as a result of a dsyfuctioning Eustachian tube. Obstruction of the Eustachian tube causes accumulation of secretions in the middle ear. Enlarged adenoids, nasopharyngeal tumors, collapse of the tube during swallowing and inefficient opening causing negative pressure which produces transudative middle ear infections, impaired ciliary are some of the causes of dysfunction. When the passage is not totally obstructed, contamination of the middle ear can take place by reflux, aspiration.

**Clinical manifestations**

As purulent fluid accumulates in the small space in the middle ear, pain results from the pressure on the surrounding structures.

Infants are irritable and indicate their discomfort by holding or pulling at their ears and rolling their head from side to side, while older children complain of pain.

Fever, at times up to 40 degrees centigrade

Diarrhea, vomiting and URTI may be present

Due increased pressure the tympanic membrane may rupture resulting in immediate relief of pain.

**Diagnosis**

Otoscopic examination- Tympanic membrane is red and bulging.

Serous/purulent fluid behind tympanic membrane

Management

Acute otitis media- Treat with antibiotics-Amoxicillin, cefaclor, clotrimoxazole (Bactrim)

For 5-10 days. I n case of poor compliance give a single dose of im ceftriaxone (Rocephin)

Serous fluid takes weeks/months to clear.

Recurrent otitis media (occurring within 6months) need prophylaxis antibiotics treatment and treat URTI early.

Tympanoplasty (myringotomy) is a surgical incision in the tympanic membrane to drain fluid, is indicated if an episode of otits media with effusion last longer than 3-4 months and associated with loss of 20 Decibels.Ear plugs are used to prevent water entry to the ear when swimming.

During breastfeeding avoid horizontal position and 2nd hand smoking to prevent irritation

**Acute laryngotracheobronchitis**

**Croup**

Def; Croup is a group of symptoms characterized by hoarseness, a resonant cough described as ‘barking’ or ‘bassy’(croupy), varying degrees of inspiratory stridor and varying degrees of respiratory distress resulting swelling of the region of the larynx.

**Acute laryngotracheobronchitis** is the commonest type of croup and affects children less than 5 years of age, and it is inflammation of the mucosa lining the larynx, trachea, and bronchi, causing narrowing of the airway.

**Etiology**

Virus parainfluenza virus, RSV, influenza virus

**Management**

Maintain airway

Nebulized epinephrine which causes mucosal vasoconstriction and subsequent decrease of subglottic oedema

Use of corticosteroids due to their ant inflammatory effects

Give oxygen

If not able to take oral fluids, iv fluid therapy may be indicated

**Pneumonia**

Def; Pneumonia is the inflammation of the lung parenchyma. It is common in childhood and affects infants and early childhood.

**Classification**

Pneumonia can be classified according to morphology, etiological agent or clinical form

**Morphological classification**

Lober pneumonia-all or a large segment of one or more pulmonary lobes is involved

Broncho pneumonia-begins in the terminal bronchioles, which become clogged with mucopurulent exudates

Interstitial pneumonia- inflammation process confined within the alveolar walls.

**Classification by etiological agent**

Viral

Bacterial

Mycoplasmal

Aspiration of foreign substances

**Kenya paediatric guidelines-2016**

History of cough or difficulty breathing, age > 60 days

1. **SEVERE PNEUMONIA**

Clinical manifestations

**One of the danger signs**

* Oxygen saturation <90%
* Cyanosis
* Inability to drink/breastfeed
* AVPU=’V’, ‘P’ or ‘U’ or
* Grunting

**Management**

* Admit
* Oxygen
* Penicillin **Benzyl Penicillin** *(Crystalline Penicillin)* **Age 7 days and over:** 50,000 iu/kg/dose 6 hourly IV/IM and
* Gentamicin 7.5 mg/kg/24 hr IM or slow IV

1. **PNEUMONIA**

**Clinical manifestations**

* Lower chest wall indrawing or
* Fast breathing (RR 50/min (Age 2-11mo)
* RR 40/min (Age 12-59mo)

**Management**

* **Oral Amoxicillin**
* Counsel carefully on danger signs and need to return if these develop

All children must be reviewed within 48 hrs (if review is not Possible **admit** children with **indrawing** and treat with amoxicillin)

1. **No pneumonia,** probably URTI

**Asthma**

Def; Asthma is a chronic inflammatory disorder of the airways in which many cells play a key role. Asthma is characterized by chronic inflammation, bronchoconstriction and bronchial hyper responsiveness.

**Etiology**

Idiopathic

Allergy

**Pathophysiology of allergic types**

* Infection or allergen causes narrowing of the airways by;
* Contraction of the smooth muscles surrounding the airways in the lungs (bronchospasm)
* Swelling of the mucosal lining of the airways
* Increased secretions in the lumen of the airways

Genetic predisposition is an important predisposing factor for asthma. Apart from allergens wheezing can be precipitated in response to exercise and certain drugs.

The classical triad of asthma is cough, dyspnea (breathlessness) and wheezing

Not all children experience these symptoms.

Older children may complain of intermittent chest pain chest tightness.

Episodes of the asthmatic attacks are common at night, with change of season or after exercise.

History of recurrent similar episodes in the past in a child presenting with cough and difficulty in breathing, will be present.

**Classification as per Kenya peadiatric guidelines 2016**

Wheeze + History of cough or difficulty breathing,

*(Likelihood of asthma much higher if age > 12m and recurrent wheeze)*

**Severe Asthma**

**Any one of these;**

• Oxygen saturation <90%

• Central cyanosis

• Inability to drink / breast feed

• AVPU= “V”, “P” or “U” or

• Inability to talk/complete sentences

• Pulse rate >200 bpm (0-3 yrs) and >180 bpm (4-5yrs)

**Immediate Management**

**ADMIT**

* Oxygen
* Nebulize 2.5 mg salbutamol or 6 puff of inhaler with spacer and mask give every 20 minutes upto 3 doses if needed
* Prednisolone 2mg/kg
* Consider ipratropium bromide 250 mcg if poor response
* Antibiotics as for severe pneumonia

**Mild or Moderate Asthma**

Wheeze PLUS Lower chest wall indrawing

**OR**

* + (RR 50/min (Age 2-11mo)
  + RR 40/min (Age 12-59mo)
  + Fast breathing

**Management**

* Salbutamol 2 puffs of inhaler (or 2.5mg nebulized) every 20 minutes upto 3 doses if needed
* If mild symptoms allow home on salbutamol MDI give 2 puffs every 6 hours.
* *Counsel caregiver on signs of deterioration and schedule review within 48 hours.*
* Monitor closely for 1-2 hours

If lack of response to salbutamol, increasing respiratory rate, worsening saturation, any signs of severe asthma.

Refer to **Immediate Management** above

NB Recurrence of asthma symptoms consider inhaled corticosteroid (ICS) therapy or adjusts the doses if already on ICS**. (L*ook out for other*** ***comorbidities)***

Demonstrate MDI and spacer use to the caregiver before discharge from the health facility. Preferably use spacer with face masks for <3 years for 4-5 years use facemask or mouthpiece

Advise on regular follow up.

Prednisolone administered for 3-5 days. Max dose of 20mg/day for < 2 years and 30mg/day for 2-5 years.

Repeat Consider ipratropium bromide 250 mcg every 20 minutes for one hour if needed



**Cardiovascular conditions**

**Rheumatic fever**

Rheumatic fever is a poorly understood inflammatory disease that occurs after pharyngitis caused by Group A beta hemolytic streptococci.

**Etiology**

This is an immunological disorder caused by damage to the heart by antibodies, which are produced in response to a primary infection of the throat or skin by Group A Beta hemolytic streptococci.

**Pathophysiology**

The exact pathogenesis is not known. Following untreated streptococcal sore throat there is a latent period that may last from 10 days to several weeks, and during this period there are antibodies from the streptococcal antigen. These antibodies have the capacity to react with human connective tissue, specifically the cardiac muscle. This antigen antibody reaction results in Rheumatic fever.

Mostly affects children aged 5-15 years of age.

**Clinical manifestations**

1. The first major cardiac manifestation of rheumatic fever is carditis involving endocardium, myocardium, and pericardium

Pericarditis present with chest pains and a pericardial rub on auscultation

Myocarditis present with tachycardia, edema, tacypnea and cardiomegally

Endocardial involvement is indicated is indicated by a presence of a thrill and precordial murmurs. Aortic and mitral valves are commonly affected.

1. The second major is polyarthritis caused by edema, inflammation and effusions in joint tissue. These nodules are reversible and migratory and favour large joints, such as the knees, elbow, hips, shoulders and wrists. Affected joints are swollen, hot, red and painful for 1-2 days after which another joint is affected. Fever is present in 1-2 weeks and can persist up to 4 weeks in untreated patients.
2. The third major manifestation is erythema marginatum –a rash that is reddish, macular and non itching, and predominantly observed on trunk
3. The fourth major manifestation is the development of subcutaneous nodules, which are small, non tender swellings that persist after the onset of the disease and gradually resolve with no resulting damage, and may be found on bony prominences such as the feet, hands, elbows, scalp, scapulae and vertebrae.
4. The last major manifestation which reflects central nervous involvement is chorea also referred to as St.Vitus dance and is characterized by sudden aimless, irregular movement of the extremities, involuntary facial grimaces, speech disturbances, emotional liability and muscle weakness. It is usually exaggerated by anxiety and attempts to use fine motor activity and is relieved by rest.

**Diagnosis**

Throat culture may be positive or not even though the child has the infection

Echocardiogram for valvular disease

**Management**

In acute phase, the aim to eradicate the organism and decease inflammation process, use oral penicillin as the initial treatment for 10 days or single IM penicillin.

Aspirin- Anti-inflammation- high dose 100/kg/day, then decrease to 70mgs/kg/day, after the acute phase discontinue Aspirin

Give antacids because of Aspirin gastric effects

Bed rest until inflammation resolves

Restrict activities because of aortic insufficient/mitral regurgitation

**Secondary prophylaxis**

Oral penicillin 250mgs bid or a monthly im injection of penicillin, this is done for at least 10 years or more

Throat infection should be treated timely and does take, avoid under treatment

**Rheumatic heart disease**

RHD is a chronic heart condition caused by RF that can be prevented and controlled. Acute rheumatic fever causes fibrosis of heart valves, leading to crippling valvular heart disease, heart failure and death.

Surgery is often required to repair heart valves in patients with severely damaged valves, the cost of which is high and a drain on the limited health resources of poor countries.

**Anaemia**

Anaemia is a condition marked by a deficiency of red blood cells or of hemoglobin in the blood, resulting in pallor and weakness. Anaemia is not a disease but a manifestation of underlying pathologic process

**Classification**

1. Etiology has manifested by erythrocyte or hemoglobin depletion
2. Morphology, the characteristic changes in RBC size, shape and color

**Causes**

1. Inadequate production of RBC or RBC components
2. Increased destruction of RBC
3. Excessive loss of RBC through hemorrhage

**Pathopysiology**

The basic physiologic defect caused by anemia is a decrease in oxygen carrying capacity of blood which leads to a reduction in the amount of oxygen available to the cells. When the anaemia develops slowly, the child usually adapts to the declining hemoglobin level.

**Red blood cell morphology**

**Size**

Normocytes-normal RBC

Microcytes- Smaller than normal RBC

Macrocytes- Larger than normal RBC

**Shape**

Spherocytes-Globular cells

Drepanocytes-Sickle shaped cells

Numerous-Other irregulary shaped cells

**Color**

Normochromic-Normal amount of hemoglobin per red blood cell

Hypochromic-Reduced amount of hemoglobin per red blood cell

Hyperchromic-Increased amount of hemoglobin per red blood cell

**Signs and symptoms of anemia**

Pallor

Tachycardia

Fatigue, headache

Muscle weakness

Systolic heart murmur due to decreased red call production

Frontal bossing

Jaundice

Dark urine

Splenomegaly

Hepatomegaly

Low blood pressure- a late sign of shock

Decreased peripheral pulses due to increased red cell loss

**Diagnosis**

* Clinical presentation and history
* Capillary blood count and hematocrit levels will show low numbers of red blood cell, low hemoglobin, low hematocrit, low reticulocytes(which indicate the body’s response to an increased demand for red blood cells) A peripheral smear may demonstrate significant changes in the shape of red blood cells such as sickled cells
* Bone marrow aspirates-which will show the body’s ability to produce normal cells

**Management**

Objective is to reverse the anaemia by treating the underlying cause

In nutritional anaemia’s the specific deficiency is treated

In blood loss from acute hemorrhage, red blood cell transfusion may be given

In patients with severe anaemia, supportive medical care may include oxygen therapy, bed rest and replacement of intravenous volume with intravenous fluids

A detailed history is necessary on nutrition, history of chronic illness, recurrent infections, eating habits, particularly pica(consumption of non nutritive substances such as soil), bowel habits, and presence of blood in stools, familial history of hereditary diseases such as sickle cell disease and Thalassemia.

Assess the child’s level of tolerance for activities of daily living and play and make adjustments to allow as much self care as possible without undue exertion

Do vital signs and have a baseline when at rest and monitor the difference after activities

**Prevent complications**

Children with anaemia are prone to infection and their immune system is weak

Protect infections by hand washing, selecting a room in a non infectious area, restricting the presence of visitors or hospital personnel with active infection and maintain adequate nutrition

Observe for signs of heart failure. So prevent this by administering oxygen to minimize hypoxia and closely monitor intravenous fluids and administer packed red blood cells.

**Diarrhea**

Diarrhea is the frequent discharge of semi-solid or liquid faecal matter from the bowels. Diarrhea is a symptom that results from disorders involving digestive, absorptive and secretory functions. It is caused by abnormal intestinal water and electrolyte transport

Types of diarrhea

**Acute**

Acute diarrhea is a leading cause of illness in children younger than five years of age which is defined as a sudden increase in frequency and a change in consistency of stools, often caused by infections agents in the GIT tract. Acute diarrhea is usually self limiting <14 days duration.

**Etiology**

Most pathogens which spread diarrhea are spread by the fecal-oral route through contaminated food or water or from person to person where there is close contact e.g. day care centers, lack of clean water, crowding poor hygiene, nutritional deficiency and poor sanitation are major risk factors. Infants are more susceptible to frequent and severe bouts of diarrhea due to poor immune system

**Causes of acute diarrhea**

Bacteria e.g. salmonella, shigella, campylobacter, Escherichia coli, clostrium dificile, staphylococcus aureas

Viruses-Rota virus which causes about 70% of diarrhea, adenovirus

Parasites- Girdia lamblia, entamoeba histolytica

Associated conditions e.g URTI, UTI,otitis media

Dietary causes e.g over feeding, introducing new foods, osmotic diarrhea from excess sugar in formula or juice

Medications- Antibiotics, laxatives

Toxic substances- Ingestion of heavy metals (arsenic, lead, mercury) organic phosphates

Functional causes – I rritable bowels syndrome

Other causes- Hirshsprung enterocolitis

**Chronic diarrhea**

Chronic diarrhea is an increase in stool frequency and increased water content within duration of more than 14 days.

**Causes**

Malabsorption syndromes

Inflammatory bowel disease

Immunodeficiency

Food allergy

Lactolose intolerance

Or chronic non specific diarrhea as a result of inadequate management of acute diarrhea

**Pathopysiology**

Invasion of the GIT tract by pathogens results in increased intestinal secretion as a result of enterotoxins, cytotoxic mediators (those damaging the cell) and decreased intestinal absorption secondary to intestinal damage or inflammation. The most serious and immediate physiologic disturbances associated with severe diarrhea diarrheal disease are;

1. Dehydration
2. Acid base balance with acidosis
3. Shock that occurs when dehydration progresses to the point that circulatory status is seriously impaired

**Diagnosis**

History- Ask about recent travel, exposure to untreated drinking water or washing water sources etc

Stool for ova/cyst

**Management of** **Diarrhoea / Gastroenteritis**

History of diarrhoea / vomiting, age > 1 months

**Hypovolaemic shock from**

**diarrhoea / dehydration**

***All four of***

• Weak/absent pulse;

• AVPU < A;

• Cold hands + Temp gradient;

• Capillary refill > 3 secs

PLUS sunken eyes and

slow skin pinch

*NB:I f Hb< 5 g/dl, transfuse urgently*

Ringer’s 20 mls/kg a second bolus may be given if required before proceeding to step 2 of Plan C (*see below)*

Treat for Hypoglycaemia

**SEVERE DEHYDRATION**

(**Plan c)**

Unable to drink or AVUP < A plus;

Sunken eyes

Return of skin pinch≥ 2 secs

**Step 1**- 30 mls/kg Ringer’s over 30 mins if ≥ 12m OR

Over 60 mins if age < 12m

Step 2- 70 mls/kg Ringer’s over 2.5 hrs if age ≥12m OR

Over 5 hrs if age < 12m

Start ORS at 5 mls/kg/hr, once able to drink

OR

NG rehydration-120mls/kg ors over 6 hrs

NB Re-assess at least hourly and after 3-6hrs, re-classty as sever some or ni dehydration and treat accordingly

**SOME Dehydration**

Able to drink adequately but **2 or more** of:

• Sunken eyes

• Return of skin pinch 1 - 2 secs

• Restlessness / irritability

**Plan B**

1) ORS by mouth at 75 mls/kg over 4 hrs**, plus,**

2) Continue breast feeding as tolerated

• Sunken eyes

Reassess at 4 hrs & treat according to classification

**NO Dehydration**

Diarrhoea with fewer than 2 of the above signs of dehydration

**Plan A**

1) 10mls/kg ORS after each loose stool

2) Continue breast feeding and encourage feeding if > 6 months

All cases to receive Zinc. Antimicrobials are NOT indicated unless there is dysentery or proven amoebiasis or giardiasis.

**Urinary tract conditions**

**Urinary tract infection**

Urinary tract infection (UTI) is a clinical condition that may involve the urethra and bladder (lower urinary tract) and ureters, renal pelvis, calyces and renal parenchyma (upper urinary tract) Since it is impossible to localize the infection, the term UTI is applied to the presence of significant numbers of micro-organisms anywhere within the urinary tract (except the lower third of the urethra which is usually colonized by bacteria

**Classification of UTI or inflammations**

Bacteriuria- Presence of bacteria in urine

Asymptomatic bacteriuria- Bacteria accompanied by physical signs of urinary infection, dysuria, suprapubic discomfort, hematuria, fever)

Recurrent urinary tract infection-Repeated episode of bacteriuria or symptomatic UTI

Persistent UTI- Persistence of bacteriuria despite antibiotic treatment

Febrile UTI- Bacteria accompanied by fever and other physical signs

Cystitis- Inflammation of the bladder

Urethritis- Inflammation of the urethra

Pyelonephritis- Inflammation of the upper urinary tract and kidneys

Urosepsis- Febrile UTI coexisting with systemic signs of bacterial illness, blood culture reveals presence of urinary pathogens

**Etiology**

A number of organisms are responsible for UTI, Escherichia (coli 80%), and other gram negative enteric organisms associated with UTI. Other organisms associated with UTI include proteus, pseudomonas, klebsiella, staphylococcus aureas, haemophilus.

A number of factors contribute to the development of UTI including anatomical, physical and conditions or properties of the host’s urinary tract.

In females- the short urethra and the closure of the urethra at the end of micturation may return contaminated urine by bacteria to the bladder

The longer male urethra(in an adult) and the antibacterial properties of prostatic secretions inhibit the entry and growth of micro-organisms, the presence or absence of the foreskin contributes to the differences in UTI rates in infants

Urine that remains in the bladder allows bacteria from the urethra to rapidly become established in the rich medium

Incomplete emptying of the bladder can cause UTI, which could result from anatomic abnormalities, (esp. those of the ureters) or bladder compression.

The pressure of over distension within the bladder may increase the risk of infection by decreasing the host resistance probably due to decreased blood flow.

Other factors include constipation which displaces the bladder and posterior urethra in a fixed and limited space of the bony pelvis, causing obstruction, incomplete micturation and urinary stasis, failure to relieve the impaction in spite of adequate treatment of UTI mat result to recurrence

Other causes of UTI include urinary catheters, tight clothing, diapers, poor hygiene, local inflammation, vaginitis, masturbation or pin worm infestation may increase the risk of ascending infection. Essential oils in bubble baths and shampoos can irritate the urethra causing painful and frequent urination. Sexual intercourse is associated with increased risk of UTI.

Pathophysiology

After bacteria invasion by bacteria, the host first line of defense in the lower urinary tract is complete evacuation by voiding. Inflammation in the bladder and urethral walls is apparent within 30 minutes of invasion by a bacterial pathogen. Polymorphonuclear leukocytes rapidly migrate to the bladder which becomes infected within 2 hours, complete evacuation of the bladder is particularly important for the eradication of bacteria from the urine. Urination removes bacteria and associated toxins contained in urine and allow more efficient destruction of the bacteria remaining on the thin film of urine that is closely adheres to the vesical wall

**Clinical manifestations**

Depend with age

Infants and children < 2 years of age the signs are non specific and they more resemble GIT disorders, which include failure to thrive, feeding problems, vomiting, diarrhea, abdominal distension, and jaundice. Newborns may have fever, hypothermia or sepsis, frequent or infrequent voiding, constant irritability, strong smelling urine and an abnormal stream. A persistent diaper rash is also a helpful clue. Children > 2 years of age the classic symptoms include enuresis or daytime incontinence in a child who has been toilet trained, fever strong or foul smelling urine, increased frequency of urination, dysuria or urgency or urgency. They may also complain of abdominal pain or flank pain. Some present with hematuria, some may present (boys) dribbling of urine, straining with urination or a decrease in the force and size of the stream, high fever and chills, leukocytosis suggest pyelonephritis. Adolescent have more specific symptoms-include frequency and painful urination of a turbulent urine that may be bloody, fever is usually absent, however upper tract infection is characterized by fever, chills, frank pain

**Diagnosis**

This depends on a high degree of suspicion, history taking and physical examination

Urine sample with possible infection appears cloudy, hazy or thick with noticeable strands of mucus and pus; it also smells fishy and unpleasant

Presumptive diagnosis; Urine test may reveal, pyuria and presence of bacteria, also normal urine may present in conditions of asymptomatic bacteriuria

Detection of bacteria in a urine culture confirms the diagnosis of UTI, but urine collection is often difficult esp. in infants and small children, which may lead to contamination of a specimen by organisms from other sources. Bag urine specimens are commonly contaminated.

Unless the specimen is a first morning sample, a recent high fluid intake may indicate a fasely low organism count. Therefore do not encourage children to drink large volumes of water in an attempt to obtain a specimen quickly. The most accurate tests of bacterial content are suprapubic aspiration and properly performed bladder catheterization (as long as the first few mls are excluded from collection). The urine specimen for culture should be taken to the laboratory immediately, if culture is delayed place the sample in a refrigerator for upto 24 hours, but storage can result in a loss of formed elements. Presence of nitrites on dipstick analysis identifies infected urine and absence of nitrites and leukocyte in combination identifies infected urine. These test results are used to initiate treatment of UTI while culture results are pending some organisms are non-nitrite producing e.g. pseudomonas organisms.

Localization of the infection may involve more specific tests including ureteral catherization, bladder washout procedures, radio isotope renograpy, ultrasonograpy, intravenous pyelograpy.

NB. For the best results, wash the perineal area thoroughly before applying the urine collection bag, and promptly remove the bag as soon as voiding occurs. Leaving the device in situ for more than one hour is more likely to yield a contaminated urine specimen.

**Management**

Objectives for treatment are;

* Eliminate current infection
* Indentify contributing factors to reduce risk of recurrence
* Prevent orosepsis
* Preserve renal function

Antibiotics therapy are prescribed on the basis of culture results, however empiric therapy on the basis of child’s history and presenting symptoms may be necessary. Antibiotics in use include penicillins, sulphonamides (including trimethoprin sulfamethoxale), the cephalosporins, nitrofurantoin and the tetracyclines. Children with suspected pyelonephritis and fever are admitted to the hospital and given appropriate antibiotics intravenously for a minimum of 48 hours. Blood and urine cultures are done on admission and after therapy, and therefore repeated at monthly intervals for 3 months and 3 months interval for another six months. Renal scarring can develop during the initial infection esp. in younger children and therefore the first UTI in childhood necessitates radiological examination

Anatomical defects such as bladder neck obstruction or bladder reflux may require surgical correction to prevent recurrent infection or may indicate the need for prophylactic antibiotics and careful follow up monitoring since relapse rate is high and recurrent infection tends to occur 1 to 2 months after termination of treatment. The aim of the therapy and careful follow up in such cases to prevent morbidity and reduce the chance of renal scarring

Educate the parents and children on prevention and treatment of infection

Encourage adequate fluid intake for the prevention and treatment of UTI, since bacterial eradication from the urinary tract is partially dependent on urine flow and voiding frequently, patient to drink clear fluids, children to avoid caffeinated and carbonated beverages because of their potentially irritative effect on bladder mucosa. A child who is febrile and unable to drink liquids is given intravenous hydration until the fever resolves and oral liquid is given intravenous hydration until fever resolves and oral liquids are tolerated.

**Prognosis**

With prompt and adequate treatment at the time of diagnosis, the long term prognosis for UTI’s is usually excellent. When infection occurs in young children < 2 years of age there is a greater hazard of progressive renal injury and is associated with congenital renal malformations and reflux, therefore early diagnosis is important.

**Acute glomerulonephritis**

This is sudden inflammation of glomeruli within the kidney which results in acute renal failure. Glomerulus gets damaged, hence referred as intrarenal acute renal failure. It may affect glomerular capillaries or membrane

**Incidence**

Glomerulonephritis can occur at any age but has a peak age of onset of 6 to 7 years and rare in children <2 years. Common in boys than girls

**Etiology**

Infectious agent is usually in the body 2-3 weeks before clinical manifestation and it could be bacterial (streptococcus group A commonest) e.g. acute post streptococcal glomerulonephritis or viral

**Pathophysiology**

The mechanism by which the reaction takes place is still speculative. The most popular proposal to explain the pathologic process is that the streptococcal infection is followed by a release of a membrane like material from the specific organism into the circulation, and since it is antigenic, antibodies are formed and an immune complex reaction occurs after the appropriate period. These immune complexes become trapped in the glomerular capillary loop and inflammation occurs. End result is scarred/ damaged glomeruli, membrane permeability altered by immune response. Protein leak into urine, glomeruli filtration rate decreases, sodium and water are retained and oedema occurs, cause of elevated BP is not explained

**Clinical manifestations**

Heamaturia

Dependent and periobital oedema and puffiness of the face and oedema is more prominent in the morning

Diminished urinary output

Proteinuria

Increased BP

Fatigue

Decreased filtration

Increased serum sodium level and increased potassium levels

Increased BUN and creatinine

Low grade fever

Urine becomes blood tinged, smoky or tea coloured or cola coloured urine

Anorexia

**Diagnosis**

Clinical manifestations

Physical exam

Immunological test to detect streptozyme and serum complement

**Management**

Depend on the kidney damage

The aim is to treat source of inflammation and maintain fluid and electrolytes and maintain the BP within normal range

Bed rest is no longer recommended during the acute phase because ambulation does not seem to have an adverse event on the course of the disease. During acute phase children are listless and experience fatigue and malaise, most children restrict their activities.

Fluid balance

Do vital signs, body weight (daily), intake and output (daily)

Water intake is calculated, insensible loss plus the volume of urine passed.

Diuretic therapy is helpful when renal is not severe. In case of severe renal failure diuretic therapy is not useful.

Hypertension: Take BP readings 4 to 6 hours, and in case of elevated BP start on antihypertensive drugs such as calcium channel blockers, beta blockers or angiotensin- converting enzyme inhibitors

Monitor seizures associated with hypertensive encephalopathy and incase of seizures give anticonvulsants.

Nutrition: Limit sodium intake- moderate sodium intake restriction, protein intake restrictions to those with azotemia and prolonged oliguria

Antibiotics are indicated only for children with evidence of persistent streptococcal infections

**Nephrotic syndrome**

Nephrotic syndrome is defined as massive proteinuria, hypoalbuminemia, hyperlipidemia and edema

**Types**

Primary neprotic syndrome is when the syndrome is restricted to glomerular injury

Secondary nephritic syndrome when it develops as part of a systemic illness e.g. toxins, lead poisoning

Congenital nephritic syndrome- infant with nephritic syndrome within 3 months of life are considered congenital e.g. due to autosomal recessive disorder

**Cause**

Idiopathic, is thought to be caused by immune response while the other is caused by infections, drugs and toxins

Common in males than females 2:1, it commonly affects those aged between 2 to 6 years.

**Pathophysiology**

The inflammation process from immune response or disease makes glomeruli to become permeable to proteins (proteinuria), this reduces serum albumin level ( hypoalbuminemia), fluid shift from the intravascular space to interstitial space which subsequently leads to oedema/ ascites which lead to hypovolemia, this in turn stimulates the rennin angiotensin system and the secretion of antidiuretic hormone and aldosterone. Tubular reabsorption of sodium and water increases in an attempt to increase intravascular volume. The elevation of lipids is not fully understood

**Clinical manifestations**

Puffness of the face, around the eyes mostly in the morning and subsides later during the day, swelling of the abdomen and lower extremities

Anorexia, irritability, abdominal pain and diarrhea (due to edema of the interstitial mucosa)

Volume of urine is decreased and appears darkly opalescent and frothy

**Diagnosis**

Dependent on proteinuria

Clinical manifestations

Reduced total serum protein (<2g/dl)

Renal biopsy- can show the type of nephrotic syndrome

**Management**

The primary aim is to reduce the excretion of urinary protein and maintain protein free urine; additional objectives include treatment of acute infection, control of oedema, and establishment of good nutrition and readjustment of any disturbed metabolic processes

General principle is generally supportive

Diet- During the period of massive oedema and corticosteroid therapy salt is restricted; a diet rich in protein is logical, however in case of azotemia or renal failure restrict high protein diet.

Corticosteroid therapy- administer per oral 60 mg/m2/day in evenly divided doses- use prednisolone continue od × 6 weeks, then decrease to 40 mg/ m2  alternate days for more 6 weeks (should be continued for at least 3 months)

Immunosuppressant therapy is used to reduce the relapse rate and induce long-term remission in children with frequent relapsing or steroid resistance nepthrotic syndrome, oral cyclosphosphamide or chlorambucil, cyclosporine

Diuretics like frusemide can be used, but monitor sodium, potassium, and hypovolemia

Antibiotics may be required in case of infection

Nursing- prevent infection and skin breakdown

Fluid restriction if child is hyponatremic

Elevate swollen scrotum with pillows to enhance the removal of fluid by gravity.

**Meningococcal meningitis**

Bacterial meningitis is an acute inflammation of the meninges and CSF

Meningcococcal meningitis occurs in epidermic form and is the only type readily transmitted by droplet infection from nasopharygeal secretions

**Etiology**

The causative organism of meningococcal meningitis is neisseria meningitidis, also called meningococcus.

Although this condition may develop at any age, the risk of meningococcal infection increases with the number of contacts, therefore it occurs in school –age children and adolescents and appears to be some seasonal variations. Meningitis caused by pneumococcal infections or meningococcal infections occur at any time

Maternal factors such as premature rupture of fetal membranes and maternal infection during the last weeks of pregnancy are major causes of neonatal meningitis

Risk factors for developing meningitis include recent exposure to someone with meningococcal meningitis, recent ear infections or sinus infection, travel to areas where bacterial meningitis is common, penetrating head trauma, cochlear input devices, and anatomic defects such as dermal sinus, urinary tract anomaly or recent placement of ventricular shunt

**Pathophysiology**

The most common route of infection is vascular dissemination from a focus of infection elsewhere.e.g. organisms from nasopharynx invade the under lying blood vessels, cross the blood brain barrier and multiply in the CSF. The infective process is that of inflammation, exudation, white blood cell accumulation and varying degrees of tissue damage. The brain becomes hyperemic and edematous and the entire surface of the brain is covered by a layer of purulent exudates. As the infection extends o the ventricles, thick pus, fibrin or adhesions may occlude the narrow passage s and obstruct the flow of CSF

**Clinical manifestations**

Usually abrupt on onset with;

Fever

Chills

Headache

Vomiting

Altered conscious level

Seizures

Irritability

Agitation

Neck rigidity

Kerning’s sign and brudzinski positive in children and adolescent

Petechial or purpuric rash

Poor feeding / breastfeeding

Neonatal bulging of the fontanelle

**Diagnosis**

Lumber puncture is the definitive diagnostic test

CSF- culture and sensitivity

**Management**

Acute bacterial meningitis is a medical emergency and requires early recognition and immediate therapy to prevent death and residual disabilities

Isolation precautions

Antibiotics – preferably cephalosporins

Maintenance hydration

Maintenance of ventilation by positioning the patient

Reduction of increased intracranial pressure by elevating the bed

Management of systemic shock

Control of seizures

Control of temperature

**Malnutrition**

Malnutrition is lack of proper nutrition, caused by not having enough to eat, not eating enough of the right things, or being unable to use the food that one does eat. It is a major health condition affecting under 5 years of age. The most extreme forms of malnutrition or protein- energy malnutrition are kwashiorkor and marasmus

**Etiology**

Diarrhea (gastroenteritis) is a major factor

Other factors are bottle feeding (in poor sanitary conditions)

Inadequate knowledge of proper child care practices

Parental illiteracy

Economic and political factors

Climate conditions

Cultural and religious food preferences

Lack of adequate food

PEM may also be seen in persons with chronic health problems

**Pathophysiology**

In general, marasmus is an insufficient energy intake to match the body’s requirements. As a result the body draws on its own stores, resulting in emaciation.

In kwashiorkor, adequate carbohydrate consumption and decreased protein intake lead to decreased synthesis of visceral proteins. The resulting hypoalbuminemia contributes to extravascular fluid accumulation; impaired synthesis of B- lipoprotein produces fatty liver. PEM also involves, an inadequate intake of many essential nutrients, low serum levels of zinc have been implicated as the cause of skin ulceration in many patients

**Kwashiorkor**

Kwashiorkor is defined as primarily a deficiency of protein with an adequate supply of calories in form of carbohydrates but inadequate amount of proteins The term is taken from the Ga language of Ghana and means ‘the sickness of the weaning’

**Clinical manifestations**

The child with kwashiorkor has thin, wasted extremities and a prominent abdomen from oedema (ascitis) and the oedema often masks severe muscular atrophy

The skin is scaly and dry and has areas of depigmentation

Dermatoses of the skin may be evident, partly resulting from vitamin deficiencies.

Permanent blindness often results from lack of vitamin A

Acute zinc deficiency is a common complication of severe PEM and results in skin rashes, loss of hair, impaired immune response and susceptibility to infections, digestive problems, night blindness, and changes in mood, defective wound healing and impaired growth. Its depressant effects limits food intake. The hair is thin, dry, coarse and dull. Depigmentation is common and patchy alopecia may occur.

Diarrhea occurs from lowered resistance to infection and further complicates electrolyte imbalance

Anaemia is also a common finding in malnourished children.

Protein deficiency increases the child’s susceptibility to infection which can result to death.

**Marasmus**

Marasmus results from general malnutrition of both calories and protein. Marasmus is usually a syndrome of physical and emotional deprivation. Marasmus is derived from the Greek word marasmus, which means withering or wasting. Marasmus involves inadequate intake of protein and calories and is characterized by emaciation.

Marasmic kwashiorkor is a form of PEM in which clinical findings of both kwashiorkor and marasmus are evident.

**Clinical manifestations**

Marasmus is characterized by gradual wasting and atrophy of body tissues, especially of subcutaneous fat. The child appears to be very old, with loose and wrinkled skin. Fat metabolism is less impaired than in kwashiorkor, thus deficiency of fat soluble vitamins is usually minimal or absent. The child is fretful (constantly worried), apathetic, withdrawn and so lethargic that prostration frequently occurs.

**Diagnosis**

**Measuring nutritional status**

Anthropometry (body measurement) quantifies malnutrition. In children, measurement of mid-upper arm circumference (MUAC) is the most simple.

Weight and height measurements can be useful to detect wasting and stunting and individual monitoring over time e.g. growth velocity.

**Mid upper arm circumference (MUAC)**

MUAC is measured using a tape around the left upper arm. MUAC is quicker in sick patients so use MUAC in acute management.



**Weight, Height and Age**

***Weight for height (W/H) :*** Measure length lying if aged <2 y to give weight for length. Low W/H (or W/L) = wasting, and indicates acute malnutrition.

***Weight for age (W/A):*** Low W/A does not distinguish acute from chronic malnutrition. W/A is thus **not used** for diagnosis of acute malnutrition, but can be used to monitor growth e.g. in the MCH booklet

In the diagnosis of acute malnutrition we use W/H ***expressed as Z scores***. Z - scores can be obtained from simple tables

**Visible Severe Wasting** tends to identify only severest cases of SAM. It is better to use MUAC or WHZ score.

**Kwashiorkor = severe malnutrition** *(at any age)*

|  |
| --- |
| **Classifying malnutrition**  *(for WHZ values)* |

|  |  |  |
| --- | --- | --- |
| **Acute Malnutrition**  *(severity)* | **MUAC**  (*cm)* | **WHZ** |
| None | >13.5 | > - 1 |
| At Risk | 12.5 to 13.4 | > - 2 to < 1 |
| Moderate | 11.5 to 12.4 | > - 3 to < - 2 |
| Severe | < 11.5 | < - 3 |
| Severe | Kwashiorkor | Kwashiorkor |

**Management**

**Complicated severe acute malnutrition**

***age 6 - 59 months***

Check using ABC approach and admit if acute illness ***and either*** of:

• MUAC < 11.5 cm (*or visible severe wasting if no MUAC* ) with WHZ < 3 used if child aged < 6 months

• Oedema / other signs of Kwashiokor ( *flaky pale skin /h air changes)*

**Step 1**

• Check blood glucose and treat if < 3 mmol/l (*5 m ls/kg1 0% dextrose*

If glucose test unavailable treat for hypoglycaemia *if not ale*rt

• Oral / NG glucose or feeds should as soon as possible (*not > 30 mins after admission)*

**Step 2**

* Check for hypothermia, axillary temperature <35⁰C.
* If present warm with blankets, warm bags of fluid or a heater

**Step 3**

Check for dehydration if has diarrhoea. If in shock, use IV fluids if not in shock use ReSoMal

• Transfuse *if Hb< 4 g/dL*, 10mls/kg whole blood in 3hrs + frusemide 1mg/kg

**Step 4**

Electrolyte imbalance. **Use commercial F75**. *If not available* mineral mix and 4 mmol/kg/day of oral potassium may needto be added to feeds, *Never use Frusemide for oedema!*

**Step 5**

All ill children with SAM should get IV Penicillin (*or Ampicillin*) **AND** Gentamicin. Give 5 days gentamicin, if improved change Pen to Amoxicillin at 48 hrs. **Add**:

• Nystatin / Clotrimazole for oral thrush if present

• Albendazole after 7 days treatment.

• TEO (*+ atropine drops*) for pus / ulceration in the eye

**Step 6**

Correct micronutrient deficiencies. **Give**:

• Vitamin A (PO) if eye signs on admission and days 2 and 14.

• Multivits for at least 2 weeks *if no RUTF or F75/F100*

• Folic acid 2.5mg alt days *if no RUTF or F75/F100*

• Iron ONLY when child is gaining weight & *If no RUTF*

**Step 7**

Prescribe feeding needed (*see chart*) and place ng.

**Steps 8, 9 & 10**: Ensure appetite and weight are monitored and start catch-up feeding **with RUTF** or F100 (*usually day 3- 7)*. Provide a caring and stimulating environment for the child and start educating the family so they help in the acute treatment and are ready for discharge.

**Fluid management *in severe malnutrition with diarrhoea***

**Shock:**

AVPU<A , *plus* absent, or weak pulse plus prolonged capillary refilling (>3s) *plus* cold periphery with temperature gradient **20** **mls/kg in 2 hrs of Ringer’s lactate with 5% dextrose** – add 50 mls 50% dextrose to 450 mls Ringer’s lactate

***If severe anaemia start urgent blood transfusion not Ringer’s.***

**If not in shock or after treating shock**

If unable to give oral / ngt fluid because of very poor medical condition use / continue with iv fluids at maintenance regimen of 4mls/kg/hr

**If able to introduce oral or ng fluids / feeds:**

**For 2 hours:** Give ReSoMal at 10mls/kg/hour

**Then:** Give ReSoMal at 7.5ml/kg over 1 hour then introduce first feed with F75 and alternate ReSoMal with F75 each hour at 7.5mls/kg/hr for 10 hours - can increase or decrease hourly fluid as tolerated between 5 - 10 mls/kg/hr.

At 12 hours switch to 3 hourly oral / NG feeds with F75

**Feeding children with severe malnutrition*(age 6 - 59 months)***

• If aged < 6 months use EBM or term formula or use diluted F100 - to each 100mls F100 add 35mls clean water

• When appetite returns (and oedema much improved) **change from F75 to F100 at 130mls/kg (the same volume as**

**F75 for no oedema) in the transition phase (about 2 days)**, if F100 not available change to RUTF for transition phase.

**• After transition phase use RUTF** that has 500 kcal in 92g packets for **rehabilitation**. All vitamins, minerals and iron are in RUTF. Allow the child to nibble RUTF very frequently. RUTF can be mixed into uji or other foods slowly introduced.

**F75 – acute feeding**

**No or moderate oedema** *(130mls/kg/day)*

**Severe oedema, even face** *(100mls/kg/day)*

*If respiratory distress or oedema gets worse or the jugular veins are engorged reduce feed volumes*

**F100 Transition phase**

Replace starter F-75 with an equal amount of catch-up F-100 for 2 days.

On the third day if on F-100, increase each successive feed by 10 ml until some feed remains uneaten(usually at 200ml/kg/day).

**F100 Rehabilitation phase**

Monitor vital signs. If both pulse and breathing rates increase (breathing by 5 breaths/min and pulse by 25 beats/min), sustained for two successive 4-hourly readings, then:

Reduce the volume fed to 100 ml/kg per day for 24 h.

**Malignancies**

**Wilm’s tumour**

Wilm’s tumour or nephroblastoma is a malignant tumour of the kidney and is the most common kidney tumour of childhood and common below 5 years of age. About 2% of Wilm’s tumour is family related

**Clinical manifestations**

* Painless swelling or mass within the abdomen. The mass is firm, non tender, confined to one side, and deep within the flank.
* Other signs and symptoms are the result of compression from the tumor mass, metabolic alterations secondary to tumor or metastasis
* Haematuria
* Anaemia due to hemorrhage within the tumor- occur occassionary
* Weight loss and fever
* Metastasis to the lungs- dyspnea, cough, shortness of breath and pain may be present

**Diagnosis**

History and physical examination

Signs of malignancy such as weight loss, enlarged liver and spleen, anemia and enlarged lymph nodes

Radiological studies, e.g. abdominal ultrasound, CT (computed tomography) and MRI (magnetic resonance imaging) of the abdomen

Complete blood count- increased RBC present if tumor is secreting excess erythropoietin

Staging of Wilm’s tumor

* Stage 1. Tumor is limited to kidney and completely resected
* Stage 2. Tumor extends beyond kidney but is completely resected
* Stage 3. Residual non hematogenous tumor to abdomen
* Stage 4.Hematogenous metastasis, deposits are beyond stage 3 namely to lung, liver, bone and brain
* Stage 5.Billateral tumor involvement is present at diagnosis

**Management**

Surgery and chemotherapy with or without radiation, is based on the clinical stage and histological pattern.

The most effective agents for treating Wilm’s tumour are actinomycin D and vincristine, doxorubin and cyclophosphamide

Nursing care include preparation for diagnostic and operative procedures

Explanation of treatment side effects

Child and family support

Explanations of chemotherapeutic reactions which may vary with specific drug regimen, the most side effects such as nausea and vomiting, body image changes e.g. alopecia and mucosal ulceration. Radiation effects may sometimes cause skin irritation and malaise

**Pre operative**

Surgery done within 24-48 hours of diagnosis

Prepare parents for surgery; prepare all laboratory and operative procedures

Observations, monitor blood pressure (due to excess production of renin BP may be high)

Do not palpate the tumor unless when necessary since by doing so may cause dissemination of cancer cells to adjacent and distant sites

Radiotherapy and chemotherapy are almost done immediately, parents should be explained the benefits

**Post operative**

Nurse like any other abdominal surgery case; observe bowel movement, bowel sounds, distension and vomiting

Evaluate blood pressure frequently and observe for signs of infection

Hygiene measures to prevent infection

Support the family

**Burkitt’s lymphoma**

Burkittt lymphoma is a form of non Hodgkin’s lymphoma in which cancer starts in immune cells called B-cells, recognized as the fastest growing human tumor. Burkitt lymphoma is associated with impaired immunity and is rapidly fatal if untreated, however intensive chemotherapy can achieve long term survival in more than half the people with Burkitt’s

**Etiology**

Burkitts lymphoma is common in young children who also have malaria and Epstein Barr virus, allowing it to change infected B cells into cancerous cells. About 98% of African cases are associated with Epstein Barr infection. Outside Africa Burkitts lymphoma is especially likely to develop in people infected with HIV, the virus that causes AIDS, however incidences have reduced markedly since the introduction of high antiretroviral therapy (HAART)

**Types**

The world health organization (WHO), has categorized three types of burkitts lymphoma namely

* Endemic (African); Endemic Burkitts lymphoma primarily affects African children ages 4 to7 and is twice as common in boys
* Sporadic (non-African) occurs worldwide, globally it accounts for up to 40% of paediatric cases
* Immunodeficiency- associated; this variant of burkitt’s lymphoma is most common in people with HIV/AIDS. It can also occur in people with congenital conditions that cause immune deficiency and in organ transplant patients who take immunosuppressive drugs

**Symptoms of Burkitts lymphoma**

The symptoms depend with the type

* The endemic (African) variant, usually starts as tumors of the jaw or other facial bones, it can also affect the gastrointestinal tract, ovaries and breast and can spread to the central nervous system, causing nerve damage, weakness and paralysis
* Sporadic and immunodeficiency-associated; usually start in the bowel and form a bulky tumor mass in the abdomen, often with massive involvement of liver , spleen and bone marrow. These variants can also start in the ovaries, testes, or other organs, and spread to the brain and spinal fluid

Other symptoms associated with burkitt’s lymphoma include

Loss of appetite, weight loss, fatigue, night sweets and unexplained fevers

**Diagnosis**

Because burkitt’s lymphoma spreads quickly, prompt diagnosis is essential

All or part of an enlarged lymph node or other suspicious disease, site will be biopsied and the sample examined under a microscope

Other tests include;

Computed tomography (CT) imaging of the abdomen, chest and pelvis

Chest x-ray

Bone marrow biopsy

Examination of the spinal fluid

Blood test to measure kidney and liver function

Testing for HIV disease

**Treatment**

Intensive intravenous chemotherapy which usually involves a hospital stay is the treatment for burkitt’s lymphoma. Because burkitts lymphoma can spread to the CSF and spinal cord, chemotherapy drugs also may be injected directly into the cerebrospinal fluid, a treatment known as intrathecal chemotherapy.

Examples of drugs used include the following; cyclophhosphamide, cytarabine, doxorubicin, etoposide, methotrexate, vincristine

Other treatments include Rituximab, a monoclonal antibody that sticks to proteins on cacer cells and stimulates the immune system to attack cancer cells

Autologus, stem cells transplantation, in which patients stem cells are removed and returned to the body

Radiation therapy

Steroid therapy

In some cases, surgery may be needed to remove parts of the intestine that are blocked, bleeding or have ruptured.

**Prognosis**

Burkitts lymphoma is fatal if left untreated in children; prompt intensive chemotherapy usually cures burkitts lymphoma, leading to prolong survival rates of 60% -90%

**Tetanus**

Def; Tetanus or lockjaw, is an acute, preventable disease caused by an exotoxin produced by the anaerobic, spore forming, gram-positive bacillus clostridium tetani. It is characterized by painful muscular rigidity primarily involving the masseter and neck muscles.

**Risk factors**

The development of tetanus has four main requirements;

* Presence of tetanus spores or vegetative forms of the bacillus
* Injury to the tissues
* Wound conditions that encourage multiplication of the organism
* A susceptible host

Tetanus spores are found in soli, dust, and the intestinal tract of humans and animals. In newborn, infection may occur through the umbilical cord. In situations in which the infants are delivered in contaminated surroundings and mother has not been properly immunized against tetanus.

**Incubation period**- 3 days to 3 weeks

**Pathophysiology**

When prevention efforts are not effective and conditions are favorable, the organisms multiply and form two exotoxins

Tetanospasmin, a potent toxin that affects the CNS to produce clinical manifestations of the disease

Tetanolysin, which appears to have no significance

The ideal conditions for growth of the organisms are devitalized (lack strength) tissues without access to air (e.g. puncture wound), wounds that have not been washed or kept clean and those that have crusted over, trapping pus inside. The exotoxin reaches the CNS by the way of either the neuron axons or the vascular system. The toxin becomes fixed on the nerve cells of the brain stem and the anterior horn of the spinal cord. The toxin acts at the neuromuscular junction to produce muscular stiffness to lower the threshold for reflex excitability

NB: Shorter incubation periods have been associated with heavily contaminated wounds, more severe disease and a worse prognosis.

Neonatal tetanus

**Clinical manifestations**

Progressive stiffness and tenderness of muscles in the neck and jaw

Difficulty in opening the mouth (trismus) which is caused is caused by sustained contraction of the jaw- closing muscles

Spasms of facial muscles produce the so called sardonic smile (risus sardonicus)

Progressive involvement of the trunk muscles produce causes opisthotonus and a board like rigidity of the abdominal and limb muscles

Patient has difficult in swallowing and is highly sensitive to external stimuli. The slightest voice, a gentle touch, or bright light triggers convulsive muscular contractions that last seconds to minutes

The contractions reoccur with increased rate until they become almost continuous. Mental state is not affected, patient is alert, but in pain and distress which is reflected in a rapid pulse, sweating and an anxious expression. Laryngospasm and tetany of respiratory muscles and accumulated secretions predispose the child to respiratory arrest and pneumonia. As the patient recovers from the disease the attacks become less frequent and gradually subside, complete recovery may take weeks

**Management**

Tetanus immunoglobulin to neutralize the toxins

Antibiotics- penicillin or erythromycin

Best treated at an intensive care facility, where close and constant observations and equipment for monitoring are available

Diazepam is the drug of choice for seizure control and muscle relaxation

Other muscle relaxants can be used e.g. baclofen

Neuromuscular blocking drugs g vecuronium- has paralytic effect on respiratory muscles, and so use of these drugs requires mechanical ventilation with endotracheal intubation

Administer analgesics

Maintain airway

Fluid and electrolyte balance and ensuring adequate fluid intake

Maintain a dark quiet room and minimize touch; however the lighting should be enough so as to observe the patient well.

Prevent complications associated with prolonged immobility, deceased bowel and bladder tone and subsequent constipation, anorexia, DVT, pneumonia and skin breakdown

Parents/patient need support, information and reassurance from the nurse

**Prevention**

Tetanus toxoid to all ANC mothers

Keep wounds clean/ receive T.T booster

During delivery minimize contamination during and after delivery and the umbilical stump, avoid applying local concoctions on the baby’s umbilical stump

Proper surgical debridement and cleaning of contaminated wounds to reduce the chance of infection

**Congenital Anomalies**

**Congenital Anomalies of GI tract**

**Cleft palate and cleft lip**

A cleft lip contains an opening in the upper lip that may extend into the nose. The opening may be on one side, both side or in the middle.

Cleft palate is a congenital split in the roof of the mouth.

Together, these birth defects are commonly called ‘orofacial clefts’

**Causes**

The causes include;

Problems with genes passed down from one or both parents, drugs, viruses or other toxins

**Pathophysiology**

The upper lip is derived from medial nasal and maxillary processes, failure of merging between the medial nasal and maxillary processes at five weeks’ gestation, on one or both sides, results to cleft lip.CL usually occurs at the junction between the central and lateral parts of the upper lip on either side.

Cleft palate is a partial or lack of fusion of palatial shelves, and it occurs in numerous ways

* Defective growth of palatal shelves
* Failure of the shelves to attain a horizontal position
* Lack of contact shelves
* Rupture after fusion of shelves

Fusion of palatial shelves begins at 8 weeks’ and continuous usually until 12 weeks’ gestation. One hypothesis is that a threshold is need beyond which delayed movement of palatial shelves does not allow closure to take place, and result in a cleft palate

**Etiology**

Genetic and environmental factor; in those instances, genetic factors create susceptibility for clefts. When environmental factors (i.e. triggers) interact with a genetically susceptible genotype, a cleft palate develops during an early stage of development.

**Images of cleft lip and cleft palate**

AB

CD

1. Lateral cleft lip B. Bilateral cleft lip C. Cleft palate D. Middle line lip

**Management**

Management require coordinated care provider in many fields of medicine and include the following; otolaryngology, dentistry, speech pathology, genetics, nursing, mental health and social medicine. Care is specialized and costly.

**Medical therapy**

**Neonatal care.**

Major concerns include;

* Risk of aspiration, because of the communication between oral and nasal cavities
* Airway obstruction
* Difficult with feeding of a child with a cleft and nasal regurgitation

Hints of feeding breast milk with a bottle are as follows;

Particularly for infants with cleft palate breastfeeding is not possible, and so the mother can express EBM

Various nipples and bottles are made specifically for infants with clefts; the goal is to find a nipple and bottle that makes feeding easy for the infant and still allow ample opportunity to suck.

A soft nipple is generally than a hard nipple, use a cross cut nipple to prevent choking; any nipple can be cross cut manually by using a single- edged razor blade; the cross cut is on the tongue side . The bottle should be squeezed and released, not continually squeezed

The nipple is angled to a side of the mouth, away from the cleft.

More upright or seated positions prevent the milk from leaking to the nose and causing the infant to choke

Advice the mother to stop feeding and allow the infant to cough or sneeze for a few seconds when nasal regurgitation occurs; palatal obturator may be used

Gaining weight and preventing aspiration and ear infections are the most important parts of caring for neonates with a cleft during their first days and weeks of life

**Surgery therapy**

Rule of 10s- weight 10 Ib, 10 g/dl of hemoglobin and 10 weeks of age however pediatricians are presently more flexible and some surgeons may well justify a neonatal lip closure, considering the rule 3 10s is still very useful

**Prevention**

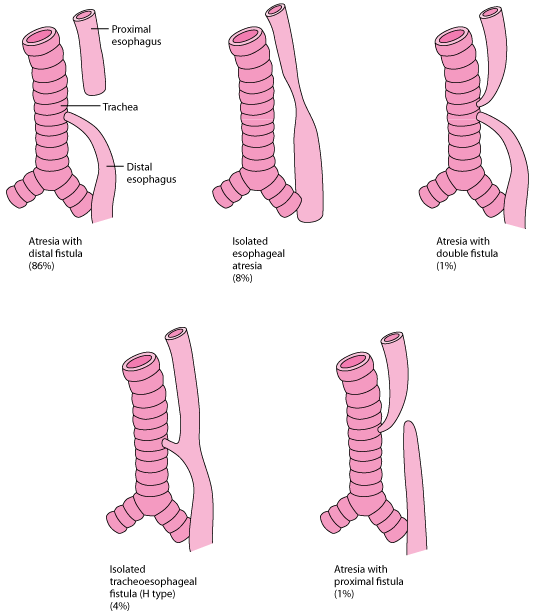
Preconception supplementation of folic acid and multivitamins especially in those situations which environmental factors represent a substantial part of the etiological background

**Esophageal atresia and Tracheoesophageal fistula.**

Esophageal atresiais when the esophagus terminates before it reaches the stomach, ending in a blind pouch while tracheoesophageal fistula Is a communication of esophagus and trachea.

**Pathophysiology**

Esophageal artesia or/and tracheosophageal fistula, results either from spontaneous posterior deviation of the tracheoesophageal septum or from mechanical factor pushing the dorsal wall of the foregut anteriorly. In its most forms the upper part of the esophagus end as a blind sac and the lower part of the esophagus end as a blind sac and the lower part is connected to the trachea by narrow canal just above the bifurcation. Atresia of the esophagus prevents normal passage of amniotic fluid into the intestinal tract, resulting in accumulation of excess fluid in the amniotic sac (polyhydraminos)



**Clinical manifestations**

* Choking, coughing and sneezing
* Infant swallows normally but begins to cough and struggle as the fluid returns through nose and mouth.
* Infant may become cyanotic due to lack of oxygen and may stop breathing as the overflow of fluid from the blind pouch is aspirated into trachea.
* Respiratory distress

**Diagnosis**

* Prenatal: Ultrasonography
* Postnatal: NGT or orogastric tube placement and x-ray

Routine prenatal ultrasonography may suggest esophageal atresia. Polyhydramnios may be present but is not diagnostic because it can occur with many other disorders.

After delivery, an NGT or an orogastric tube is inserted if esophageal atresia is suspected by prenatal ultrasonography or clinical findings; diagnosis of esophageal atresia is suggested by inability to pass the tube into the stomach. A radiopaque catheter determines the location of the atresia on x-ray.

**Treatment**

* Surgical repair

**Preoperative management** aims to get the infant into optimal condition for surgery and prevent aspiration pneumonia, which makes surgical correction more hazardous. Oral feedings are withheld. Continuous suction with an NGT in the upper esophageal pouch prevents aspiration of swallowed saliva. The infant should be positioned prone with the head elevated 30 to 40° and with the right side down to facilitate gastric emptying and minimize the risk of aspirating gastric acid through the fistula. If definitive repair must be deferred because of extreme prematurity, aspiration pneumonia, or other congenital malformations, a gastrostomy tube is placed to decompress the stomach. Suction through the gastrostomy tube then reduces the risk that gastric contents will reflux through the fistula into the tracheobronchial tree.

**Hypertrophic pyloric stenosis**

Also referred to as infantile hypertrophic pyloric stenosis, hypertrophic pyloric stenosis occurs when the circular muscle of the pyloric sphincter becomes thickened, resulting in elongation and narrowing of the pyloric canal, this results to outlet obstruction and compensatory dilatation, hypertrophy and hyperperistalsis of the stomach. This condition develops in the first few weeks of life causing projectile vomiting, dehydration, metabolic alkalosis and failure to thrive

**Epidemiology**

* Incidence: 1:300-500 live births.
* Male to female ratio = 4:1
* Classically presents in a 1st born male between 3-6 weeks of age.
* Causes: familial link

**Pathophysiology**

Caused by diffuse hypertrophy and hyperplasia of the smooth muscle of the anthrum of the stomach and pylorus

It is postulated that this abnormal innervation of the muscular layer leads to failure of relaxation of the pyloric muscle, increased synthesis of growth factors, and subsequent hypertrophy, hyperplasia, and obstruction.

**Clinical features**

* Non bilious vomiting
* Projectile vomiting increasingly
* Child has voracious appetite
* Dehydration due to vomiting
* Less flatus passage
* Fewer stool/constipation
* Hypochloremic, hypokalemic metabolic alkalosis
* Typical “olive” mass in RUQ on palpation
* Visible gastric (peristalsis) waves moving from left to right in upper abdomen
* **Failure to gain weight & lethargy**: most babies with this condition will fail to gain weight.
* **Hyperbilirubinemia:** Starvation can exacerbate diminished hepatic glucoronyl transferase activity, and indirect hyperbilirubinemia may be seen in 1-2% of affected infants.

**Diagnosis**

Initially suggested by the typical clinical presentation

**Physical examination** : The mass is firm, mobile, approximately 2 cm, best palpated from the left, located in the midepigastrium beneath the liver edge.

**Palpation** of the hard muscle mass or olive is diagnostic in conjunction with a typical history.

**Ultrasonography** is used to measure the thickness of the pyloric wall and the length of the pyloric canal. It is reliable and easily performed and has become the main investigation

**Upper GI barium studies** is cost-effective than ultrasound and can identify other possible diagnoses such as GERD.

**Serum electrolytes** (for correction of imbalances before surgical repair) there is often metabolic alkalosis with severe K+ depletion.

**Management**

Fluid resuscitation- Fluid therapy should be continued until the infant is rehydrated

Correct electrolyte imbalance - acid-base, and electrolyte losses.

Stop feeding and do a stomach wash out to reduce edema of stomach wall

Intravenous fluid therapy is begun with 0.45–0.9% saline, in 5–10% dextrose, with the addition of potassium chloride in concentrations.

Fluid therapy should be continued until the infant is rehydrated and the serum bicarbonate concentration is less than 30mEq/dL, which implies that the alkalosis has been corrected.

Most infants can be rehydrated within 24 hours

Surgery- **Fredet- Ramstedt pyloromyotomy**; In this procedure the thickened muscles of the pylorus are divided along the length of pylorus till the mucosa is seen.

After surgery, the baby can be fed after 18-24 hours with small quantity of clear fluids and increase gradually if patients tolerates.

Withhold feeds in case of vomiting.

**Common postoperative complications.**

* Vomiting due to incomplete myotomy
* Perforation of the pyloric mucosa
* Hemorrhage
* Wound infection.
* Surface mucous producing cell hyperplasia which causes persistent gastric outlet obstruction.

**Danger signs for pyloric stenosis post surgery**

* Persistent or projectile vomiting after feeding
* Poor weight gain or weight loss
* Decreased activity or lethargy
* Few or no stools over a period of 1 or 2 days
* Signs of dehydration such as decreased urination (more than 4-6 hours between wet diapers)

**Prognosis**

* Excellent unless diagnosis is delayed and prolonged severe dehydration occurs.
* Mortality is rare after pyloromyotomy.

**ABDOMINAL WALL DEFECTS**

**Omphalocele**

Omphalocele involves herniation of abdominal viscera through an enlarged umbilical ring. The viscera which may include liver small and large intestines, stomach, spleen or gall bladder are covered by amnion, Wharton’s jelly and peritoneum.

**Pathophysiology**

The origin of the defect is a failure of the bowel to return to the body cavity from its physiological herniation during the 6th to 10th weeks

**Epidemiology**

Omphalocele occurs in 2.5/10,000 births and is associated with a high mortality rate and severe malformations such as cardiac anomalies and neural tube defects. Approximately 15% of live-born infants with omphalocele have chromosomal abnormalities



Note the membrane covering the abdominal contents in this omphalocele

**Management**

* Vital signs
* Maintain body temperature
* Cover with saline-soaked gauze and trunk wrap circumferentially.
* Prophylactic antibiotics if ruptures
* Large defects ˃ 7 cm in diameter treat with desiccating substances e.g povidone-iodine, silver sulfadiazine e.t.c.
* Takes 2-3 months before re-epithelialization occurs.

**Gastroschisis**

Gastroschisis is the term applied to a protrusion of abdominal contents through the body wall. It occurs lateral to the umbilicus usually on the right, and the defect is most likely due to abnormal closure of the body wall. Viscera are not covered by peritoneum or amnion, and the bowel may be damaged by exposure to amniotic fluid. Size of defect much smaller (˂ 4 cm) than omphalocele



**Epidemiology**

Gastrochisis occurs in 1/10,000 births but is increasing in frequency, especially among young women (< 20 years old) and the reason for this increase is not known. Gastrochisis is not associated with chromosomal abnormalities or other severe defects

**Management**

* Need urgent surgical intervention
* Surgical closure
* Plastic spring-waded silo can be placed onto the bowel and secured beneath the fascia.
* The silo covers the bowel and allows for graduated reduction on a daily basis as the edema in the bowel wall decreases.

**PRENATAL MANAGEMENT**

* Maternal serum alpha-fetoprotein
* Maternal ultrasonograghy (look for other abnormalities)
* Counseling on prognosis
* Transfer to a centre where the newborn can be managed best.

**POSTNATAL MANAGEMENT**

GENERAL CARE.

* Manage in nursery in an incubator or under over head warmer.
* Pass nasogastric tube to decompress the stomach and prevent aspiration.
* Start fluid resuscitation and slightly higher volumes are required.
* Ventilatory support in case of respiratory distress.
* Urinary catheter to measure the urinary output.

NB:Omphalocele with an intact sac, cover and support it with sterile dry gauze dressing.

Omphalocele with a ruptured sac or gastroschisis, cover contents with wet sterile gauze dressing to prevent drying of the contents.

Nurse child on-side to prevent damage or vascular compromise due to drag on the vascular pedicle.

Cover lower body including defect in a sterile transparent plastic bag to prevent hypothermia and evaporative water loss while permitting inspection of the contents.

**Definitive treatment**

Operative treatment – aim of surgery is to replace the exposed viscera into the abdominal cavity and provide cover with the abdominal wall.

Only skin may be closed creating a ventral hernia, or a silo made of artificial material may be put, which is reduced later for closure of the abdomen.

**Non operative treatment**

Carried out when severe congenital malformations preclude definitive surgery or omphalocele too large for the surgical procedure to be successful.

Paint the sac with mercurochrome or silver nitrate to promote epithelization and formation of ventral hernia which can be repaired later.

**Hirschsprung’s disease**

Hirschsprung’s disease also known as congenital megacolon is due to an absence of parasymphathetic ganglia in the bowel wall (agangionic megacolon), together with hypertrophy of the nerve trunks.

Results from failure of neuroblasts into the gut from vagal nerve trunks at the end of the 1st trimester of fetal life

HD is characterized by the absence of myenteric and submucosal ganglion cells (Auerbach and Meissner plexuses) along a variable length of the distal gastrointestinal tract.

The disease results in decreased motility in the affected bowel segment, lack of propagation of peristaltic waves into the aganglionic colon, and abnormal or absent relaxation of this segment and of the internal anal sphincter.

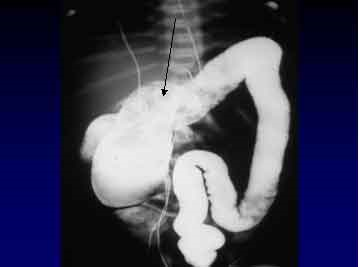
2/3 of patients, the rectum and lower sigmoid colon are involved.

Absence of ganglion cells gives rise to a contracted non-peristaltic segment with a dilated hypertrophied segment of normal colon above it, due to absence of the nerve supply in the distal part of the colon.

Due to the absence of the ganglion cells in the affected gut, peristaltic waves are deficient and the proximal large intestine becomes dilated and distended.

**Epidemiology.**

* Occurs in about 1:4500 live births.
* Shows a familial tendency
* More common in males than in females
* Associated with Down syndrome in 10% of patients.



Classically the bowel just before the segment with Hirschsprung's disease becomes distended and enlarged. This distended bowel has ganglion cells. The characteristic problem in the bowel with Hirschsprung's is the absence of ganglion cells.

Figure: A barium enema showing a “long segment” Hirschsprung’s disease. The transition zone is in the transverse colon. The arrow demonstrates the area of the transition zone between the enlarged area which has ganglion cells (normal) and the small area which does not (Hirschsprung’s disease).

**Pathophysiology**

The aganglionic segment almost always includes the rectum and proximal portion of the large intestine.

Lack of innervation produces the functional defect that results in absence of propulsive movements (peristalsis) that causes accumulation of intestinal contents and distension of the bowel proximal to the defect (megacolon).

Failure of the internal anal sphincter to relax contributes to the clinical manifestation of obstruction because it prevents evacuation of solids, liquids and gas.

Intestinal distention and ischemia may occur as result of distention of the bowel wall, which contributes to the development of enterocolitis, the leading cause of death in children with Hirschsprung disease.

**Clinical manifestations**

In Newborns presents as delayed passage of meconium or acute intestinal obstruction with or without infection i.e. enterocolitis

Neonate fails to pass meconium for more than 48 hours and develops distension of abdomen and bilious vomiting.

In old age, the child may present as chronic constipation and abdominal distension.

Failure to thrive and the child looks malnourished.

O/E, the abdomen is distended with visible loops or palpable fecaloma, severe constipation

Rectal examination shows empty rectum and withdrawal of fingers results in passage of stool.

Delayed passage of meconium (>24 h after birth)

Abdominal distension that is relieved by rectal stimulation or enemas

Vomiting

Neonatal enterocolitis

**Older children and adult symptoms are as follows:**

Chronic abdominal distension

Classification;

* Patients can be classified by the extension of the aganglionosis, as follows:
* Classic short-segment HD (75% of cases) - Aganglionic segment does not extend beyond the upper sigmoid
* Long-segment HD (20% of cases)
* Total colonic aganglionosis (3-12% of cases)

Investigations

* Plain X-ray of the abdomen reveal air fluid levels in cases presenting with intestinal obstruction.
* Rectal contrast studies (barium enema) demonstrate the transition zone in the recto sigmoid colon with retained barium even after 24 hours.
* Rectal biopsy is taken from the rectum to confirm the diagnosis.
* Arorectal pressure measurement is very helpful in diagnosis.

**Treatment**

Includes rehydration, systemic antibiotics, nasogastric decompression and rectal irrigation while diagnosis is being confirmed

Depends on age of patient, length of involved segment, severity of symptoms and presence of enterocolitis

With presenting enterocolitis, an initial colostomy is performed low in the ganglionated segment.

Child with constipation alone, dilated intestine can be evacuated with repeat rectal saline washouts and enema as a first step.

In neonates definitive operation is delayed until the weighs 10kgs (10 months – 1 year)

Aims at removal of aganglionic segment of bowel and restoring the continuity of the gut.

Done in 2 or 3 stages.

Nursing considerations

Record the time of first passage of meconium.

If diagnostic barium enema is planned, it is essential to avoid preparatory rectal wash. This distorts the anatomy of the rectosigmoid region and transition zone may be missed.

Daily rectal washes should be given to the child if primary pull through is planned or there is delay in the colostomy.

Rectal wash is done with normal saline.

Antibiotics and fluid should be given as per instructions.

Rectal manipulations are kept to a minimum as these may cause rectal injury.

**Anal rectal malformations**

These malformations may range from simple imperforated anus to include other associated complex anomalies of GU and pelvic organs

**Classifications**

**Male**

Perineal fistula

Rectal urethral bulbar fistula

Rectal urethral prostatic fistula

Rectal vesicular (bladder neck) fistula

Imperforate bladder without fistula

Rectal atresia and stenosis

**Female**

Perineal fistula

Vestibular fistula

Imperforate anus without fistula

Rectal atresia stenosis

Cloaca

Rectal atresia occur when the anal opening appears normal, there is a midline intergluteal groove and usually no fistula exist between the rectum and urinary tract.

Rectal atresia is a complete obstruction (inability to pass stool) and requires immediate surgical intervention

Rectal stenosis presents later in infancy when the infant has a history of difficulty stooling, abdominal distension, and ribbon like stools

A persistent cloaca is a complex anorectal malformation in which the rectum, vagina and urethra drain into a common channel opening into the opening

Imperforate anus includes several forms of malformation without obvious opening

**Pathophsiology**

During embryonic development the cloaca (end of the digestive tract) becomes the common channel for the developing urinary, genital and rectal systems. The cloaca is divided at the six week of gestation into an anterior urogenital sinus and a posterior intestinal channel by the urorectal septum. After the lateral folds joins the urorectal septum, separation of the urinary and rectal segment takes place. Further differentiation results in the anterior GU system and the posterior and rectal channel.An interruption of this development leads to incomplete migration of the rectum to its normal perineal postion

**Diagnosis**

Physical finding of an absent anal opening, abdominal distension, vomiting, absence of meconium passage or presence of meconium in urine

A flat perineum with the absence of a midline intergluteal groove

In anal fistula a prominent anal dimple and a band of skin tissue is seen commonly called a ‘bucket handle’

Abdominal and pelvic ultrasonograpy

An IV pyelogram and voiding cystourethrogram

Pelvic MRI, radiograpy, ultrasound and fluoroscopic examination of the pelvis

**Management**

Surgery

Stabilise the newborn and keep NPO

Give IVF’s

Nursing

Responsibility is assisting in identification of anal rectal malformations. A newborn that does not pass stools in the first 24 hours nor has meconium that appears at a location other than the anal opening requires further assessment

Family support depending on the operation carried out and advice on discharge about home care of the baby which includes bowel irrigation programs, toilet training, stool softeners use and diet.

**Congenital Neurologic Anomalies**

**Hydrocephalus**

Hydrocephalus is accumulation of excessive amounts of CSF, causing cerebral ventricular enlargement and/or increased intracranial pressure.

**Causes**

Congenital (myelomeningocele, intrauterine viral infection- cytomegalovirus, toxoplasmosis)

Aqueduct stenosis

Acquired conditions such as intraventricular hemorrhage, tumor, CSF infections or head injury

**Pathophysiology**

CSF is circulates throughout the ventricular system and is then absorbed within the arachnoid spaces by a mechanism that is not entirely clear.1. Impaired absorption of CSF fluid within the subarachnoid space, damage of the subarachnoid cisterns or malfunction of arachnoid villi leads to nonobstructive or communicating hydrocephalus. 2. Obstruction of the flow of CSF through the ventricular system leads to obstructive or noncommunicating hrocephalous. Myelomeningocele is characterized by herniation of a small cerebellum, medulla,pons and fourth ventrical into the cervical canal through an enlarged foramen magnum, and the resulting obstruction cause hyndrocephalous

**Classification**

* Communicating hydrocephalus (non obstructive)

Caused by impaired CSF reabsorption within the subarachnoid space .There is no obstruction of CSF between ventricles and subarachnoid space.

* Non communicating (obstructive) Caused by CSF flow obstruction in ventricles.
* Congenital
* Acquired: Due to CNS infections like meningitis, brain tumors, head trauma, intracranial hemorrhage.

**Symptoms and Signs**

Neurologic findings depend on whether intracranial pressure is increased, symptoms of which in infants include irritability, high-pitched cry, vomiting, lethargy, strabismus, and bulging fontanelle. Older, verbal children may complain of headache, decreased vision, or both. Papilloedema is a late sign of increased intracranial pressure; its initial absence does not exclude hydrocephalus.

**Diagnosis**

* Prenatal ultrasonography
* Neonates: Cranial ultrasonography
* Older infants and children: CT or MRI
* After birth, diagnosis is suspected if routine examination reveals an increased head circumference; infants may have a bulging fontanelle or widely separated cranial sutures.

**Treatment**

* Sometimes observation or serial lumbar punctures
* For severe cases, a ventricular shunt procedure

Progressive hydrocephalus usually requires a ventricular shunt. Shunts typically connect the right lateral ventricle to the peritoneal cavity

**Shunt complications**

* Infection
* Malfunction

**Key Points**

* Hydrocephalus is usually caused by obstruction to the normal flow of CSF but can be due to impaired resorption of CSF.
* If the disorder occurs before the cranial sutures have fused, the head may be enlarged, with bulging fontanelles.
* Neurologic symptoms develop mainly if intracranial pressure increases; infants may have irritability, high-pitched cry, vomiting, lethargy, and strabismus.
* Diagnose using ultrasonography prenatally and in neonates; use MRI or CT for older children.
* Treat with observation or serial lumbar punctures or a ventricular shunt procedure depending on the etiology and severity and progression of symptoms.

**Spinal Bifida**

Spinal bifida is defective closure of the vertebral column.

**Causes**

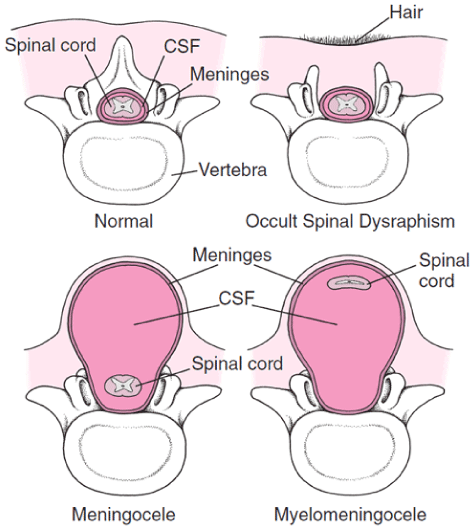
Although the cause is not known, low folate levels during pregnancy increase risk. There seems to be a genetic component. Other risk factors include maternal use of certain drugs (eg, valproate) and maternal diabetes.

**Types**

-Spinal bifida occulta which refers to a defect that is not visible externally

-Spinal bifida cystic which refers to a visible defect with an external sac like and the two major forms include;

* Meningocele which encases meninges and spinal fluid, but no neural elements
* Myelomeningocele(or meningomyelocele), which contains meninges, spinal fluid and nerves



**Pathophysiology**

Primarily defect in neural tube malformations is a failure of neural tubal closure; however some evidence indicates that the defects are as a result of splitting of the already closed neural tube as a result of abnormal increase in CSF pressure during the first trimester

**Symptoms and Signs**

Many children with minor defects are asymptomatic.

**Neurologic:** Paralysis and sensory deficits are present below the lesion. Rectal tone is usually decreased.

Hydrocephalous

**Orthopedic:** Lack of muscle innervation leads to atrophy of the legs.

**Urologic:** Paralysis also impairs bladder function, occasionally leading to a neurogenic bladder and, consequently, urinary reflux, which can cause hydronephrosis, frequent UTIs, and, ultimately, kidney damage.

**Diagnosis**

* Ultrasonography or MRI
* Prenatal screening can be done by doing fetal ultrasonography and by measuring maternal serum levels of α-fetoprotein

**Treatment**

* Surgical repair of the spinal lesion
* Sometimes a ventricular shunt
* Various measures for orthopedic and urologic complications

**Prevention**

* Folate supplementation

**Disorders of sex development**

**Ambiguous genitalia**

Ambiguous genitalia are birth defect where the outer genitals don’t have the typical appearance of either a boy or girl.

**Etiology**

Abnormalities of chromosome, defects of embryogenesis, or biochemical (hormonal) abnormalities, disturbances in any of these leads to abnormal development resulting to ambiguous genitalia

**Pathophysiology**

**Stages of normal development**

For the first six weeks of life the developing embryo is morphologically asexual, neither male nor female. The primitive bipotential( abli to form testicle or an ovary) gonad consist of an outer layer (the cortex) and the inner medulla.

Differentiation into testes or ovaries takes place during the seventh and the eighth weeks of gestation. At this time the medullary portion develops and the cortical zone regresses. In female the cortical zone is preserved while the medullary regresses

Active factors cause the mullerian duct system to regress and without these factors the primitive gonad has a tendency to feminize. The embryonic ovary develops in the absence of male hormone stimulation.

The final stage of genital and reproductive organ development is differentiation of the external genitalia, which in the early consist of urogental sinus, two lateral scrotal swelling and an anteriorly situated genital tubercle. Depending on the presence or absence of male hormones the genital tubercle differentiates into a penis or a clitoris. In response to testicular androgens, labosacral folds fuse to form a scrotum ant the ventral skin of the penis;the urethral fold form the perineal and penile urerthra, without the influence of masculinizing secretions, the urethral folds do not fuse and instead become labia minora, the labia sacral folds remain fused to separate into labia majora and the urogenital sinus differentiates into a lower vagina and the vagina and urethral openings.

**Abnormal genitalia and reproductive organ development**

Disturbances of normal order of events in gender determination produce abnormal genitalia and reproductive organ development with the presence of ambiguous or indeterminate external genitalia at birth. Ambiguous genitalia can be variable and can conform to one gender or other. In some, external sexual structures represent those of normal male or female, where as the karyotype is the direct opposite. situation in which the phenotypic gender differs from chromosome gender is a disorder of sex development. A failure or abnormality in any of the four steps of genital and reproductive organ development can lead to abnormal development in subsequent stages. The mechanisms and sites of defective development include:

* Abnormal gender determination- chromosome abnormalities result in disturbance of secondary sexual characteristics and reproductive organ development.
* Abnormal differentiation of gonads- when induction of the bipotential gonad fails, gender differentiation proceeds in the direction of the female phenotype, regardless of the karyotype.
* Abnormal differentiation of ductal systems-Biological inactivity of androgenic male organizer substances or insensitivity of ductal tissue to the action of these substances, result in a persistent female duct system, which leads to the presence of uterus and uterine tubes
* Abnormal secretion of or tissue insensitivity to testicular androgen- complete failure of male hormones secretion produces female external genitalia in a genetic male.. Partial or incomplete failure results in incomplete masculinization with ambiguity of the external genitalia. The female genital fetus exposed into large amounts of andrenogenic hormone may exhibit varying degrees of masculinization of the external genitalia (congenital andrenal hyperplasia)

**Types of abnormalities**

Some disorders with abnormal genital development are not characterized by ambiguous genitalia in the newborn period. e.g. the most common chromosome disorders do not become apparent until later in childhood, adolescence and adulthood when the individual seeks medical attention because of delayed development and infertility

The four conditions of producing ambiguous genitalia in the newborn that require prompt and accurate evaluation are the:

* The masculinized female
* The incompletely masculinized male
* The presence of both male and female sex organs
* Mixed gonad dysgenisis

An ambinguous genitalia in the newborn is often as a result of the virilization in the female by adrenal androgens after the time of early gonadal differentiation. The most common type is **congenital adrenogenital hpyeplasia,** which isan inherited deficiency of adrenal corticoid hormones. Due to decreased cortisol the pituitary gland is stimulated and produces ACTH which causes the adrenal cortex to increase the production of adrenal hormones, including the androgens, because the adrenal gland differentiates later than the gonadal duct system, but before differentiation of the external genitalia, masculization of the external genitalia is the prominent feature. The internal female anatomy is normal.

The external genetalia in the incompletely masculinized male may be incompletely male, ambiguous or completely female. Defects may be as a result of deficient production of fetal androgen, deficiency in any of the enzymes needed testosterone biosynthesis, or unresponsiveness or subresponsiveness of genital structures to testosterone. Individuals who may be either genetic males or females with both ovarian and testicular tissues with an ovary on one side and a testis on the other or a combination of ovotestis, are rare. The external genitalia may be a male (possibly cryptorchid with a micro penis) or normal female, but are ambiguous in the majority of cases

Mixed gonadol dysgenesis, in which the affected infants are sex chromosome mosaics (an individual with varying cell composition). Genitalia vary greatly, but in those who appear predominantly, the dysplastic testis may cause masculization at puberty.

**Diagnosis**

History- previous miscarriages (may help indentify chromosome which deviated from normal) maternal ingestion of steroids, relatives with disorders of sex development or unplanned death in the first weeks of live, maternal ovarian tumor in pregnancy

Physical examination- presence of gonads strongly suggestive a male genotype, uterus palpable by rectal examination, length of penis stretched to measure location of urethral orifice, location of vagina orifice. Prader staging to determine degree of virilization of external genitalia

Chromosome analysis- chromosome abnormalities and precise genetic picture of a person chromosome (karyotype) DNA analysis

Endoscopic, ultrasonograpy and radiographic contrast media studies, presence or absence or nature of internal genital and urinary structures

Biochemical test- include urinary steroid secretions to help detect several of the andrenocortical syndromes (congenital adrenogenital hyperplasia)

**Clinical manifestations**

**Ambiguous findings**

Micro penis (2.5-3 cm) in newborn, maybe enlarged clitoris

Urethral meatus anywhere along dorsal or ventral surface of penis, especially on perineum

Small scrotum with smooth, tight skin and any degree of separation in midline may be enlarged labia

Absent testes, may be undescended, if combined with small scrotum, may be evidence of enlarged labia

Enlarged clitoris that protrudes from labia, may suggest small penis

Urethral meatus located in clitoris, may suggest small penis

Prominent labia, partially or completely fused with palpable masses on each side, may be small scrotum with testis

**Management**

The overall goal of management is to enable the affected child to grow into a well-adjusted, psychologically stable person, who is able to identify with the assigned gender and is content with the same

Gender identity should be established by two and half years

Female s with congenital adrenogenital hyperplasia or virilization are treated with steroids and surgical intervention

Males with hpospadias and crytorchidism (under virilization) may be successfully reared males and surgical repair done

The child with mixed gonadal dysgenesis may be assigned gender on phallus and androgen exposure

Male infants with micro penis may respond to testosterone and be successfully reared as boys

Nursing

Family needs a great deal of support, and encouragement from nurses, and the health care team to cope with the emotionally charged situation, parents are confused, anxious, and overwhelmed by feeling of guilt and shame.

**Assignment**

* **Epispadias**
* **Hypospadias**
* **Phimosis and paraphimosis**
* **Cryptorchidism (Undescended Testes)**

**Convulsive disorders**

A seizure is an abnormal, unregulated electrical discharge that occurs within the brain’s cortical gray matter and transiently interrupts normal brain function.

**Etiology**

Common causes of seizures vary by age of onset:

* Before age 2: Fever, birth or developmental defects, birth injuries, and metabolic disorders
* Ages 2 to 14: Idiopathic seizure disorders
* Adults: Cerebral trauma, alcohol withdrawal, tumors, strokes, and an unknown cause (in 50%)
* The elderly: Tumors and strokes

**Classification**

Seizures are classified as **generalized** or **partial**.

**1. Generalized**

In generalized seizures, the abnormal electrical discharge diffusely involves the entire cortex of both hemispheres from the onset, and consciousness is usually lost.

Generalized seizures include the following:

* Infantile spasms
* Absence seizures
* Tonic-clonic seizures
* Tonic seizures
* Atonic seizures
* Myoclonic seizures (eg, in juvenile myoclonic epilepsy)

**2. Partial seizures**

In partial seizures, the excess neuronal discharge occurs in one cerebral cortex, and most often results from structural abnormalities.

Partial seizures may evolve into a generalized seizure (called secondary generalization), which causes loss of consciousness. Secondary generalization occurs when a partial seizure spreads and activates the entire cerebrum bilaterally.

Partial seizures- sub divided into three types:

* Simple partial seizures
* Complex partial seizures
* Simple or complex seizures secondarily generalized

**Signs and symptoms**

**1. Generalized seizures**

The seizure occurs without warning and consists of two distinct phases: **Tonic and clonic**;

In **tonic** phase the person rolls the eyes upward and immediately loses consciousness. If standing the child falls to the ground. The muscles stiffen in a generalized symmetric tonic contraction of the entire body. The arms usually flex, and the legs, head and neck extend and the mouth is shut tongue may be bitten, and this tonic phase may last for 10 to 30 seconds. The BP and the heart rate are increased and flushing and increased salivation

In **clonic** phase the tonic rigidity is replaced by intense jerking movements as the trunk and extremities undergo rhythmic contraction and relaxation, the child cannot control secretions at this time and may be incontinent of urine and feces. The average clonic phase lasts 30 to 50 seconds

In post ictal phase the child may remain semiconscious and difficult to arouse. The average duration ot the postictal phase is from 1 to 15 minutes

**Absence seizures**- They have a sudden onset and are characterized by a brief loss of consciousness, a blank stare and automatisms

**Atonic seizures**- are manifested as a sudden, momentary loss of muscle tone

**Myclonic seizures**- are characterized by sudden, brief contractions of a muscle or group of muscles

**2. Partial seizures**

* **Simple partial seizures:** simple symptoms and no alteration of consciousness
* **Complex partial seizures:** complex symptoms and impairment of conscious
* **Simple or complex seizures secondarily generalized**: simple or complex partial seizures that evolve into generalized seizures, usually a tonic-clonic event.

Other terms used in convulsive disorders include;

* **Epilepsy:** Epilepsy is a condition characterized by two or more unprovoked seizures and can be caused by a variety of pathologic process in the brain
* **Status epilepticus**: is a continuous seizure that last more than 30 minutes or a series of seizures from which the child does not regain a premobid level of consciousness
* **Febrile Seizures:** Febrile seizures are diagnosed in children < 6 yr with body temperature > 38°C and no previous afebrile seizures when no cause can be identified and no underlying developmental or neurologic problem exists.

**Diagnosis**

* Clinical evaluation
* For new-onset seizures, neuroimaging, laboratory testing, and usually EEG
* For known seizure disorders, usually anticonvulsant levels
* For new-onset or known seizure disorders, other testing as clinically indicated

**Management**

* Elimination of the cause if possible
* Avoidance of or precautions during situations when loss of consciousness could be life threatening
* Drugs to control seizures, which include diazepam, pnenobarbitone, phenytoin sdium valporate
* Surgery if ≥ 2 drugs in therapeutic doses do not control seizures

**Acute seizures and status epilepticus**

* Most seizures stop spontaneously in several minutes or less and do not require emergency drug treatment. However, status epilepticus and most seizures lasting > 5 min require drugs to terminate the seizures, with monitoring of respiratory status. Endotracheal intubation is necessary if there is any indication of airway compromise.
* The sooner anticonvulsant therapy is started; the better and the more easily seizures are controlled.

**Poisoning in children**

Poisoning can be accidental in young children or suicidal in older children.

**General principles in management of poisoning**

**Treatment**

Supportive

Activated charcoal for serious oral poisonings

Occasionally use of specific antidotes

Only rare use gastric lavage

**Initial stabilization**

Maintain airway, breathing and circulation

IV fluids and sometimes vasopressors

IV dextrose to children with altered conscious level

IV naloxone should be tried on patients with apnea

IV thiamine should be given with or before to people suspected of thiamine deficiency e.g alcoholics, undernourished patients

IV fluids for hypotension

**Topical decontamination**

Any body surface (including the eyes) exposed to the toxin is flushed with large amounts of water or saline, contaminated clothing, including shoes, and even jewelry should be removed

**Activated charcoal**

Gastric emptying- should not be done routinely since it does reduce the overall morbidity and mortality

Whole bowel irrigation- can be used in some serious poisoning like heavy metals and drugs packets e.g. heroin and cocaine

Dialysis e.g. salicylate poisoning, theophylline

Specific antidotes

Supportive measures for hypoglycaemia, coma, and cerebral oedema

Seizures- anticonvulsant

Hyperthermia- cooling measures

**Prevention**

Clearly labeling house hold products and prescription drugs

Storing drugs and toxins in cabinets that are locked and inaccessible to children to children

Promptly disposing of expired drugs

Use of carbon monoxide detectors

Use safety caps and containers

**Acetaminophen poisoning (paracetamol)**

Acetaminophen poisoning can cause gastroenteritis within hours and hepatotoxicity 1 to 3 days after ingestion. Severity of hepatotoxicity after a single acute overdose is predicted by serum acetaminophen levels.

**Symptoms and Signs**

Anorexia, nausea, vomiting and right upper quadrant abdominal pain, renal failure and pancreatitis may occur.

Later –Hepatic involvement causes:

* Pain
* Jaundice
* Confusion
* Stupor
* Coagulation abnormalities

**Diagnosis**

* Serum acetaminophen levels
* Rumack-Matthew nomogram

**Treatment**

* Oral or IV *N*-acetylcysteine to prevent or minimize hepatotoxicity.
* Possibly activated charcoal

Activated charcoal may be given if acetaminophen is likely to still remain in the GI tract.

**Carbon Monoxide Poisoning**

Carbon monoxide (CO) poisoning causes acute symptoms such as headache, nausea, weakness, angina, dyspnea, loss of consciousness, seizures, and coma. Neuropsychiatric symptoms may develop weeks later. Diagnosis is by carboxyhemoglobin levels and ABGs, including measured O2 saturation. Treatment is with supplemental O2. Prevention is often possible with household CO detectors.

**Aspirin and Other Salicylate Poisoning**

After ingestion, acetylsalicylic acid (ASA) is converted to salicylic acid, its active form. Salicylic acid is absorbed in stomach and small intestines . Salicylate poisoning can cause vomiting, tinnitus, confusion, hyperthermia, respiratory alkalosis, metabolic acidosis, and multiple organ failure. Diagnosis is clinical, supplemented by measurement of the anion gap, ABGs, and serum salicylate levels. Treatment is with activated charcoal and alkaline diuresis or hemodialysis.

**Organophosphate Poisoning and Carbamate Poisoning**

Organophosphates and carbamates are common insecticides that inhibit cholinesterase activity, causing acute muscarinic manifestations (eg, salivation, lacrimation, urination, diarrhea, emesis, bronchorrhea, bronchospasm, bradycardia, miosis) and some nicotinic symptoms, including muscle fasciculations and weakness. Neuropathy can develop days to weeks after exposure.

**Pathophysiology**

Organophosphates and carbamates are absorbed through the GI tract, lungs, and skin. They inhibit plasma and RBC cholinesterase, preventing breakdown of acetylcholine, which then accumulates in synapses. Carbamates are cleared spontaneously within about 48 h after exposure. Organophosphates, however, can irreversibly bind to cholinesterase.

**Diagnosis** is clinical and sometimes with a trial of atropine, measurement of RBC acetylcholinesterase level, or both.

**Treatment**

Bronchorrhea and bronchospasm are treated with titrated high-dose atropine. Neuromuscular toxicity is treated with IV pralidoxime.

**Key Points**

* Organophosphates have been used in insecticides, medical treatments, and biologic weapons.
* Suspect toxicity if patients have a muscarinic cholinergic toxidrome with prominent respiratory and neuromuscular findings.
* Confirm the diagnosis by the response to atropine and sometimes RBC cholinesterase levels.
* Treat supportively by giving atropine to relieve bronchospasm and bronchorrhea and by giving 2-PAM to relieve neuromuscular symptoms.

**Hydrocarbons**

Examples of hydrocarbons include;

* Gasoline
* Kerosene
* Turpentine
* Paint thinner and remover

**Clinical manifestations**

* Gagging, choking and coughing
* Nausea
* Lethargy
* Weakness
* Respiratory symptoms of pulmonary involvement
* Tacypnoea
* Cyanosis,
* Retractions
* Grunting

Immediate danger is aspiration (even small amounts) can cause bronchitis and chemical pneumonia; Gasoline, kerosene, lighter fluid and turpentine cause severe pneumonia.

**Treatment**

Inducing emesis is generally contraindicated. Gastric decontamination are questionable even when the hydrocarbons contain a heavy metal or pesticide, if gastric lavage must be performed, a cuffed endotracheal tube should be in place before lavage because of a high risk of aspiration. Symptomatic treatment of chemical pneumonia includes high humidity oxygen, hydration and antibiotics for secondary infections.

**Lead Poisoning**

**(Plumbism)**

Lead poisoning often causes minimal symptoms at first but can cause acute encephalopathy or irreversible organ damage, commonly resulting in cognitive deficits in children.

**Diagnosis** is by whole blood lead level.

**Treatment**

Treatment involves stopping lead exposure and sometimes using chelation therapy.

**Paediatric HIV**

HIV stands for human immunodeficiency virus

AIDS stands for acquired immunodeficiency syndrome

**WHO Clinical Staging of HIV Infection in Infants and Children**

Stage I

• Asymptomatic

• Persistent generalized lymphadenopathy (PGL)

• Unexplained, asymptomatic Hepatosplenomegaly

Stage II

• Papular pruritic eruptions (PPE)

• Seborrheic dermatitis

• Fungal nail infections

• Angular cheilitis

• Linear gingival erythema

• Extensive HPV or molluscum infection (>5% of body

area/face)

• Recurrent oral ulcerations (>2 episodes/ in 6 months)

• Parotid enlargement

• Herpes zoster (>1 episode/12 months)

• Recurrent or chronic upper respiratory infection (URI):

otitis media, otorrhoea, sinusitis (>2 episodes/6 months)

Stage III

• Unexplained moderate malnutrition (-2SD or Z score) not responding to standard therapy

• Unexplained persistent diarrhoea (>14 days)

• Unexplained persistent fever (intermittent or constant, > 1 mo.)

• Oral candidiasis (outside neonatal period)

• Oral hairy Leucoplakia

• Pulmonary tuberculosis

• Severe recurrent presumed bacterial pneumonia (>2 episodes/12 months)

• Acute necrotizing ulcerative gingivitis/ periodontitis

• Lymphoid interstitial pneumonitis (LIP)

• Unexplained anaemia (<8g/dL), neutropenia (<1000/mm3), or thrombocytopenia (<30,000/mm3) for >1 mo.

• HIV-related cardiomyopathy

• HIV-related nephropathy

Stage IV

• Unexplained severe wasting or severe malnutrition (-3 SD or Z score) not responding to standard therapy

• Pneumocystis pneumonia

• Recurrent severe bacterial infections (>2 episodes/12 months, excluding pneumonia)

• Chronic orolabial or cutaneous HSV (lasting > 1 mo)

• Extra-pulmonary tuberculosis

• Kaposi’s sarcoma

• Oesophageal candidiasis

• CNS toxoplasmosis

• Cryptococcal meningitis

• Any disseminated endemic mycosis

• Cryptosporidiosis or Isosporiasis (with diarrhoea > 1 month)

• CMV infection of organ other than liver, spleen, lymph nodes (and onset age >1 month)

• Disseminated mycobacterial disease other than tuberculosis

• Candida of trachea, bronchi or lungs

• Acquired recto-vesicular fistula

• Cerebral or B-cell non-Hodgkin’s lymphoma

• Progressive multifocal leucoencephalopathy (PML)

• HIV encephalopathy

**Diagnosis**

**Birth Testing and Early Infant Diagnosis**

**Birth Testing**

Birth testing is defined as HIV testing (with DNA PCR) at birth or first contact within 2 weeks after birth, for infants born to known HIV-positive mothers.

**HIV DNA PCR testing should be done at birth or first contact thereafter within two weeks of birth for infants born to HIV positive mothers**

Birth testing has the potential to greatly improve survival for infants who are infected during pregnancy and around labour and delivery by identifying them early for rapid ART initiation.

For birth testing to be effective, facilities and programmes must provide the necessary resources, capacity, and infrastructure for optimum function of EID (early infant diagnosis) with a turn-around-time of ≤ 7 days for infant DBS PCR results.

Where capacity is limited to provide birth testing for all new-borns, it should be prioritized for the following infants at highest risk of antenatal HIV infection:

• Mother has been on ART for < 6 months

• Mother’s most recent viral load (VL) before delivery was ≥ 1,000 copies/mL

• No maternal VL available within the past 6 months

• Mother known or suspected to be failing current ART regimen

• Pre-term infants

• Low birth-weight infants

A new-born with a negative HIV DNA PCR at birth (or within 2 weeks of birth) should continue infant ARV prophylaxis and be followed as an HEI (HIV exposed infant) . A repeat HIV DNA PCR test should be performed at 6 weeks and subsequent testing as recommended for all HEIs, following the EID (early infant diagnosis) algorithm

A new-born with a positive HIV DNA PCR is presumed to be HIV positive and should be started on ART immediately. A confirmatory HIV DNA PCR and baseline viral load should be taken at the time of initiating ART (initiation of ART is based on the first result). A positive 2nd PCR test confirms HIV infection; continue ART and routine follow-up as for HIV-positive infants. If the 2nd PCR is negative, these are discordant results: the infant should continue ART and send a 3rdDBS sample to NHRL (National HIV referral laboratory) to confirm HIV status.

**Age Counseling Approach**

< 6 years old - The counseling sessions will focus on engaging all of the child’s caregivers

6-12 years old

Both the caregiver and the child will be involved. The counseling will focus on the caregiver; younger children can be given a paper and pen and asked to draw their family, school, etc, and talk about their experiences. Disclosure of HIV status to the child should commence by 5 years of age and be completed by 10-12 years of age

**Package of HIV Testing Services**

An HIV testing and counselling session consists of:

• A pre-test session

• HIV test

• A post-test session

• Assessment for other health-related conditions or needs

• Referral and linkage to other appropriate health services

**Summary of HIV Testing Services Package**

**Pre-test counselling/Pre-test information**

Client-initiated HT (CITC)

• Introduction and orientation to session

• Risk assessment

• Consent for the test

Provider Initiated HT (PITC) in health facility setting

• Introduction and information on importance of testing for HIV

• Consent for the test

• Test preparation

**Perform test using approved rapid HIV antibody test kit**

**Post-test counselling for negative results**

• Risk reduction plan

• Linkage to other HIV prevention services

• Re-testing where applicable

**Post-test counselling for positive results**

• Enrolment into comprehensive care and treatment

• Risk reduction and positive living counselling

• Partner/family testing

**Management**

**PMTCT Nevirapine Prophylaxis:**

**Age and dosing**

0 - 6 wks- give **Nevirapine dose-** 10 mg (*1ml*) once daily (*Birth weight < 2,500 grams)*

15 mg (*1.5ml*) once daily (*Birth weight > 2,500 grams)*

6 - 14 wks give 20 mg (2mls) once daily

14 wks - 6 months give 25 mg (2.5mls) once daily

6 - 9 months give 30 mg (3mls) once daily

**Duration of infant ARV propylaxis:** Immediately initiate Nevirapine (NVP) prophylaxis for 12 weeks

• Do HIV PCR test in accordance with national recommendations on early infant diagnosis;

• Initiate treatment if the infant is Infected Infant identified as HIV exposed after birth (through infant or maternal HIV antibody testing) and is breastfeeding Nevirapine Immediately initiate NVP prophylaxis

• Do HIV PCR test in accordance with national recommendations on early infant diagnosis

• If results positive, initiate ART and stop NVP prophylaxis

• If results negative, continue NVP prophylaxis up to 12 weeks

Infant identified as HIV exposed after birth (through infant or maternal HIV antibody testing) and is not breastfeeding/on replacement feeding- give no drug and Do HIV PCR test in accordance with national recommendations on early infant diagnosis;

• No infant ARV prophylaxis;

• Initiate treatment if the infant is infected

**Ongoing treatment/feeding**

1) If breast fed encourage exclusive breast feeding until 6 months. If an alternative to breast feeding is affordable, feasible, accessible, safe and sustainable (AFASS) discuss this option before delivery.

2) Do not abruptly stop breast feeding at 6 months, just add complementary feeds and continue nevirapine until 1 week after breast feeding stops.

3) Refer child and carers to an HIV support clinic.

4) All HIV exposed / infected infants should start CTX prophylaxis from age 6 wks.

Infants and children depend on their caregivers for adherence to medication. Palatability can also occasionally cause children to refuse medication. Caregivers should be adequately prepared for the role of administering ARVs to infants and children, including anticipated challenges and possible solutions.

< 2 weeks AZT + 3TC + NVP3

2 weeks - < 4 weeks ABC + 3TC + LPV/r

4 weeks - < 3 years ABC + 3TC + LPV/r4

3 - 15 years (< 35 kg body weight) ABC + 3TC + EFV5

3 - 15 years (≥ 35 kg body weight)

TDF + 3TC + EFV

> 15 years TDF + 3TC + EFV6,7

PWID > 15 years TDF + 3TC + ATV/r

**Oral pre –exposure prophylaxis (PrEP)**

**Infant ARV propylaxis is niverapine**

**Read;**

* Disorders of the special senses includes conditions of the ear, nose, throat and eyes
* Infectious diseases which include the communicable disease and diseases under immunization coverage