Breastfeeding

"Breastfeeding should be initiated as soon after birth as possible, depending on the infant's ability to tolerate enteral nutrition. This not only maintains normal metabolism during the transition from fetal to extrauterine life but also promotes maternal-infant bonding. Most infants can start breast-feeding shortly after birth, almost always within 4-6 hr".

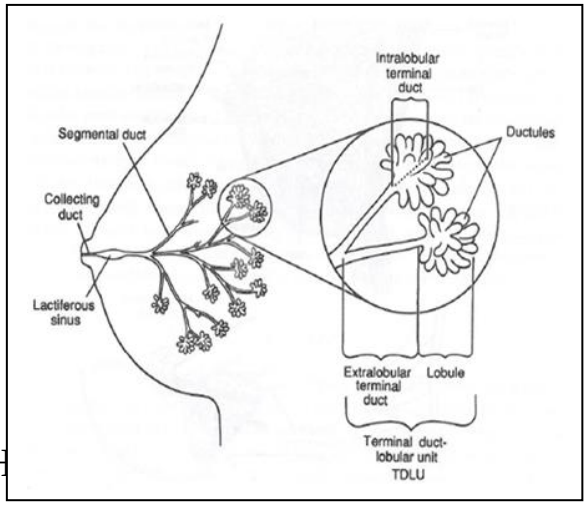
Advantages of Breastfeeding:

1- Composition: breast milk is nutritionally superior to any alternative and is easily digestible.
2- Convenience: breast milk is ready to serve anytime and anywhere, always fresh and supplied at the proper temperature.
3- Sterility: breast milk is sterile.
4- Anti-infective properties
   a. Immunoglobulins (IgA): these are present in colostrum and to a lesser extent in mature milk. IgA acts in the gut against bacteria and viruses.
   b. Lysozyme: it destroys harmful bacteria and viruses.
   c. Lactoferrin: it acts against harmful bacteria.
   d. White blood cells: these are abundant in breast milk in the first 2 weeks. They secrete IgA, lysozyme, lactoferrin and interferon. The latter inhibits certain viruses.
   e. Bifidus factors: this is necessary for growth of lactobacillus bifidus bacteria which produce lactic acid that interferes with the growth of harmful bacteria causing diarrhea.

5. Anti-allergic: allergy to breast milk is practically nonexistent. Breast milk gives partial protection against some allergic conditions e.g. infantile eczema.
6. Inexpensive.
7. Psychologic: breastfeeding is a satisfying experience for both mother and infant, the mother feels a sense of accomplishment, the infant is afforded close and comfortable physical and sensual contact essential for his emotional development.

Physiology of lactation:

Milk is produced by the secretory alveoli then it goes to lactiferous ducts towards the nipple. The milk collects in milk sinuses beneath the areola, that is, the dark skin around the nipple.



Mechanisms of Milk Production:

1- Maternal reflexes:

a. **Prolactin reflex** (Milk secreting reflex): as the baby suckles, impulses pass from the areola up to the hypothalamus then to the anterior pituitary producing prolactin which makes the breasts produce milk.

H

b- **Oxytocin reflex** (Milk ejection or let-down reflex): suckling also stimulates the production of oxytocin hormone by the posterior pituitary. Oxytocin causes contraction of the smooth muscles surrounding the alveoli, squeezing milk out. The milk ejection reflex is the key reflex for successful lactation.

2- Infant`s reflexes:

a. Rooting reflex: the circumoral area or cheek of the infant are touched by the nipple, leads to turning of the head to the side on which the nipple is felt and the infant draws it into his opened mouth.

b. Suckling reflex: The tactile stimulus caused by the nipple and areolar tissues filling the mouth lead to milking action by the tongue against the hard palate.

c. Swallowing reflex: this enables the baby to ingest the milk that is obtained by suckling, and allows interruption of breathing to prevent choking during swallowing.

Technique of Breastfeeding:

1- General cleanliness:

General cleanliness of the mother and infant should be encouraged.

2- Position:

a- The infant, who should be hungry at the feeding time, is held in a semi-sitting position.
b- The mother should be completely at ease.
c- The mother bends forwards slightly so that her nipple finds its way easily into the infant's mouth after initiating the rooting reflex. A considerable portion of the areola is taken into the mouth.
d- The nipple is steadied by the index and middle fingers of the free hand which also holds the breast away from the baby's nostrils.

3- The end of a feed:

a- This is determined by the infant who releases the nipple.
b- Prolonged suckling while the infant falls asleep and the breast is already empty should be discouraged.
c- After feeding, leave few drops of expressed breast milk to dry on the nipple.

4- Eructation or burping the baby:

After feeding, the infant is held against the mother's shoulder and tapped gently on his back several times to drive off any swallowed air and then allowed to sleep.

Exclusive Breastfeeding:

This means that the baby has no other food or drink but breast milk. This is the best and the safest way to feed a baby up to the age of 6 months. Babies do not normally need anything other than breast milk even in the first few days after birth.

Composition of breast milk:

<u>Colostrum</u>

This is the bright yellow, alkaline, viscous fluid, produced during the first few days (5-7 d) after delivery. Colostrum has the following characteristics:

1- The amount produced, about 30-100 ml, is sufficient for the first few days of life.
2- High content of IgA, leucocytes, lactoferrin and lysozyme to protect against infection.
3- High protein content (2-3 gram/dI) less carbohydrate (5-7 g/dl).

4- Fat (2.3 g/dl) and lactose are less in colostrums than in mature breast milk.
5- High vitamin A content.
6- Laxative effect.
7- Rich in (Na^+, K^+, Cl^-) and microminerals (Zinc, Copper).

Mature Breast Milk: few days, colostrum changes to mature breast milk which is grayish in color.

Protein:

Human milk contains a higher proportion of soluble whey protein (lactalbumin and lactoglobulin) than casein. Thus, a soft easily digestible curd is formed.

Fat:

1. Lipase enzyme is present to help digestion.
2. Sufficient amount of the essential fatty acid linoleic acid is present.
3. Fat globules are small and easily digestible.
4. High level of arachidonic acid and cholesterol essential for brain development.

Lactose:

This is the only carbohydrate in breast milk. Some lactose is converted in the intestine to lactic acid which prevents growth of pathogenic bacteria and also helps calcium absorption.

Minerals:

1- The ash content is low to avoid any excess osmolar load and renal solute load.
2- The calcium/phosphorus ratio is optimal for absorption of both.
3- Iron absorption and utilization are efficient.

Vitamins:

If the mother's diet is adequate her milk will satisfy all the vitamin requirements including **vitamin C** during the first 4-6 months of life.

The **vitamin K** content of human milk also is low and may contribute to hemorrhagic disease of the newborn. Parenteral administration of 1 mg of vitamin K_1 at birth is recommended for all infants, and this is especially important for those who will be breast-fed.

Milk from the mother whose diet is sufficient and properly balanced will supply all the necessary nutrients except fluoride and vitamin D. **Vitamin D** intake should be 200 IU/day, starting at 2 mo of age for all breast-fed infants. If the water supply is not adequately fluoridated (≤0.3 ppm), the breast-fed infant should receive at least 10 mg of **fluoride** daily for the 1st 6 mo of life; thereafter, the fluoride intake should approximate the adequate intake.

The **iron** content of human milk is low, but most normal term infants have sufficient iron stores for the 1^{st} 4–6 mo of life. Human milk iron is well absorbed. Nonetheless, by 4–6 mo of age, the breast-fed infant's diet should be supplemented with iron-fortified foods and/or a ferrous iron preparation.

Foremilk & Hind milk:

Breast milk is not exactly the same throughout a feed.

Foremilk: at the beginning of a feed the milk looks grey and watery. The foremilk contains plenty of nutrients and water but not much fat.

Hind milk: towards the end of a feed, the milk looks much whiter. The hind milk is rich in fat due to the squeezing effect of the smooth muscles surrounding the alveoli as an effect of the oxytocin.

Problems and Difficulties during Breast Feeding

1- Suckling in a poor position:

Signs of good versus poor suckling positions:

Signs that a baby is suckling in a GOOD position	Signs that a baby is suckling in a POOR position
• The baby's whole body is facing his mother and his face is close to the breast. • His chin is touching the breast. • His mouth is wide open. • His lower lip is curled outwards. • You see more areola above the upper lip than below the lower lip. • The baby takes slow deep sucks. • You can see or hear the baby swallowing. • His cheeks are round, and not pulled in. • The baby is relaxed, happy, and satisfied at the end of a feed. • The mother does not feel any pain.	• The baby's body may be turned away from his mother's and not close to her. • His chin is separated from the breast. • His mouth looks closed. • His lips point forwards. • You can see most of the areola. • The baby takes many quick, small sucks. • You do not see or hear the baby swallowing you may hear smacking sounds as he sucks. • The baby's cheeks may be pulled in. • The baby may fuss or refuse to feed because he does not get enough breast milk. • The nipple looks flattened as it comes out of the baby's mouth, and there may be a line across the tip.

2- Insufficient Breast Milk:

Many mothers falsely believe that the amount of breast milk is not sufficient. However, the most valuable sign that the mother is producing sufficient milk is adequate weight gain of the infant as shown by follow-up of the weight curve.

Meanwhile, give the mother advice to promote breastfeeding by increasing the frequency of the feeds on demand day and night. If, however, the weight curve is not satisfactory, the mother should continue frequent on-demand breastfeeding and complement the feeds with yoghurt or formula.

Certain rules should be fulfilled with complementary feeding to avoid inhibition of breast milk:

1- Use cup and spoon, sucking from a bottle, which is an easier passive process may cause the baby to refuse breastfeeding (nipple confusion).
2- Both breasts should be given and completely emptied at each feed.
3- The feed should always be immediately after breastfeeding.
4- The feed should not be too sweet.
5- The amount of the complementary feed is determined by trial and errors i.e. give an amount to the infant and see how much he accepts.

3- Delayed Appearance of Milk:

Normally the milk flow starts after 2 or 3 days but in primipara it is usually delayed till the 4^{th} or 5^{th} day. Inform the mother to allay her anxiety and encourage her to breastfeed as colostrum is all that is needed.

4- Inability to Suckle or Refusal to Breastfeed:

This may result from:

1. Improper position during feeding.
2. Sore mouth due to thrush or ulcer.
3. A baby who is accustomed to bottle feeding.
4. Blocked nose from nasal catarrh.
5. Pendulous breasts obstructing the nostrils.
6. Anorexia due to infection.
7. Sleepy feeder: this is common in the first few days after birth particularly in prematures and following heavy maternal analgesia.
8. Scanty breast milk causes the infant to cry and refuse breastfeeding.

5- Mother Breast Engorgement:

This may result from poor suckling due to improper position or obstruction of milk ducts by epithelial secretions and debris. The breasts become distended, tense, tender, reddened and firm with lumps of accumulated milk. In severe cases, the baby holds the nipple only in his mouth causing cracked or fissured nipple and infection may occur on top.

Treatment: manual expression of breast milk, analgesics and warm fomentation.

Prevention: proper antenatal care, on demand feeding, maintaining proper feeding position.

6- Fissured or Cracked Nipples:

This occurs when the infant is not taking enough of the areola inside his mouth while suckling. It results from engorgement, improper position or retracted nipple.

Treatment includes:

1. Nurse the baby in a different position from the usual one; in this way the part of the nipple which is subjected to repeated trauma from suckling is allowed to rest and heal.
2. Local application of hydrous lanolin to soothe the sore nipple may help. Wetting the nipple with breast milk is sometimes helpful.
3. Let the baby suckles first from the least affected breast, because the initial suckling is the strongest and thus the most painful.

Prevention: proper antenatal care, ensure proper feeding position and early treatment of engorgement.

7- Flat or Retracted Nipples:

Retracted or nonprojectile nipples are common. Most nipples in fact do protract even if they look flat. Nipple exercises before and after delivery and normal feeding cure most cases.

8- Regurgitation after Breastfeeding:

Commonly the infant may regurgitate a small amount of milk after feeding. This should not worry the mother; reassure her by demonstrating adequate weight gain of her infant.

Work and Breastfeeding:

At the time of returning to work advise the mothers about the following:-

1- Breastfeed shortly before leaving for work and soon after return.
2- At work, avoid engorgement by milk expression.
3- Increase the frequency of breastfeeding in the afternoon and at night.
4- If the mother is absent for more than 3 hours, she can express breast milk in a clean bottle for her baby or supplementary food e.g. yoghurt or milk by cup and spoon may be given.
5- Keep the infant hungry at the time when the mother is back at home and ready to breastfeed.

Maternal Diet during Breastfeeding:

The mother is allowed to eat whatever she likes but the diet should be nourishing with lot of fluids.

Drugs and Breastfeeding:

Some drugs are excreted in breast milk in amounts sufficient to harm the infant, these include: radioiodine, anticancer drugs, chloramphenicol, diazepam, barbiturates, tetracyclines, nalidixic acid, metronidazole, steroids, oral contraceptives, ergot alkaloids and cascara laxatives.

Contraception and Breastfeeding:

Estrogen containing oral or injectable contraceptives decreases the amount of breast milk and should not be given to lactating mothers. Advise for an alternative method.

Menstruation and Breastfeeding:

The resumption of menstruation should not deter continued breast-feeding. However, temporary behavior changes of the mother and/or the baby may call for reassurance.

Twins and Breastfeeding:

Mothers can easily breastfeed healthy twins without difficulty. However, she needs proper nourishment and adequate fluid intake. Follow up the weight curves of the twins to see the progress.

Duration of Breastfeeding:

The Holy Koran declares that "mothers shall give suck to their children for two whole years". Breastfeeding should continue for 2 years with supplements from the age of 6 months.

Contraindications of Breastfeeding

I. Absolute contraindications: (*i.e. breast milk is harmful to the infant*)

Causes related to the infant:

1. Inborn errors of metabolism as galactosemia and phenylketonuria.
2. Inborn errors of digestion as monosaccharides and disaccharides intolerance.

Maternal causes:

1. Maternal hepatitis B: Unless the newborn receives Hepatitis B immune globulin and Hepatitis B vaccine at birth, and then completes the hepatitis B vaccination schedule.
2. Maternal HIV/AIDS: Breast-feeding is not recommended if a safe alternative is available.
3. Intake of dangerous toxic drugs which are secreted in milk in considerable amounts:
   A. Anticoagulants, antineoplastics (cyclophosphamide, cyclosporine, etc.), thiouracil, ergotamine, phenindione, lithium.
   B. Radioactive substances. C, Cocaine, heroin, marijuana.

II. Temporary Contraindications:

Causes related to the infant:

1. Severe cleft palate, micrognathia.
2. Infant infections: Oral herpes simplex.

Maternal causes:

1. Psychosis, neurosis and epilepsy.
2. Maternal infections:
   a. Herpes simplex lesions on the breast (until healed).
   b. Chicken pox: Zoster immune globulin (ZIG) is given to non infected neonate. The neonate is separated from the mother until she is no longer infectious.
   c. Active tuberculosis: Mother is treated. Infant receives INH and is repeatedly tested with tuberculin test. INH is discontinued if tuberculin is still negative after 3-4 months of age and the mother response to treatment is satisfactory.
   d. Breast abscess: do not feed from the affected breast until healed.
   e. Septicemia, typhoid fever, pneumonia until treated.
3. Eclampsia.
4. Debility: Malignancy, chronic cardiac and renal disease, neoplasia or severe malnutrition.
5. Pregnancy: Although pregnancy is not a contraindication, it is wise to stop breast-feeding after the 5^{th} month of pregnancy because the mother cannot afford the combined nutritional demands of supplying milk to the baby and nutrients to the fetus.

Promotion of Breastfeeding

These include helping & encouraging mothers to breastfeed and prolong lactation for at least 2 yrs.

1- Prenatal period: As previously discussed. Involvement of the obstetrician is crucial.

2- Delivery and neonatal period:

- Avoid maternal fatigue, anxiety and pain by avoiding unnecessary episiotomy, early allowing eating and a relaxed atmosphere.
- Avoid excess maternal anesthesia which causes sedated newborn unable to suckle properly.
- Start breastfeeding within the first half hour after delivery.
- Allow rooming-in to allay mother's anxiety and permit frequent suckling.
- Allow on-demand breastfeeding day and night.

- Avoid bottle feeding as it may make a baby gives up or refuses breastfeeding (nipple confusion).
- In neonatal intensive care unit, use expressed breast milk, allow contact between mother and baby as early as feasible with the earliest return to direct breastfeeding.

3- Post-neonatal period:

- Avoid estrogen containing oral and injectable contraceptives.
- No solids or semisolids supplements in the first 4 months.
- Mothers with insufficient milk or other problems should be given the extra support, advice and encouragement.
- Breastfeeding should be prolonged as much as the mother and child wish preferably till 2 years with proper supplementation from 6 months.

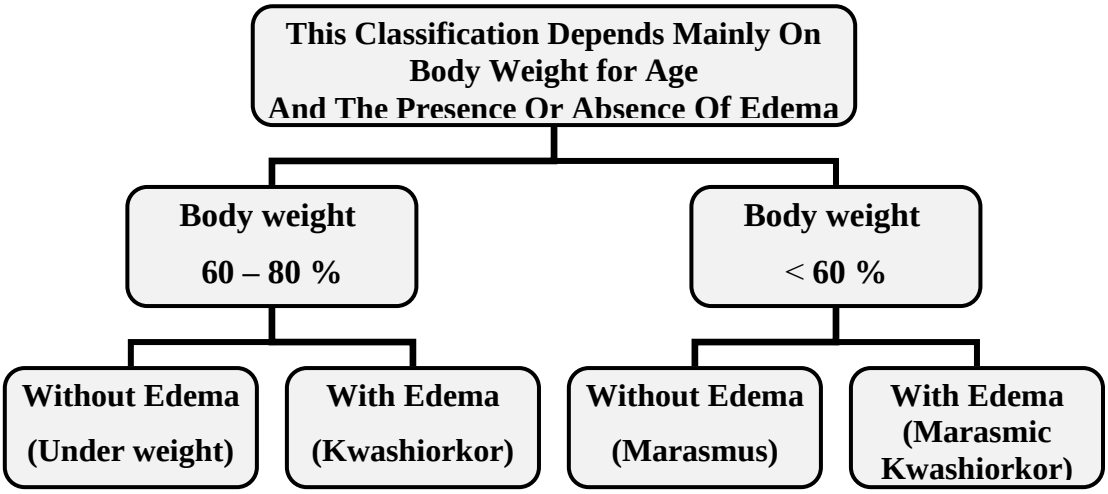
No factor is more important for successful breast-feeding than a happy, relaxed state of mind.

Protein Energy Malnutrition (PEM)

A group of clinical conditions, that results from varying degrees of lack of protein and / or energy.

Classification of PEM:

1-Welcome classification: It depends on changes in weight and the presence or absence of edema.



2- Water-Law classification: This classification gives an idea about the duration of occurrence of the disease.

- **Wasted**: ⟶ **Weight for length < 80 % of the standard**

  - This means acute malnutrition (within 6 months).

- **Stunted**: ⟶ **Length for age < 90 % of the standard**

  - This means chronic malnutrition (more than 6 months).

- **Wasted & stunted**: ⟶ **Weight for length < 80 % of the standard Length for age < 90 % of**

Abnormal nutrition:

a- Undernutrition.

b- Overnutrition: e.g. caloric excess (obesity).

c- Malnutrition: deficiency of one or more elements (proteins, vitamins, or minerals), Kwashiorkor is a very important example

Undernutrition

- It is the simple & commonest type of abnormal nutrition.
- Body weight = 60 – 80 %.
- No edema.
- Can be avoided by careful regular weighing of the body during routine examination.

Malnutrition

Kwashiorkor

- Kwashiorkor results from lack of protein in diet.

Age: from 6 months to 3years.

Etiology:

General causes: 1- Maternal ignorance 2- Poverty

Dietetic errors: Excess starchy feeding.

Infections: 1- Diarrhea 2- Measles 3- TB

Clinical pictures:

Constant features:

1- Growth failure:

- Weight at 60 – 80 % of standard age.
- Failure to gain weight then followed by weight loss.
- Wasted → decreased weight for length.
- Lastly, height and head circumference may be affected.

2- Muscle wasting:

- Mostly affected the biceps and the triceps.
- Leading to hypotonia and weakness.
- Muscle wasting: detected by:

• Decreased mid-arm circumference.
• Decreased muscle / fat ratio.
• Decreased skin folds thickness.

3- Edema (nutritional edema):

- Mainly due to hypoproteinemia.
- There is increased level of ADH and aldosterone.
- Starts early at the dorsum of feet and legs, then it becomes generalized bilateral & pitting.
- Buffy checks with moon face appearance.
- Not associated with ascites. Ascites does not occur in kwashiorkor; rarely occurs as in septic peritonitis and during nutritional recovery syndrome.

4- Mental changes:

- The patient becomes apathetic, disoriented with his surroundings.

Variable features:

1- Hair changes:

- Color: lighter progressively, black to dark brown, and light brown to orange to yellow.
- Texture: soft and easily broken.
- Distribution: sparse.
- Attachment: - Loose easily pickable without pain.

- Flag sign (diagnostic sign) due to repeated attacks and affection of hairs in segmental manner which leads to bands of light color alternating with bands of darkening in the same hair.

2- Skin changes:

- Dry scaly skin followed by erythema.
- Areas of hyperpigmentation which is followed by exfoliated skin.
- Areas of hypopigmentation.
- Fissuring and cracking of the skin.
- Purpura.
- Secondary bacterial infection.

3- Hepatomegaly (fatty liver):

- Soft to firm and smooth with rounded border.
- Caused by increased fat mobilization to the liver from the body.

4- Gastro-intestinal manifestations:

- Anorexia caused by infections and mental changes.
- Diarrhea, caused by: Infection, maldigestion, malabsorption and lactose intolerance.
- Vomiting.

5- Anemia may be:

- Microcytic hypochromic anemia: due to iron deficiency anemia.
- Normocytic normochromic anemia: due to infection and hypoproteinemia.
- Megaloblastic anemia: due to folic acid and vitamin B_{12} deficiency.

6- Infections: occurs due to:

- Epithelial cells lining of the gastro-intestinal tract and respiratory tract usually becomes unhealthy and this leads to invasion by micro-organisms.
- Impaired immunity.

• ***Infection*** in malnourished patients may be masked by *absence of fever* due to*:*

· Edema leading to increase heat loss.
· Hypoglycemia leading to decrease heat production.
· Impaired shivering due to muscle wasting.

- *Most common infections are: gastro enteritis, pneumonia, otitis media, T.B., urinary tract infection, Candida infections.*

7- Signs of vitamin deficiencies:

- Vitamin A deficiency: xerosis, keratomalacia, night blindness, corneal opacities.
- Vitamin B deficiency: glossitis, angular stomatitis.
- Vitamin C deficiency: scurvy, bleeding gums.
- Vitamin D deficiency: rickets.

8- Hemorrhagic manifestations: mainly due to:

- Vitamin K deficiency.
- Protein deficiency and increased capillary fragility.

Investigations:

Plasma proteins:

1- Decreased total serum proteins.
2- Decreased serum albumin.
3- Decreased ∂ and β globulins.
1- Decreased essential aminoacids.

5- Reversed albumin / globulin ratio ($N = \frac{4.5}{2.5}$).

6- Normal or increased non-essential aminoacids.
7- Special proteins:

- Decreased transferrin (used to transfer iron).
- Decreased ceruloplasmin (used to transfer copper).
- Decreased haptoglobulin.

Blood sugar: Hypoglycemia.

Water and electrolyte disturbances:

1- Increased total body water (intra and extra cellular).
2- Increased sodium level but water retention is excessive (dilutional hyponatremia).
3- Decreased potassium level, mainly due to vomiting and diarrhea.
4- Decreased calcium level.

Minerals:

Mg, Fe, Cu, Zn and all other trace elements are reduced.

Hematological changes:

1- Anemia.
2- Leucocytosis, may be leucopenia.
3- Thrombocytosis.

Tuberculin test.

Chest X-ray.

Complications of kwashiorkor:-

- **Infections:**

1- Bronchopneumonia is the most common cause of death.
2- Others: otitis media, U.T.I., T.B., monilial infections.
3- Gastroenteritis: diarrhea, malabsorption and dehydration.

- **Hypoglycemia.**
- **Heart failure:** due to:

1- Anemia. 2- Volume overloads (fluid or blood).
3- Weak myocardial contractility (due to vitamin B deficiency).

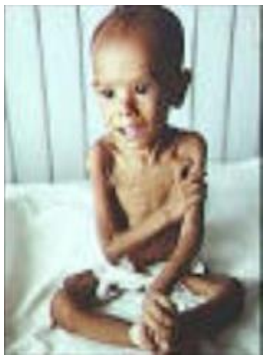
Marasmus

Definition:

It is a state of chronic malnutrition due to deficiency of total caloric requirements.
- Commonly seen in the first 2 years of life.

Etiology:

A) Socio-economic causes: Ignorance, poverty, depression.



B) Dietetic errors (nutritional Marasmus):

1- Quantitative disorders:
- Scanty breast milk (in amount or number of feeds).
- Small amount of feed. Delayed weaning.

2- Qualitative disorders:
- Over dilutional formula in artificial feeding.
- Cow's milk protein allergy.

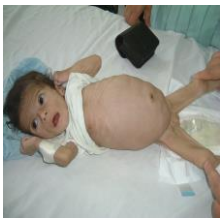
C) Non-dietetic errors (secondary Marasmus):

1- Gastroenteritis:
Recurrent attacks of diarrhea or chronic diarrhea usually associated with P.E.M.

2- Malabsorption syndromes:

3- Infections:
Recurrent acute or chronic infections: as T.B., pyelonephritis, chronic suppurative lung disease.

4- Congenital abnormalities:
- G.I.T: congenital pyloric stenosis, cleft lip and palate.
- Liver: congenital hepatic cirrhosis.
- C.V.S: Fallot`s Tetralogy, V.S.D.
- Chest: congenital interstitial fibrosis.
- Renal: renal agenesis, obstructive uropathy.
- C.N.S: defective cerebral development.



5- Metabolic disorders:
- Renal tubular acidosis. - Fructosemia. - Urea cycle defects.
- Galactosemia. - Amino acid defects.

6- Endocrinal disorders:
- Juvenile D.M.- Adrenal insufficiency.

Clinical picture of Marasmus:-

I. Growth failure:

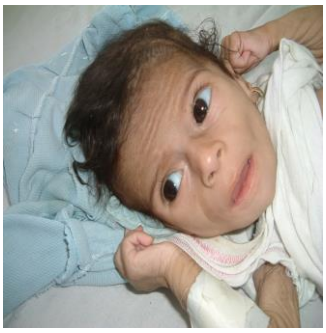
1- At first, there is failure to gain weight then loss of weight occurs.
2- Weight less than 60% of the ideal weight for age.

II. Loss of subcutaneous fat:

First degree: loss of subcutaneous fat in the abdominal wall.
Second degree: loss of subcutaneous fat in limbs, buttocks and abdominal wall.
Third degree: loss of subcutaneous fat in face limbs and abdominal.

III. Muscle wasting:

Detected by decreased mid-arm circumference.



IV. Gastro-intestinal manifestations:

1- Anorexia.
2- Constipation due to lack of food intake.
3- Diarrhea due to gastroenteritis and malabsorption.

V. Signs of vitamin deficiencies.

VI. Infections: Pneumonia, Gastroenteritis, Otitis media, U.T.I and T.B.

VII. General manifestations: Marked pallor due to associated anemia. Subnormal temperature due to loss of subcutaneous fat.

Investigations:

To detect the causes in cases of secondary marasmus and to detect the complications.

- ***Complete blood picture:***

1- RBCs: anemia (all types of anemia can be found).
2- WBCs: leucocytosis or leucopenia.
3- Platelets: thrombocytopenia.

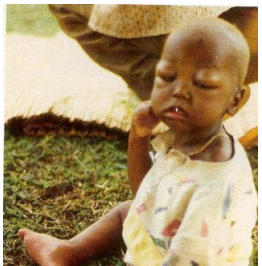
- ***Total proteins and serum albumin:***

1- Slightly reduced.

- ***Urine analysis:***

1- Culture in case of U.T.I.
2- Glucosuria in case of D.M.

- ***Stool analysis:*** For parasites or steatorrhea.
- ***Chest X-ray:*** For bronchopneumonia or congenital heart disease.



- Tuberculin test.

- Intestinal biopsy: If there is malabsorption.

Complications:-

1-Edema: marasmus kwashiorkor.
2- DIC.
3- Pressure sores.
4- Fatal hypothermia.

Vitamin Deficiencies and Excesses

Vitamins are essential nutrients that must be supplied exogenously either as part of a well-balanced diet or as supplements.

Summary of clinically relevant information on vitamins are listed in table at the end of this chapter.

VITAMIN A

Retinol (vitamin A_1) is an alcohol of high molecular weight; 1 μg of retinol = 3.3 IU vitamin A. Provitamin A: the plant pigments α-, β-, and γ-carotenes and cryptoxanthin. Fat soluble, heat stable, destroyed by oxidation and drying. Bile is necessary for absorption and stored in liver.

Clinical Manifestations:

1- Ocular lesions of vitamin A deficiency develop insidiously and rarely occur before 2-3 yr of age. The posterior segment of the eye is affected initially with impairment of dark adaptation, resulting in night blindness. Later, drying of the conjunctiva (xerosis conjunctivae) and cornea (xerosis corneae) occur, followed by wrinkling and cloudiness of the cornea or keratomalacia. Dry, silver-gray plaques may appear on the bulbar conjunctiva (Bitot spots), with follicular hyperkeratosis and photophobia.
2- Apathy, retardation of mental and physical growth.
3- Anemia with or without hepatosplenomegaly is usually present.
4- The skin is dry and scaly, and follicular hyperkeratosis may be found on the shoulders, buttocks, and extensor surfaces of the extremities.
5- Epithelial metaplasia of the urinary tract may result in pyuria and hematuria.
6- Increased intracranial pressure with wide separation of cranial bones may occur, but hydrocephalus, is an infrequent manifestation of vitamin A deficiency.

Diagnosis:

Dark adaptation tests may be helpful in diagnosing vitamin A deficiency. Xerosis conjunctivae can be detected by biomicroscopic examination of the conjunctiva. The plasma carotene concentration falls quickly, but that of vitamin A decreases more slowly.

Prevention:

Infants should receive at least 500 μg of vitamin A daily, and older children, like adults, should receive 600-1500 μg of vitamin A or carotene. Because maternal vitamin A status is reflected in breast milk vitamin A content, mothers of breast-fed infants living in regions where vitamin A deficiency is common should be given 30,000 μg [100,000 IU]) of vitamin A postpartum.

Treatment:

A daily supplement of 1,500 μg of vitamin A is sufficient for treating latent vitamin A deficiency. Xerophthalmia is treated by giving 1,500 μg/kg orally for 5 days followed by daily intramuscular injection of 7,500 μg of vitamin A in oil until recovery occurs.

Hypervitaminosis A

Clinical Manifestations:

Acute hypervitaminosis A: may occur in infants after ingesting 100,000 μg or more. *The symptoms* are nausea, vomiting, drowsiness, and, in young infants, bulging of the fontanel. Diplopia, papilledema, cranial nerve palsies, and other symptoms suggestive of brain tumor **(pseudotumor cerebri)** may also be present.

Chronic hypervitaminosis A: results from ingestion of excessive doses for several weeks or months. *The symptoms* are anorexia, pruritus, and a lack of weight gain. Irritability, limitation of motion, with tender swelling of the bones, alopecia, seborrheic cutaneous lesions, fissuring of the corners of the mouth, increased intracranial pressure, and hepatomegaly also may develop. Craniotabes and desquamation of the palms and soles are common. **Radiographs** show hyperostosis affecting several long bones; it is most notable at the middle of the shafts. In addition, **the serum vitamin A** level is elevated.

- Severe congenital malformations may occur in infants of mothers who consume large amounts of oral retinoids for treatment of acne.
- Ingested carotenoids, although nontoxic, may result in yellow discoloration of the skin *but not of the sclera*. This disorder, ***carotenemia***, is especially likely to occur in children with liver disease, diabetes mellitus, or hypothyroidism and in those who have congenital absence of enzymes that convert provitamin A carotenoids.

Rickets and Hypervitaminosis D

Rickets is a metabolic disease, occurs in children only before fusion of the epiphyses. It is associated with failure of mineralization of growing bone or osteoid tissue.

The early changes are seen radiologically at the ends of long bones with evidence of shaft demineralization, if healing is not initiated → clinical manifestations appear.

Causes of Rickets:

<table>
<tr><th>VITAMIN D DISORDERS</th><th>CALCIUM DEFICIENCY</th></tr>
<tr><td>Nutritional vitamin D deficiency</td><td rowspan="3">Low intake
Diet
Premature infants (rickets of prematurity)</td></tr>
<tr><td>Congenital vitamin D deficiency</td></tr>
<tr><td rowspan="4">Secondary vitamin D deficiency
Malabsorption
Increased degradation
Decreased liver 25-hydroxylase
Conditions that interfere with vit D metabolism:
(Anticonvulsants: phenytoins, phenobarbitone)</td></tr>
<tr><td>Malabsorption</td></tr>
<tr><td>Primary disease</td></tr>
<tr><td>PHOSPHORUS DEFICIENCY</td></tr>
<tr><td>Vitamin D–dependent rickets type 1</td><td>Inadequate intake</td></tr>
<tr><td rowspan="2">Vitamin D–dependent rickets type 2
(End organ resistance)</td><td>Premature infants (rickets of prematurity)</td></tr>
<tr><td>Aluminum-containing antacids</td></tr>
<tr><td>Chronic renal failure</td><td>RENAL LOSSES</td></tr>
<tr><td></td><td>X-linked hypophosphatemic rickets</td></tr>
<tr><td>DISTAL RENAL TUBULAR ACIDOSIS</td><td>Autosomal dominant hypophosphatemic rickets</td></tr>
<tr><td></td><td>Fanconi syndrome</td></tr>
<tr><td></td><td>Hereditary hypophosphatemic rickets with hypercalciuria</td></tr>
</table>

Vitamin D deficiency rickets

This is common in developing countries; the prevalence is 5% among infants and preschool children.

Epidemiology:

Age: 6months – 2years – peak at 18months.

Sex: more in males.

Race: Negros is more susceptible.

Growth: more in rapidly growing infants e.g. twins, preterm.

Environment: smoke, dust, clouds & ordinary window glass prevent ultraviolet rays to reach the skin.

Rachitogenic diet:

- High phosphate content in unmodified animal milk → decrease calcium absorption.
- Cereals rich in phytates and phosphates interfere with calcium absorption due to formation of insoluble salts with calcium.

Pathology:

New bone formation is initiated by osteoblasts which are responsible for matrix deposition and with mineralization (Ca and ph are essential for mineralization).

Normally: There are two types of ossification:

- Subperiosteal →↑ in bone thickness.
- Intercartilagenous →↑ in bone length.
- The growth plate at the end bone contains 4 different zones:
  - Zone of resting cartilage: formed of single layer of columnar cells.
  - Zone of proliferating cartilage: 4-6 layers, irregular, avascular with no calcium.
  - Zone of provisional calcification or degenerating cartilage: the chondrocyte becomes swollen, vacuolated; Ca and Ph are precipitated in the matrix forming well defined line (epiphyseal line).
  - Zone of ossification: the degenerating chonchocytes are invaded by capillaries and the osteoblasts deposit a layer of osteoid with normal Ca / Ph ratio → mineralization of osteoid occurs.
  - The calcified cartilage is ultimately replaced by bone.

In rickets:

- Zone of resting cartilage → no change.
- Zone of proliferating cartilage → formation of many layers → enlarged zone.
- Zone of provisional calcification: ↑ Vasculature. Failure of chondrocytes to die, no Ca deposition in the lower end of long bone → frayed, irregular.
- Zone of ossification: Poor mineralization of the new osteoid → uncalcified osteoid → yield with pressure → cupping.
- Generalized rarefaction of bone shaft leading to deformities and greenstick fracture.

Clinical picture:

Early manifestation:

- Anorexia. Irritability, sweating.
- Craniotabes: caused by thinning of the outer table of the skull. It can be detected by a ping-pong sensation on pressure over the occiput and posterior parietal bone.
- Rachitic rosaries: It is a prominant enlargement of the costochondral junction and felt as a raw of beads (due to excess osteoid).
- Broadening of wrists and ankles due to epiphysial enlargement.

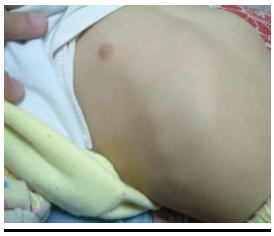
Advanced manifestation:

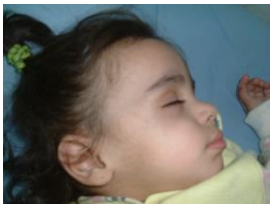
Head:

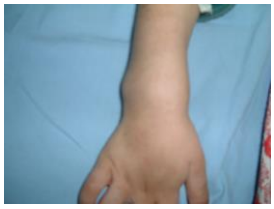
- Large if rickets develops early in the 1st year.
- Asymmetric skull.
- Large anterior fontanel with delayed closure.
- Bossing of the skull ---- due to thickening of the central parts of parietal and frontal bone (caput quadratum).
- Delayed teething with enamel defect and caries may occur.

Thorax:

- Rachitic rosaries.
- Longitudinal grooves developed posterior to the rosaries with flattening of sides of chest cage.
- Harrison sulcus: a horizontal depression at the lower part of the chest along the costal attachment of the diaphragm which is dragged in during inspiration.
- Pigeon breast deformity: the sternum with its adjacent cartilag forwarc







Rackitic Rosaries **Frontal Bossing** **Epiphysial Broadening**

Vertebral spine:

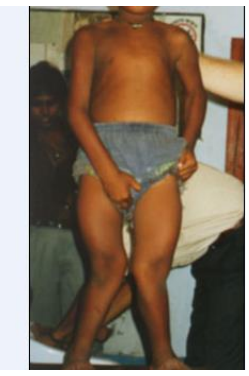
Kyphosis: dorso-lumber and is apparent while sitting due to laxity of spinal muscles and ligament. It disappears if the child is suspended from his shoulder and this differentiates it from those caused by bony deformities (Pott's disease).

Scoliosis: lateral curvature of the spine.

Lordosis: may be seen in the lumber region while standing.

Pelvis: concomitant deformity (contracted inlet and outlet) may occur, in girls if become permanent leads to obstructed labor.

Extremities:



- Broadening of epiphysis of long bone especially at wrist and ankle.
- Marfan sign: a transverse groove over the medial malleolus due to defect in osteoid deposition in the centres of ossification
- Deformities: due to weight bearing at the shaft of bones leading to:
    - Bowing of forearm in creeping infants.
    - Bowlegs or knock-knees (genuvarus, genuvalgum).
    - Genue recruvature (over extension) → during walking.

Non skeletal signs:

Hypotonia and laxed ligament lead to:

- Delayed motor milestones e.g. delayed sitting, waddling, walking.
- Pot belly abdomen due to weakness of abdominal muscles.
- Ptosis of liver and spleen: due to chest deformity and weak abdominal muscles.

Complications:

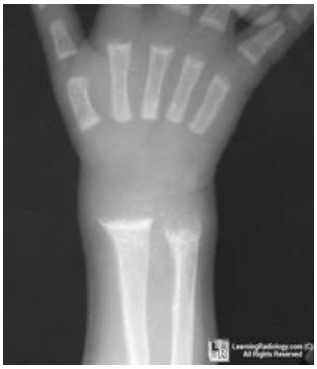
- Respiratory tract infection: due to chest deformity.
- Tetany: is uncommon with nutritional rickets.
- Iron deficiency anemia.
- bone deformities and fracture.
- Dental caries.

Diagnosis:

The diagnosis of rickets is based on typical history (of inadequate intake of vit. D and inadequate exposure to sunlight), clinical examination confirmed by:

Laboratory investigations:

- Serum calcium: may be normal or low (N: 9 -11 mg/dl).
- Serum phosphate level: almost always is less than 4mg/dl (N: 4.5 – 6.5 mg/dl).
- Serum alkaline phosphatase level is increased > 500 IU/1 (N: 50 – 200 IU /dl) due to increased osteoblastic activity.
- Serum parathormone hormone (PTH) → high.
- Serum 25-D → is low.



Non specific findings:

- Generalized ominoaciduria.- Low bone citrate level.
- Elevated urinary citrate excretion.- Impaird renal acidification.
- Phosphaturia and occasionaly glucosuria.

B. Radiological changes:

By X rays: best seen at the lower end of long bone especially wrist and ankle.

- **Active rickets:**

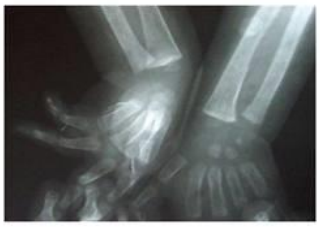
Lower end: broadening

- Cupping (concavity). - Wide joint space.
- Fraying and epiphyseal line (faint, irregular) indistinct.

Shaft:

- Rarefaction → decreased bone density.
- Double periosteal line: along lateral outline.
- Greenstick fracture: may occur in the long bone with no clinical symptoms.

- **Healing rickets (2 – 3 weeks after treatment):**
  - A line of preparatory calcification appears with no fraying.
  - Other features of active rickets are less evident.



- **Healed rickets (after 4 weeks):**

The lower end becomes straight, thick and slightly irregular than normal.

Prevention:

Vitamin D supplementation.

Full term: 400 IU /day from the 3rd month.

Premature: 1000 IU /day from 2nd week.

Treatment:

There are 2 strategies for administration of vitamin D:

a. With stoss therapy, 300,000-600,000 IU of vitamin D is administered orally or intramuscularly as 2-4 doses over 1 day. Stoss therapy is ideal in situations where adherence to therapy is questionable.

b. The alternative is daily, oral vitamin D, with high doses ranging from 2,000-5,000 IU/day over 4-6 wk.

Either strategy should be followed by daily vitamin D intake of 400 IU/day if <1 yr old or 600 IU/day if >1 yr, adequate dietary calcium and phosphorus; this dietary intake is usually provided by milk, formula, and other dairy products.

Hypervitaminosis D

This condition results from excess intake of Vitamin D for a long time (1-3 months).

Clinical picture:

- Anorexia, nausea, vomiting, irritability, pallor.
- Polyuria, polydepsia, constipation.
- Hypercalcemia, hypercalcuria.
- In some cases metastatic calcification of long bones.

Treatment:

- Discontinuation of Vitamin D.
- Corticosteroid (prednisone 2mg/kg) which antagonize calcium transport.

Tetany

Tetany is a state of hyperexcitability of the central and peripheral nervous system due to decreased ionized calcium resulting in a state of neuromuscular irritability. It occurs in severe rickets when calcium level falls below 7mg/dl.

Etiology:

- Hypocalcemia: Poor intake. Malabsorption. Hypoparathyrodism. Neonatal hypocalcemia.
- Hyperphosphatamia.
- Alkalosis in:
  - Excessive vomiting leading to HCL depletion → respiratory alkalosis → decreased ionized calcium.
  - Hyperventilation: leading to Co_2 wash → respiratory alkalosis → tetany.
  - Excess alkali intake: e.g. citrate, bicarbonate.
- Hypomagnesemia:
  - Chronic diarrhea. - Nephrotoxic medication. - Diuretic therapy.

Clinical manifestations of manifest tetany (serum calcium ≤ 7mg/dl):

- Convulsions may be the only manifestation in neonates and young infants, which is usually brief, generalized and occasionally localized with no loss of consciousness between seizures.
- Carpopedal spasm → flexion of the wrist and metacarpophalangeal joints with finger extension.
- Lanyngeal spasm: in the form of inspiratory obstruction (stridor).
- Parathesia: numbness, tingling in hands, feet, perioral especially in older children.
- **ECG:** prolonged corrected QT interval.

Latent manifestation of tetany (serum calcium 7-9mg/dl) detected by:

- Chevostek sign: taping the facial nerve anterior to external auditory meatus elicits a twitch of the upper lip or entire mouth → not specific as it occurs in 10% of patients without hypocalcemia.
- Trousseau sign: is more specific: blood pressure cuff is inflated slightly above the systolic blood pressure for more than 3 min → carpopedal spasm due to ischemia of motor nerve.

Treatment: Emergency treatment: calcium gluconate is given at a dose of 1-2 ml/kg of 10% solution slowly IV while monitoring for bradycardia. The dose can be repeated.

- Maintenance: oral calcium supplementation e.g. Ca carbonate, Ca gluconate.
- ***Hypomagnesemia:*** Mg so_4 IM 0.2 ml of 50% solution.
- ***Alkalosis:*** breath into bag (to ↑Co_2) trying to compensate by inducing respiratory acidosis.

OBESITY

Obesity: The prevalence of childhood obesity has increased, and the prevention and treatment of obesity has emerged as an important focus of pediatric research and clinical care.

Obesity: is the excessive accumulation of fat in the subcutaneous and other body tissues.

Diagnosis of obesity:

A- Body mass index (BMI) is the best parameter to screen for obesity:

1- Overweight: BMI > 85 percentile (child) and BMI 25 - 29.9 (adult).

2- Obesity: BMI > 95 percentile (child) and BMI > 30 (adult).

B- Weight for height:

1- Overweight: body weight > 110 % of the standard weight for height.

2- Obesity: body weight > 120 % of the standard weight for height.

Aetiopathogenesis:

- Exogenous (95%): The causes of exogenous obesity are multifactorial: Genetic factors, increased caloric intake, diminished energy expenditure and unhealthy eating behavior.
- Endogenous (5%):
  - Genetic/chromosomal: Laurence Moon Beidl syndrome , Turner syndrome.
  - Endocrinal: hypothyroidism, Cushing syndrome, GH deficiency.
  - Hypothalamic: Frohlich syndrome, postencephalitic. - Drug induced: steroid.

Height in Obesity:

- ***Exogenous*** *obesity:* height is normal or slightly above average. - ***Endocrine:*** the child is short.

Health Hazards of Obesity:

1- Hypertension & CVD. 2- Hypercholesterolemia.

3- Type II diabetes mellitus. 4- Orthopedic problems, back pain.

Management:

1- Promotion of healthy nutrition and modification of eating behaviour:

- Motivate child and family, all should participate. - Teach the basic aspects of nutrition.
- Avoid unnecessary sweet or fatty foods and soft drinks. - Encourage slow eating.

2- Balanced weight reduction program by experts. **3-** Exercise.

4- Psychological support. **5-** Eat meals as a family in a fixed place & time.

6- Do not skip meals, especially breakfast. **7-** Use small plates.

8- No television during meals. Remove televisions from children's bedrooms; restrict times for television viewing and video games.

8- Schools, eliminate fundraisers with candy and cookie sales.

9- Educate teachers, about basic nutrition and the benefits of physical activity.

10- Educate children from preschool through high school on appropriate diet and lifestyle.

11- Mandate minimum standards for physical education, including 30 - 45 min of strenuous exercise 2-3 times weekly