Dr.saad fawwaz

College of medicine

Al-Anbar University

Malnutrition

Malnutrition ; is a general term for the medical condition caused by an improper or insufficient diet.

Undernutrition : Structural and functional changes due to inadequate intake of nutrients and energy sources.

#Primary Malnutrition: resulting from inadequate food intake.

Secondary Malnutrition: resulting from inadequate absorption ,abnormal metabolism and increased nutrient losses.

Types of undernutrition

--macroneutrient malnutrition ;(protein energy malnutrition PEM) deficiency of protein ,carbohydrates or fat .

--microneutrient malnutrition;deficiency of vitamins and minerals .

-wellcome classification

+ , - Edema → / Wt\age ↓	No edema	edema
60 -80 %	Underweight	kwashiorkor
Less than 60%	marasmus	Marasmic kwashiorkor

-who classification

	Normal	Mild	Moderate	Severe
Weight\height (wasting)	120_90 % +2 _ -1	89_80% -1 _ -2	79_70% -2 _ -3	<70 % < -3

Height \age (stunting)	110 _95 % +2 _ -1	94 _90% -1 _ -2	89 _85% -2 _-3	<80 % < -3
Edema	_ ve	_ ve	_ ve	+ ve

-**Ht\age** ;every 5% of the standard ht\age =1 Z score .

-**Wt\ht** ; every 10% of the standard wt\ht =1 Z score

-**wt\age** ;first 10% of the standard wt\age =1 Z score , then every additional 15% of the standard wt\age =1 Z score .

-Expected wt for of achild less than one year =(age in months +9)\2 .

-from 1 year -12 years = (age in years *3)+7 .

Example ;

Assess the degree of malnutrition of 8 months old male baby weight 5 kg ,length 60 cm and of normal birth wt .

Answer ;

Actual wt =5kg .Expected wt =8.5 kg (according to age).

→Percent of standard wt\age =5\8.5 *100 = 58.8%

Actual ht =60 cm .Expected ht =74 cm(according to age).

→Percent of standard ht\age =60\74 *100= 81 %

→Percent of standard wt \ht = (Percent of standard wt\age)\ (Percent of standard ht\age) =58.8\81=72.5%

100% -72.5% =27.5%

So number of Z score = 27.5 \10 (every 10% of the standard wt\ht =1 Z score) = 2.75 moderate to severe malnutrition .

Diagnosis of malnutrition

#history .

#Physical examination .

#Anthropometric measurements ;

- weight\age ;indicates combined recent and chronic malnutrition .

-height \age(stunting),indicated chronic malnutrition .

-weight\height(wasting),indicates recent malnutrition .

- Skin fold thikness .(subcutaneous fat)

-mid arm circumference (muscle mass)

- OFC , affected only in severe cases ,especially during firt two years of life .

BMI (body mass index); indicates degree of fat excess and obesity = wt (kg)\ht$(m)^2$. under weight <18 , normal 18.5 - 25 , overweight 25 – 30 , obese ; 30 -35 , gross obesity >35

biochemical test ,hb ,serum albumin ,elecrolytes ,enzymes , vitamins and amino acids .

#radiological examinations ,signs of vit C , D deficiency .

Kwashiorkor

Deficiency of protein with relatively adequate energy intake.60 -80 % of the expected wt\age with edema

* Failure of growth ,the muscles are wasted

* Oedema: pitting, bilateral including lower extremities.(may masks the failure to gain weight)

 Hair: becomes fine, straight and often sparse. Children with long straight black hair may show a pale band across the hair, the 'flag sign'.

 Skin: pigmentation, desquamation and ulceration. A severe case may look like an extensive burn. The legs, buttocks and perineum are most frequently involved, but any region may be affected.

 the Liver may be enlarged due to fatty liver .

 Gastrointestinal system: anorexia is usually present and sometimes vomiting.

diarrhea may be due to impaired secretion of digestive enzymes, to intestinal mucosal atrophy or to an intestinal infection.

 Anaemia .

 Associated vitamin deficiencies .

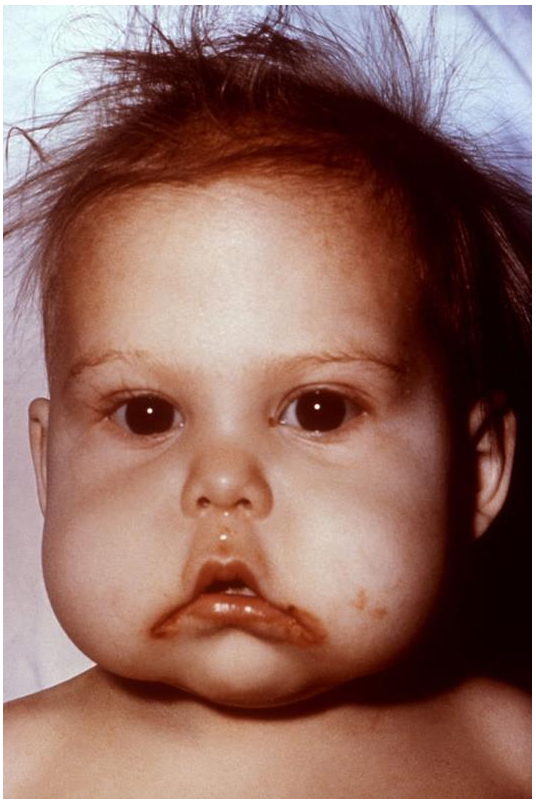
Causes ;

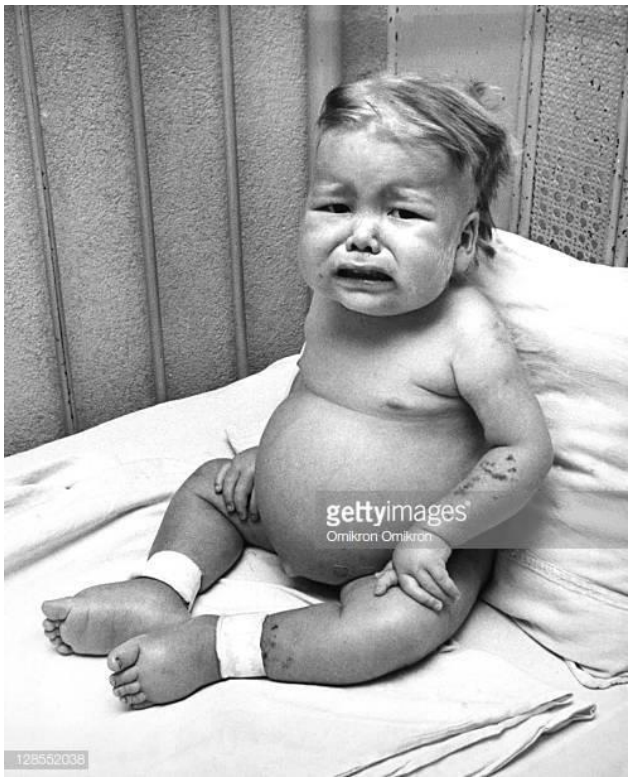
-insufficient intake of protein of good biological value , poverity ,starvation ,enadequate intake

-impared absorption ;,chronic diarrhea ,celiac desease ,obstructive jaundice .

-abnormal protein loss ; nephrotic syndrome ,protein losing enteropathy ,skin burn ,chronic bleeding .

-failure of protein synthesis ; chronic liver desease .





Nutritional marasmus

. is the most common form of PEM, Weight for age < 60% expected with No edema

. Retardation of growth ,Wasting of muscles & loss of subcutaneous fat.

• Appetite is usually preserved or enhanced & the liver is usually normal, with no fatty infiltration.

• Episodes of hypothermia and fasting hypoglycemia are common.

• Emaciated thin, flaccid skin ,fat and muscle tissue grossly reduced, prominence spine and ribs.

• Behaviour: alert and irritable.

• Normal hair.

. Pot belly: the child's abdomen protruded, because the muscles of the abdominal wall are wasted and weak

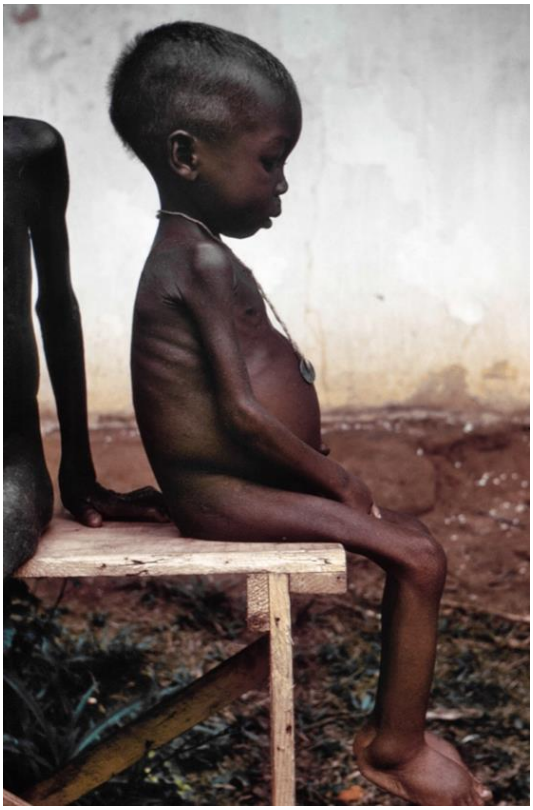
Causes ;

starvation , disturbed maternal child relationship .

#chronic diarrhea and vomiting .

#metabolic abnormalities .

#congenital G.I. tract anomalies ;pyloric stenosis ,esophageal atresia .



TREATMENT ;Phases of Management of severe malnutrition is best divided into three phases.

Initial Phase

Following clinical evaluation, the Initial Phase (days 1-7)..

The principal tasks are:

– To treat or prevent hypoglycemia and hypothermia;

– To treat or prevent dehydration and restore electrolyte balance;

– To start feeding to the child

– To treat infection;

– To identify and treat any other problems, including vitamin deficiency, severe anemia and heart failure

* Severely malnourished patients do not tolerate the usual amounts of dietary protein, fat, and sodium, and require a diet low in these component and in osmolality**, but high in carbohydrate. Start with 80 – 100 kcal\kg\day of actual weight and increase gradually .start with formula 75 (75 kcal\100 ml)** . At each feed the food should be offered by mouth, after which the remainder is given by NG tube.

*avoid giving iron because will increase the liberation of free radicals .

Rehabilitation Phase ;

The second Phase (weeks 2-6)

_ To encourage the child to eat as much as possible. Or (re-initiate breastfeeding) , and to stimulate emotional and physical development;

Started when the appetite and general conditions improved , so , shift to formula 100(100kcal\100ml) .give the other vitamins and add iron .stop the F100 and shift to normal diet when the child catches up 90% of his weight \height .

Follow-Up Phase ;

to ensure that recurrence of malnutrition is prevented

**Direct causes of death:

1. Hypoglycemia2. Hypothermia3. Dehydration 4. Infection5. Severe anemia

**give broad spectrum antibiotics fof 10 days even if the child has no signs of infection

malnourished child has low body potassium and high sodum . so if mildly or moderately dehydrated give **resomal solution(contain lower NA and higher k) .

if severly dehydrated , give ivf .

VITAMIN AND MINERAL DEFICIENCIES

Micronutrients include vitamins and trace elements

WATER- SOLUBLE VITAMINS;

Ascorbic Acid (VIT C)

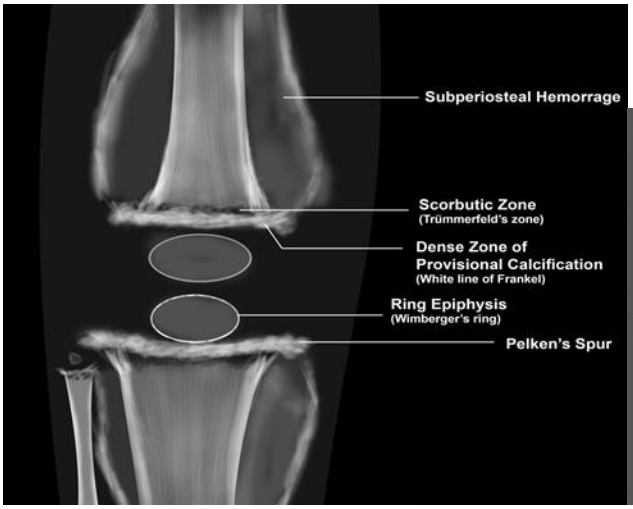
Ascorbic acid accelerates hydroxylation reactions in many biosynthetic reactions, including the formation of collagen AND folate metabolism .

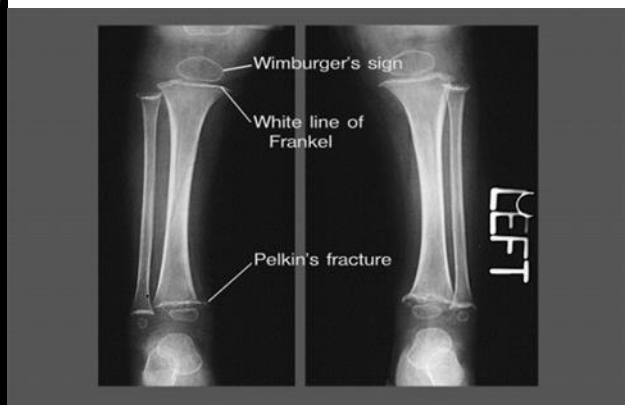
A deficiency of ascorbic acid results in the clinical manifestations of **scurvy**. The disease may occur if infants are fed unsupplemented cow's milk in the first year of life or if the diet is devoid of fruits and vegetables .

Infantile scurvy is manifested by irritability, bone tenderness with swelling, and pseudoparalysis of the legs.. Subperiosteal hemorrhage, bleeding gums and loosening of teeth ,petechiae, bleeding of skin and mucous membranes and Anemia (secondary to ;1- bleeding,2- decreased iron absorption, or 3-abnormal folate metabolism is also seen in chronic scurvy).

X ray shows thin cortex with generalised osteopenia , white metaphysical line (frankel line) with area of bone resorption under that line , subperiosteal hemorrhage then calcification

Treatment ;100-200 mg\day vit c orally or iv , give vit c rich food





B Vitamins; most of B vitamins must be replaced daily since any excess is excreted in the urine**.**

Thiamine (Vitamin B1)

functions as a coenzyme in biochemical reactions related to carbohydrate metabolism. Thiamine is lost during milk pasteurization and sterilization.

deficiency causes **beriberi**

Infantile beriberi occurs between 1 and 4 months of age in breastfed infants whose mothers have a thiamine deficiency , in infants with protein-calorie malnutrition and in infants receiving unsupplemented fluid boiled milk.

Affects mainly the CVS Aand CNS. Anorexia, apathy, vomiting, restlessness, and pallor progress to dyspnea, cyanosis, and death from heart failure. Infants with beriberi have a characteristic aphonic cry(they appear to be crying, but no sound is uttered). Other signs include peripheral neuropathy and paresthesias.

Treatment ;10mg im thiamin \day IM for children ,50 mg \day for adults. continue on thiamin rich diet .and treat with anti failure if heart failure is present .

Riboflavin Vitamin B2 ;

deficiency of riboflavin affects glucose, fatty acid, and amino acid metabolism. Riboflavin is heat stable but destroyed by exposure to light . Ariboflavinosis is characterized by an angular stomatitis; glossitis; cheilosis; seborrheic dermatitis around the nose and mouth; and eye changes that include reduced tearing, photophobia, and cataracts.

Treatment;3-10 mg\day orally +well balanced diet .

Niacin; B3(niacinamide)

Important for the synthesis of (NAD) and (NADP) used for glycolysis and electron transfer . the content of tryptophan in the diet must be considered because tryptophan(found in the egg and milk) is converted in the body to niacin. Niacin is stable in foods and withstands heating and prolonged storage.

Pellagra, or niacin deficiency (Diarrhea , Dysphagia , Dermatitis ,Dementia)

is characterized by weakness, lassitude, dermatitis, photosensitivity, inflamation of mucous membranes, diarrhea, vomiting, dysphagia, and, in severe cases, dementia .

Treatment ;50-300 mg\day niacin orally .if severe 100 mg \day IM .and avoid sunlight .

Vitamin B6

The metabolic functions of vitamin B6 include conversion of tryptophan to niacin and serotonin, metabolic reactions in the brain, carbohydrate metabolism, immune development, and the biosynthesis of heme and prostaglandins. The pyridoxine forms of the vitamin are destroyed by heat;(heat was responsible for vitamin B6 deficiency and seizures in infants fed improperly processed formulas). Goat's milk is deficient in vitamin B6.

Deficiency of vitamin B6 in children results in ; anemia,vomiting, diarrhea, dermatitis , peripheral neuritis, and seizures.

Children receiving isoniazid or penicillamine may require additional vitamin B6 because the drug binds to the vitamin .

Newborns of mothers with history of taking large doses of B6 during pregnancy must receive 10 mg\day orally after birth for severalweeks , to prevent convulsions . Convulsions wheather B6 dependent or deficient ,must be treated by 100 mg pyridoxine IM .& keep on well balanced diet to prevent recurrence .

Folic acid

*Food sources include vegetables, liver, whole grains ;

* Folate deficiency, characterized by hypersegmented neutrophils, macrocytic anemia,neutropenia ,thrombocytopenia and glossitis .

Risk factors for Deficiency ;

* low dietary intake, malabsorption, or drug interactions.

* infants require high ammounts but have scant stores of folate in the newborn period.

* Folate is particularly heat labile. Heat-sterilizing home-prepared formula can decrease the folate content by half.

*goat's milk are low in folate.

*Patients with chronic hemolysis (sickle cell anemia, thalassemia) may require extra folate to avoid deficiency because of the relatively high requirement of the vitamin to support erythropoiesis.

First occurrence and recurrence of neural tube defects are reduced significantly by maternal supplementation during embryogenesis. **Because closure of the neural tube occurs before usual recognition of pregnancy, all women of reproductive age are recommended to have a folate intake of at least 400 µg/day as prophylaxis**.

Vitamin B12(cobalamin)

Vitamin B12 is essential for protein biosynthesis and DNA synthesis .

The cobalamin is efficiently absorbed from the distal ileum.

*Dietary sources of the vitamin are animal products only (meat ,egg , milk) ,so ,Strict vegetarians should take a vitamin B12 supplement.

* Pernicious anemia,Gastric or intestinal resection and small bowel bacterial overgrowth also cause vitamin B12 deficiency. Exclusively breastfed infants ingest adequate vitamin B12(unless the mother is a strict vegetarian without supplementation).

* deficiency of B12 , presented as hypersegmented neutrophils and and megaloblastic anemia.

Vitamin B12 deficiency also causes neurologic manifestations, including depression, peripheral neuropathy, posterior spinal column signs, dementia, and eventual coma

. Maintenance therapy consists of repeated monthly intramuscular injections, although a form of vitamin B12 is administered intranasally.

FAT-SOLUBLE VITAMINS

Fat-soluble vitamins generally have stores in the body, and dietary deficiencies generally develop more slowly than for water-soluble vitamins.

Vitamin A

The clinical manifestations of vitamin A deficiency in humans appear as xerophthalmia. The earliest symptom is night blindness, which is followed by xerosis of the conjunctiva and cornea ,bitot spots (silver gray spots on the bulbar sclera).

* Untreated, xerophthalmia can result in ulceration, necrosis, keratomalacia, and a permanent corneal scar.

* Clinical and subclinical vitamin A deficiencies are associated with increased risk of infection.

Treatment ; for latent deficiency ; 1500 ug \day ..

for xeropthalmia ; 1500 ug \day orally for 5 days then daily IM injection of 7500 ug\day till recovery .

*Hypervitaminosis A also has serious sequelae, including headaches, pseudotumor cerebri, and hepatotoxicity.

Vitamin E

Acts as anti oxidant , and for nucleic acid metabolism

Deficiency Causesed by ;malabsorption ,high unsaturated fatty acid diet ,prematurity .

* progressive sensory and motor neuropathy develops; anemia cerebellar ataxia , the first sign of deficiency is loss of deep tendon reflexes.

Deficient preterm infants at 1 to 2 months of age have hemolytic anemia ,retinopathy ,increased platelet count and adhesiveness so prone for intravascular thrombosis .

All the abnormalities are corrected after oral vitamin E therapy.

Treatment ;for premature15 -25 iu\day vit E orally .

For children 100 -200 Iu \day .

Vitamin K

Plasma factors II (prothrombin), VII, IX, and X in the cascade of blood coagulation factors depend on vitamin K. Other vitamin K–dependent proteins include proteins C, S .

__. **Vitamin K deficiency has been observed in subjects with ;** impaired fat absorption caused by obstructive jaundice, pancreatic insufficiency, celiac disease; prolonged use of antibiotics that change intestinal flora ,more common among breastfed infants .

, occurs in the first few weeks of life. It is rare in infants who receive prophylactic intramuscular vitamin K on the first day of life. Hemorrhagic disease of the newborn usually is marked by generalized ecchymoses, gastrointestinal hemorrhage, or bleeding from a circumcision or umbilical stump; intracranial hemorrhage can occur .

Prophylaxis ;parenteral vitamin K (0.5 to 1 mg) is recommended tobe given to all newborns shortly after birth.

Treatment ;mild deficiency 2 mg \day orally .. if bleeding occure 5mg \day IM .

IF bleeding is severe or the patient has liver disease ,give fresh blood or fresh frozen plasma .

Vitamin D;

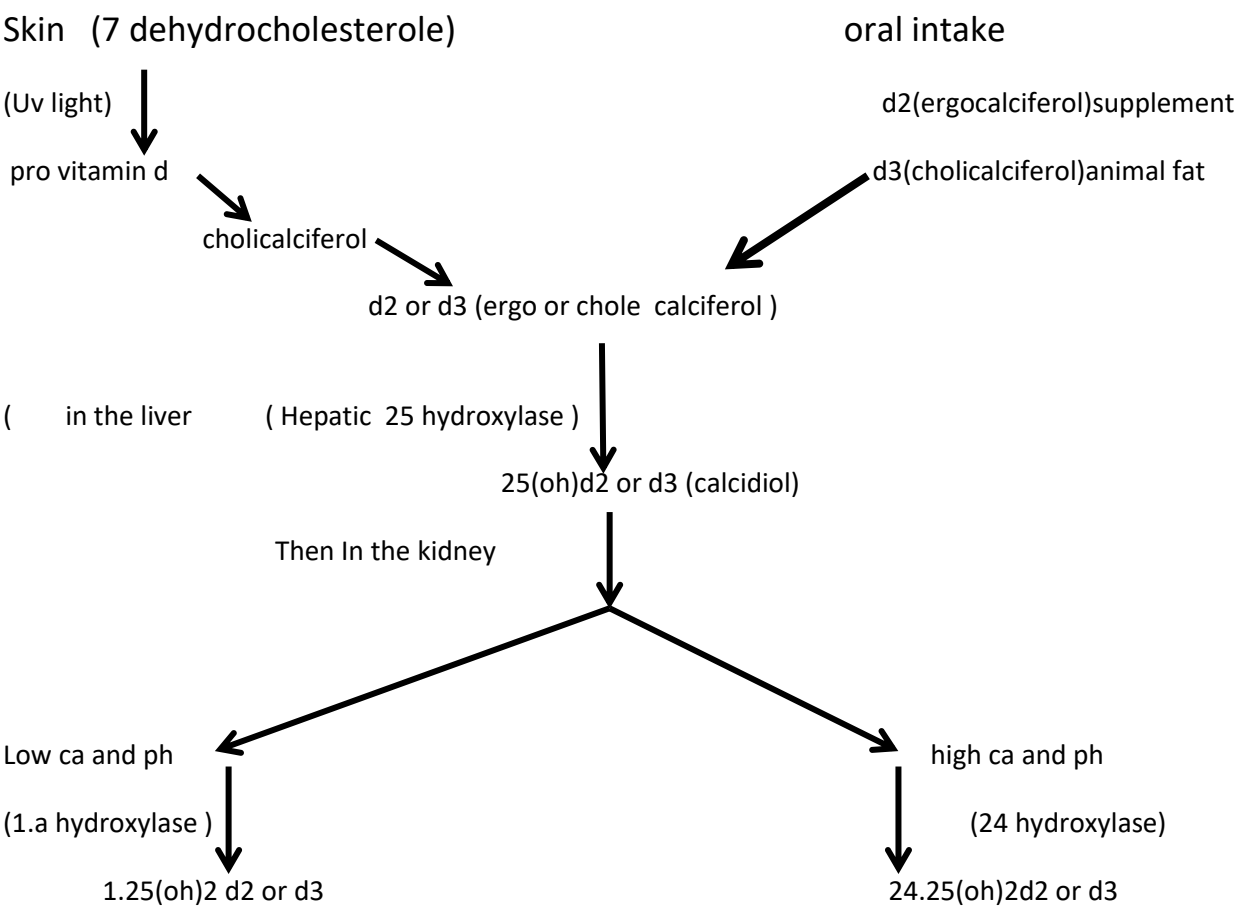
Group of sterols supplied to the body either from the diet (d3,cholecalciferol ,from animals) and (d2 , ergocalciferol , from fungus) or from the skin by direct uv light exposure .

Regulate absorption of calcium and phosphorous from the intestine ,and their deposition in bones .

Sources ; margarin , fish liver oil and by the skin through the sun light exposure to u v light type b of 288 -312 mm wave length .

Daily requirement ; 400 iu/day (10 ug) for infant and children .

Vitamin d deficiency and the interference with its metabolism will cause rickets .



(calcitriol)

*promote ca & ph absorption from intestine

* promote bone mineralization and resorption