It refers to growth below the 3rd or 5th percentile of his or her peers or a change in growth that has crossed two major growth percentiles (e.g. from above the 50th percentile to below the 25th) in a short time, serial measurements are especially important for these children.

2 types:

1) **Non-organic or psychosocial FTT** occurs in a child who is usually <5 yr old; more in developing countries, may be due to poverty, errors in food preparation, child/parent interaction problems, food refusal, child neglect.

2) **Organic FTT** is marked by an underlying medical condition; more in developed countries.

- Organic and non-organic etiologic factors may also occur together, e.g. in neglected child, difficult premature infants, or child infected with HIV.

**Organic FTT**: according to the system:

1- **CNS**: CP.

2- **Renal**: UTI, RTA, RF.

3- **Endocrine**: DM, Diabetes insipidus, G H deficiency, Adrenal insufficiency, Hypothyroidism/hyperthyroidism.

4- **GIT**: Malabsorption, Celiac disease, Milk intolerance, cystic fibrosis, Hirschsprung disease.
5-Cardiac: CHD.

6-Respiratory: severe asthma, bronchiectasis, Bronchopulmonary dysplasia.

7-Infection: TORCH, TB, HIV.

8-Genetic/Chromosomal/Metabolic: Inborn errors of metabolism, Chromosomal disorders.

9-Miscellaneous: Collagen disease, Malignancy.

Another classification, according to pathophysiology:

1- Inadequate nutrient intake
2- Inadequate appetite or inability to eat large amounts: e.g. Psychosocial problems, cardiopulmonary, cleft lip and palate, hypotonia
3- Inadequate nutrient absorption or increased losses: e.g. Malabsorption, chronic diarrhea.
4- Increased nutrient requirements or ineffective utilization: e.g. Hyperthyroidism, malignancy, inborn errors of metabolism.

Clinical Manifestations

The clinical presentation of FTT ranges from failure to meet expected age norms for weight and height to loss of subcutaneous fat, reduced muscle mass, dermatitis, alopecia, recurrent infections, marasmus, kwashiorkor. The degree of FTT is usually calculated as for malnutrition, by standard deviation (wt/age, Ht/age, & wt/Ht).

Diagnosis & treatment

- The treatment requires an understanding of all the elements that contribute to a child's growth, including: parent-child interaction & child's health. Regardless of cause, an appropriate feeding atmosphere at home is important.
- Indications for **hospitalization** include: severe malnutrition, further diagnostic and laboratory evaluation, lack of catch-up growth, and evaluation of the parent-child feeding interaction.

- The **history, physical examination, and observation** of the parent-child interaction usually suggest the diagnosis.

- The **laboratory evaluation** is often not helpful and, therefore, should be used judiciously. A CBC, lead level, and urinalysis are initial screen. Bone age is often helpful in distinguishing family short stature (bone age equivalent to chronological age) from endocrine or nutritional abnormalities (bone age is less than chronological age). Other tests, such as thyroid function studies, should be performed if indicated by the history or physical examination.

- For children with organic FTT, the underlying medical condition should be treated.

- If the history, clinical examination, & investigations fail to find the cause, it is unlikely to be organic failure to thrive. So; Give a full caloric diet & vitamins, & observe the daily weight (mealtimes should be approximately 20-30 min), solid foods should be offered before liquids, environmental distractions should be minimized, and children should eat with other people and not be force fed. The intake of water, juice, and low-calorie beverages should be limited. High-calorie foods, such as peanut butter, whole milk, cheese, dried fruits, & formulas containing more than 20 calories per ounce (PediaSure) are sometimes necessary. Weight gain (30-60 gm/day) in response to adequate caloric feedings usually establishes the diagnosis of psychosocial FTT, so continue the nutritional supplements.

  If after 1 wk no satisfactory increase of weight, or if the child has severe malnutrition, so admit the child to hospital & give full caloric diet (as above) & observe the daily increase in weight, if there is satisfactory gain in weight, so non-organic FTT, so no need for further investigations & continue nutritional supplementations. A nursing plan should include careful charting
of intake, weight, and observations of the mother's feeding style and interactions with the child. Staff should instruct the mother how to improve behaviors that may be deprivational, including instructions on how to hold the baby close during feeding.

If the child failed to gain weight on admission & full diet, so organic FTT is highly possible, start more specific investigations for diagnosis.

- In all the conditions above, follow up is needed to prevent the recurrence.

**MICRONUTRIENT DEFICIENCY**

**VITAMINS**

They are organic substances, required in minute amount. 2 types:

- Water soluble: C, B- complex, folic acid. Toxicity is not common.
- Fat soluble: A, K, E, D. toxicity is common esp. A & D. Vitamins which are produced from the intestinal flora: vit K, biotin, & pantathonic acid.

**VITAMIN C**

It has many functions, e.g. increasing the absorption of iron, but the major role is the formation of normal collagen.

Sources are fruits, tomatoes, & green vegetables.

Breast milk produced by a vitamin C-sufficient mother contains adequate vitamin C, as do all infant formulas. However, low maternal vitamin C intake may result in a low breast milk content of the vitamin. Bovine milk contains very little vitamin C; thus, infants fed bovine milk must receive vitamin C supplements.

Deficiency causes scurvy, while excess causes oxalluria.
Scurvy

The defective formation of collagen causes fragile blood vs. & defective tooth dentin, Common between 6-24 mo, leads to:

- easy bleeding (skin & mucous membrane, hematuria, malena, and orbital or subdural hemorrhages), also subperiosteal hg. (bone tenderness, irritability, pseudo paralysis, frog like posture, edematous swelling of extremities).

- gum hypertrophy & bleeding, losing of teeth.

- anemia.

- A "rosary" at the costochondral junctions and depression of the sternum are other typical features.

- Wound healing is slow.

Diagnosis

X ray: thin cortex, absent trabiculation (ground-glass appearance), white metaphyseal line (Fraenkel line) represents the zone of well-calcified cartilage with area of destruction below.

Low serum & WBC ascorbic acid.

Treatment

Vitamin C orally.

B- COMPLEX VITAMINS

They share in the same sources, so the def. of one is usually associated with def. of the others.

Sources: animal products followed by grains & vegetables.

THIAMIN (B1)

It is a co-enzyme for CHO metabolism & acetylcholine synthesis in CNS and deficiency results in impaired nerve conduction.
Breast milk (from a vitamin B-sufficient mother) and bovine milk are good sources of thiamine

Def. cause beriberi

**Beriberi**

Affect mainly CVS & CNS, presented with congestive heart failure & polyneuropathy: generalized weakness, ptosis, constipation, hoarseness of voice, ataxia, & signs of lower motor neuron lesion.

Treatment

Vitamin B1 orally + anti-failure measures.

If a breast-fed infant develops beriberi, both the mother and child should be treated with thiamine.

**RIBOFLAVIN (B2)**

Important for fat, CHO, & protein metabolism & retinal pigmentation for light adaptation.

**Def. cause:**

- Mucositis, chelitis, angular stomatitis, glossitis, anemia, anorexia, conjunctivitis, photophobia, eye pain, & corneal damage.

Treatment:

Vitamin B2

**NIACIN (NICOTINAMIDE)**

Niacin forms part of two cofactors, NAD & NADP.

Niacin deficiency cause pellagra

**Pellagra**

The early symptoms of pellagra are vague: anorexia, lassitude, weakness. After a long period of deficiency, the classic triad (3D) of dermatitis, diarrhea, and dementia appears. Dermatitis, the most characteristic manifestation of pellagra, may be elicited by intense
sunlight. The lesions first appear as symmetric areas of erythema on exposed surfaces, resembling sunburn, then it progress to vesicles, crusts, & desquamation. The lesions are usually sharply demarcated from the healthy skin around them, on the hands often have the appearance of a glove & on the foot and leg (pellagrous boot).

It occurs chiefly in countries where corn (maize) is a basic foodstuff.

**Treatment**

50–300 mg/ day of Niacin. Sun exposure should be avoided during the active phase of pellagra, and the skin lesions may be covered with soothing applications.

**VITAMIN B₆ (PYRIDOXINE)**

It is a co-enzyme for protein, fat, & CHO metabolism, important for CNS function (serotonin & GABA formation), and in the synthesis of heme. It has anti-emetic properties.

The pyridoxine content of human milk and infant formulas is adequate. Pyridoxine antagonists (e.g., isoniazid used in the treatment of tuberculosis) & pregnancy increase the requirements for pyridoxine.

Many clinical disturbances caused by vitamin B₆ deficiency have been described in humans: vitamin B₆ dependence syndromes (including vitamin B₆-dependent convulsions with abnormal EEG, a vitamin B₆-responsive anemia), peripheral neuritis, dermatitis, & others, e.g. cheilitis, glossitis, facial seborrhea

*Vitamin B₆ dependence syndromes* result from errors in enzyme structure or function, and patients with these syndromes respond to very large amounts of pyridoxine. The use of high doses during pregnancy has been implicated in some cases of transient vitamin B6 dependent syndrome in infants, so for those infant & patients on large doses for long periods of time, withdrawal of vitamin B6 should be gradual.
Diagnosis & Treatment

All infants with seizures should be suspected of having vitamin B₆ deficiency or dependence. If more common causes of infantile seizures (e.g., hypocalcaemia, hypoglycemia, infection) are eliminated, 100 mg of pyridoxine should be injected. Newborn of a mother took large amount of pyridoxine during pregnancy must receive 10 mg/day orally after birth for several wks to prevent convulsions.

VITAMIN B12

Important for DNA synthesis.

It needs intrinsic factor (IF), which is secreted from the stomach, for its absorption at the terminal ileum, then carried in the serum by transcobalamin to the liver.

Sources: animal product & low in vegetable.

Body store is sufficient for 3-5 yr.

Causes of deficiency

Extreme vegans (vegetarian), juvenile pernicious anemia (absent IF), problems of terminal ileum (surgical resection, tuberculous & regional enteritis, diphyllobothrium latum infestation, malabsorption), & congenital transcobalamin deficiency.

Clinical manifestations

General: Irritability, anorexia, diarrhea, FTT.

Hematological: megaloblastic anemia, thrombocytopenia, & neutropenia with polysegmented nucleus.

Neurological: ataxia, paraesthesia, hypotonia, Babenski sign, clonus, & coma.

Diagnosis
Low serum B12, normal Folate, +ve schilling test.

**Treatment**

Administration of a minidose (1–5μg/day) may be used as a therapeutic test when the diagnosis of vitamin B$_{12}$ deficiency is in doubt. If there is evidence of neurologic involvement, 1 mg should be injected intramuscularly daily for at least 2 wk.

**FOLIC ACID**

Folate coenzymes for synthesis of DNA and purine. Maternal folic acid status is known to be protective against neural tube defects

Limited body store, so depleted within 2-3 mo.

**Causes of deficiency:**

Deficient diet (goat milk)

Increased requirement (hemolytic anemia, prematurity, pregnancy, & infection)

Malabsorption syndrome.

Drug interaction (anticonvulsants, Methotrexate).

**Clinical manifestations:**

Irritability, anorexia, FTT, diarrhea, megaloblastic anemia, thrombocytopenia, & neutropenia with polysegmented nucleus.

**Diagnosis:**

Low serum & RBC Folate with normal vit B12.

**Treatment**

0.5 – 1 mg/ day folic acid orally or IM for 3-4 wks. If the specific diagnosis is in doubt, smaller doses (0.1 mg/day) may be used for 1 week as a diagnostic test, because a hematologic response can be expected within 72 hr. Doses of folate >0.1 mg can correct the anemia of vitamin B$_{12}$ deficiency but may aggravate any associated neurologic
abnormalities. Maintenance therapy with a multivitamin (containing 0.2 mg of folate) is adequate.

**FAT- SOLUBLE VITAMINS**

In general, they are deficient in cases of fat malabsorption.

**VITAMIN A**

Important for synthesis of rhodopsin & iodopsin, & for skin & mucous membrane integrity.

Sources: yellow & green vegetables, fruits, eggs, butter, liver.

The liver content of vitamin A is low at birth but is rapidly augmented by the large amounts in colostrum and breast milk as well as infant formulas.

Overdose may lead to toxicity (pseudotumour cerebri: benign increase of intracranial pressure) & in pregnancy, to congenital anomalies.

**Deficiency** result in:

**Eye:** 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, then photophobia,

Later, corneal keratinization & cloudness, then xerophthalmia: dry, scaly layers of cells, then infection occur makin the cornea wrinkled; then it degenerates irreversibly (keratomalacia), resulting in blindness.

Dry, silver-gray plaques may appear on the bulbar conjunctiva (Bitot spots), with follicular hyperkeratosis and photophobia.

Its def is highly correlated with measles & its severity & - mortality rate.

**Skin:** follicular hyperkeratosis with dry scaly skin.

**Mucous membrane:** epithelial changes lead to:

Bronchial obstruction with recurrent chest infection.
Infection of urinary tract & salivary gland.

**Brain:** Increased intracranial pressure with wide separation of cranial bones at the sutures.

**Treatment**

For latent deficiency: 1500 μg / day vit. A.

For xerophthalmia or other major complications: 1500 μg / kg/ day orally for 5 days, then daily IM injection of 7500 μg till recovery, with eye care by ophthalmologist. Morbidity and mortality rates from viral infections such as measles may be lower in non-deficient children who are given daily doses of 1,500-3,000 μg of vitamin A.

**VITAMIN K**

Required for production of factors (10, 9, 7, 2) for blood clotting, protein Z & M (stimulate platelet activity), for protein C & S (anticoagulant).

It presents in natural form (K1 from food), K2 from intestinal flora, & in synthetic form (Large doses of may cause hyperbilirubinemia & kernicterus in neonate & in patient with G6PD def).

**Deficiency**

Causes:

Breast feeding; prolong use of AB that kills the bacterial flora, fat malabsorption, & chronic diarrhea.

Diseases of the liver may limit synthesis of prothrombin. Hypoprothrombinemia from this cause usually does not respond to administration of vitamin K.

It will leads to hemorrhagic disease of newborn & bleeding tendency at any age.

**Treatment**

For mild def.: 1- 2 mg orally every 24 hr should be given.

If bleeding occurs, 5 mg IM every 24 hr should be given.
If bleeding is severe, or the patient has liver diseases give fresh blood or fresh frozen plasma.

Hypoprothrombinemia that is corrected by vitamin K administration establishes the diagnosis.

**VITAMIN E**

It acts anti oxidant & in nucleic acid metabolism.

Sources: seeds, nut, green leafy vegetables.

**Deficiency:**

Causes: fat malabsorption, high unsaturated fatty acid diet in premature baby (0.7 mg/g of unsaturated fat in the diet appears adequate).

Leads to:

- muscle weakness
- degenerative, potentially reversible, neurologic syndrome consisting of cerebellar ataxia, motor & sensory neuropathy.
- In premature: hemolytic anemia, edema, & retinopathy of prematurity.

**Treatment:**

For premature: 15-25 IU /day, orally.

For children: 100-200 IU/day, orally.