LOCOMOTOR SYSTEM IN CHILDREN

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Bone system

1. ***organic part*** – collagen type I (glicin, prolin, hydroxiprolin), proteoglicans, osteocalcin, osteonectin, lipoprotein, sialoprotein, phosphoprotein, collagen type II
2. ***mineral part*** – Ca, phosphorus
3. ***bone cells*** – osteoblasts, osteoclasts, osteocytes

Skeleton and its functions

- gives the form and support to the body
- represents the storage of easy mobilizing calcium (97%)
- some elements of skeleton protect the body from external and internal forces.
- the bones offer the attaching surfaces for muscles and represents the movement levers.
- the dynamic agent of locomotion is represented by skeletal muscle.

Function of bone tissue

- Protection
- Shape
- Blood production
- Mineral storage
- Fat Storage
- Movement
- Acid-base balance
- Detoxification
- Sound transduction

- ***Protection*** - Bones can serve to protect internal organs, such as the skull protecting the brain or the ribs protecting the heart and lungs.
- ***Shape*** — Bones provide a frame to keep the body supported.
- ***Blood production*** — The marrow, located within the medullary cavity of long bones and interstices of cancellous bone, produces blood cells in a process called haematopoiesis.
- ***Mineral storage*** — Bones act as reserves of minerals important for the body, most notably calcium and phosphorus.

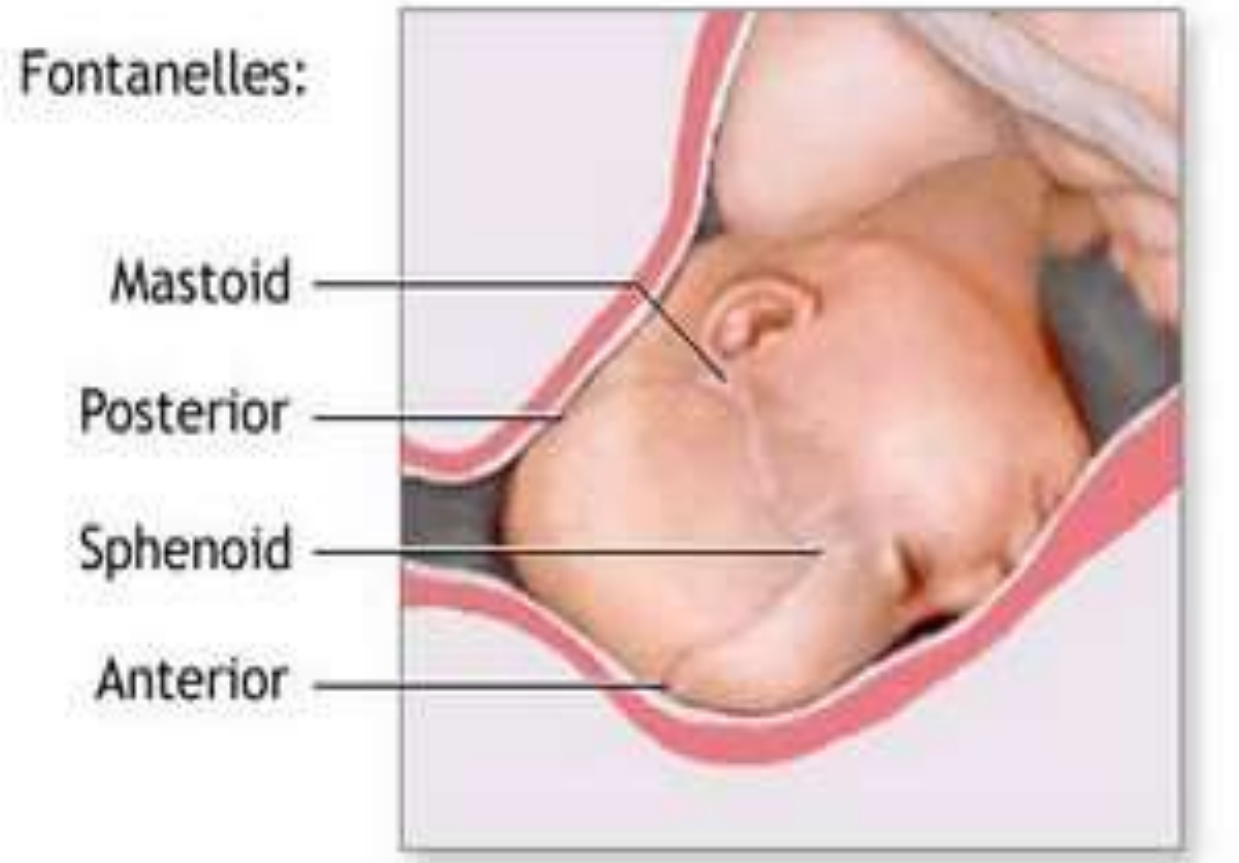
- ***Fat Storage*** — The yellow bone marrow acts as a storage reserve of fatty acids
- ***Movement*** — Bones, skeletal muscles, tendons, ligaments and joints function together to generate and transfer forces so that individual body parts or the whole body can be manipulated in three-dimensional space. The interaction between bone and muscle is studied in biomechanics.
- ***Acid-base balance*** — Bone buffers the blood against excessive pH changes by absorbing or releasing alkaline salts.

- ***Detoxification*** — Bone tissues can also store heavy metals and other foreign elements, removing them from the blood and reducing their effects on other tissues. These can later be gradually released for excretion.
- ***Sound transduction*** — Bones are important in the mechanical aspect of hearing.

Anatomophysiological peculiarities of osseous (bone) tissue in children

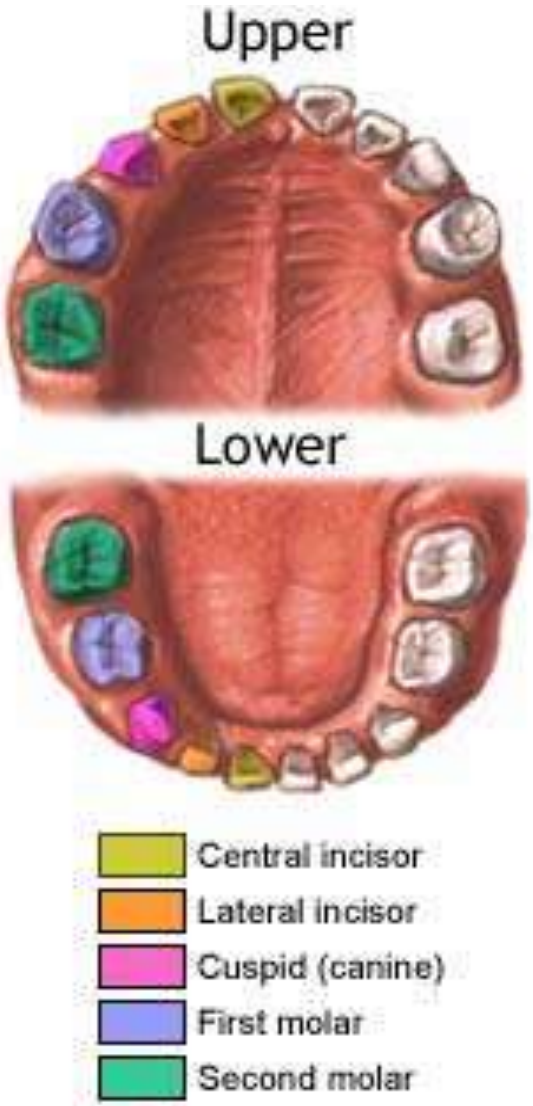
- The energy of growth and bone regeneration is high
- Higher content of water and lesser percentage of solid constituents
- Comparatively greater softness and resilience to pressure bending, and a lesser tendency to fractures
- Fibrous structure of bone in children (greater extent in the fetus) and laminar structure in the adult
- Greater vascularisation of children's bones
- Periosteum is thick, with a particularly well-defined internal layer
- Presence growing zone of cartilage in long bones

Fontanelles



A.D.A.M.

Deciduous (milk) teeth

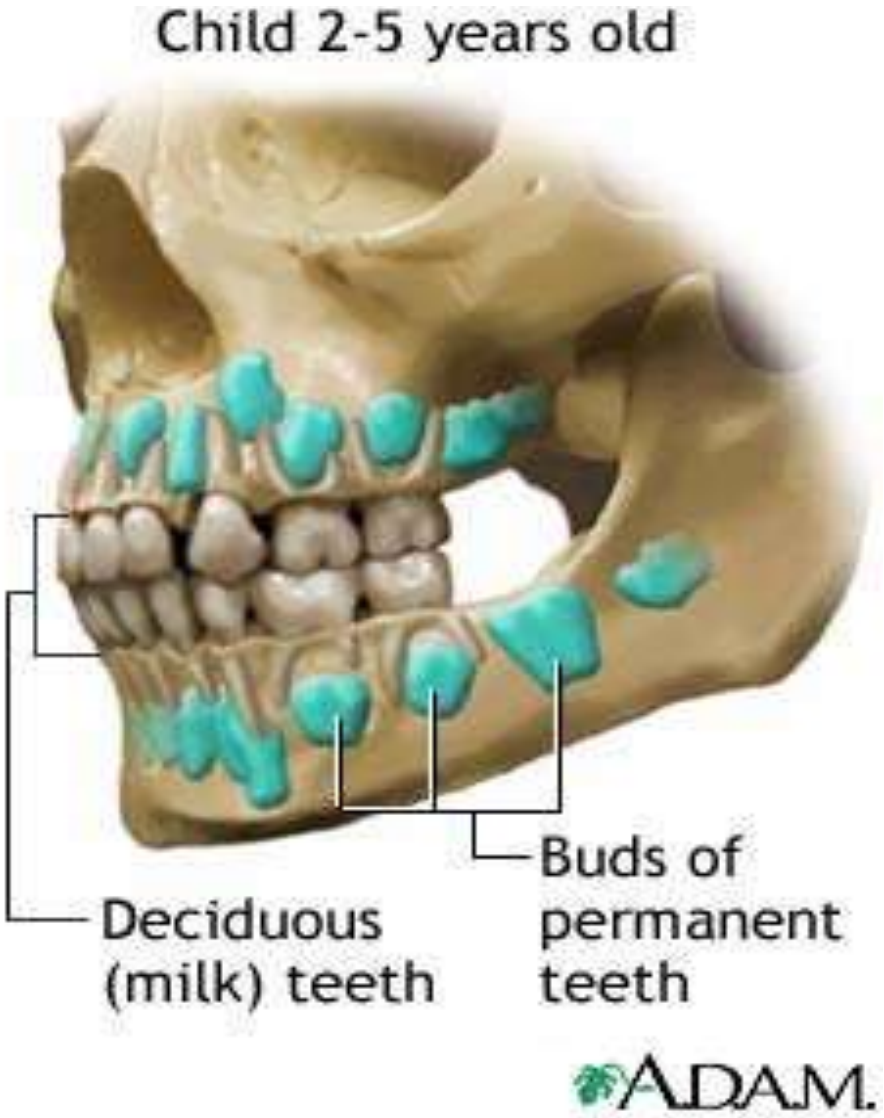


X=n-4

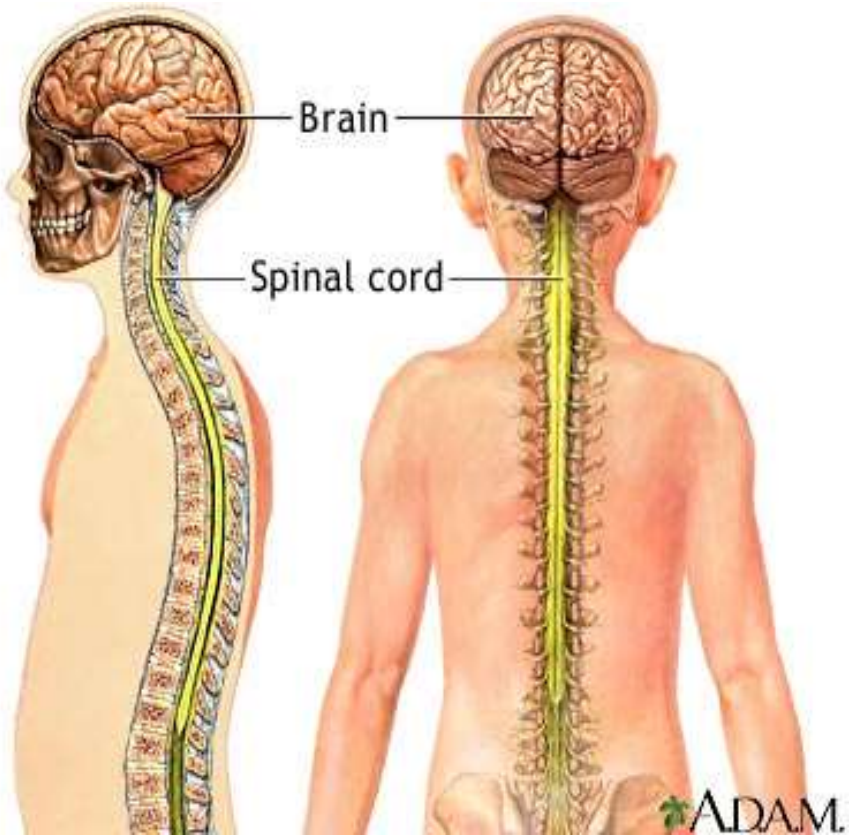
UPPER	ERUPTS BY	LOWER	ERUPTS BY
Central incisor	8-10 Mo	Central incisor	6-9 Mo
Lateral incisor	8-10 Mo	Lateral incisor	15-21 Mo
Canine (Cuspid)	16-20 Mo	Canine (Cuspid)	15-21 Mo
First molar	15-21 Mo	First molar	15-21 Mo
Second molar	20-24 Mo	Second molar	20-24 Mo

Permanent teeth

X= 4n-20



The physiological curvatures of spinal (or vertebral) column

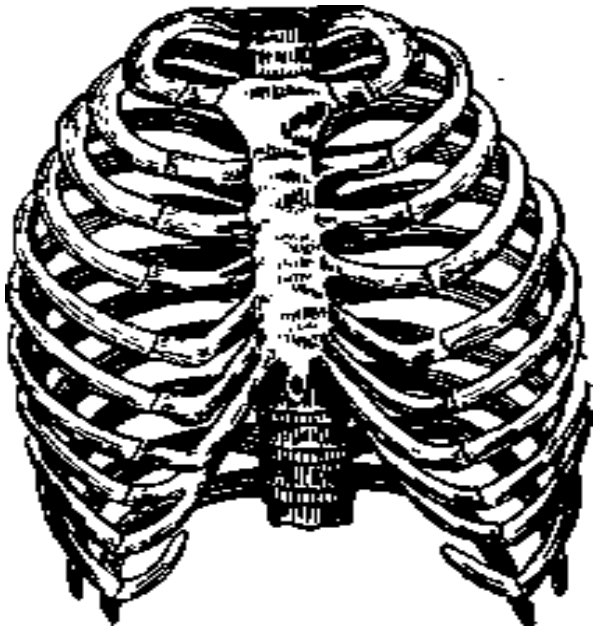


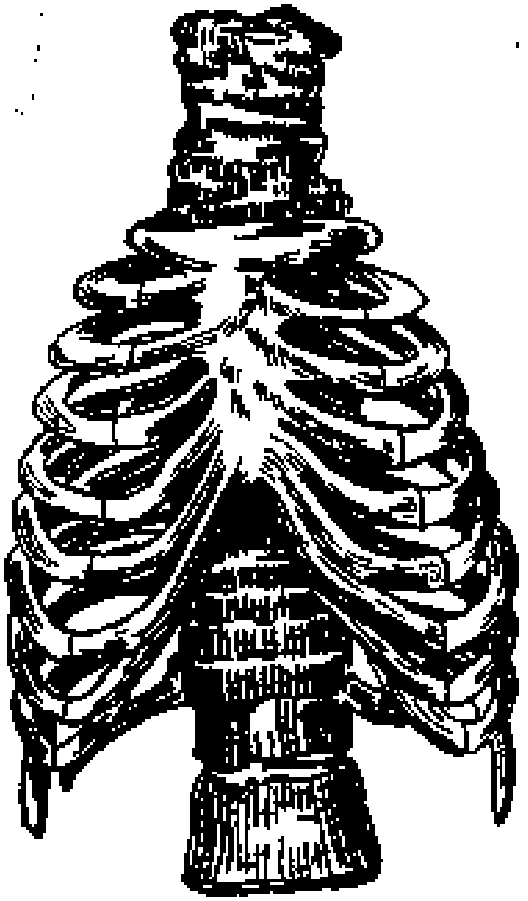
cervical lordosis

thoracic kyphosis

lumbar lordosis

Chests of children and adults





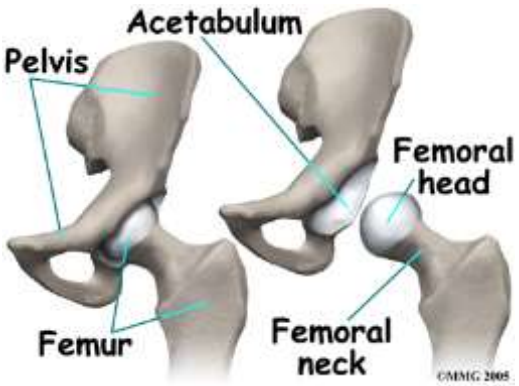
Paraclinical methods of investigation

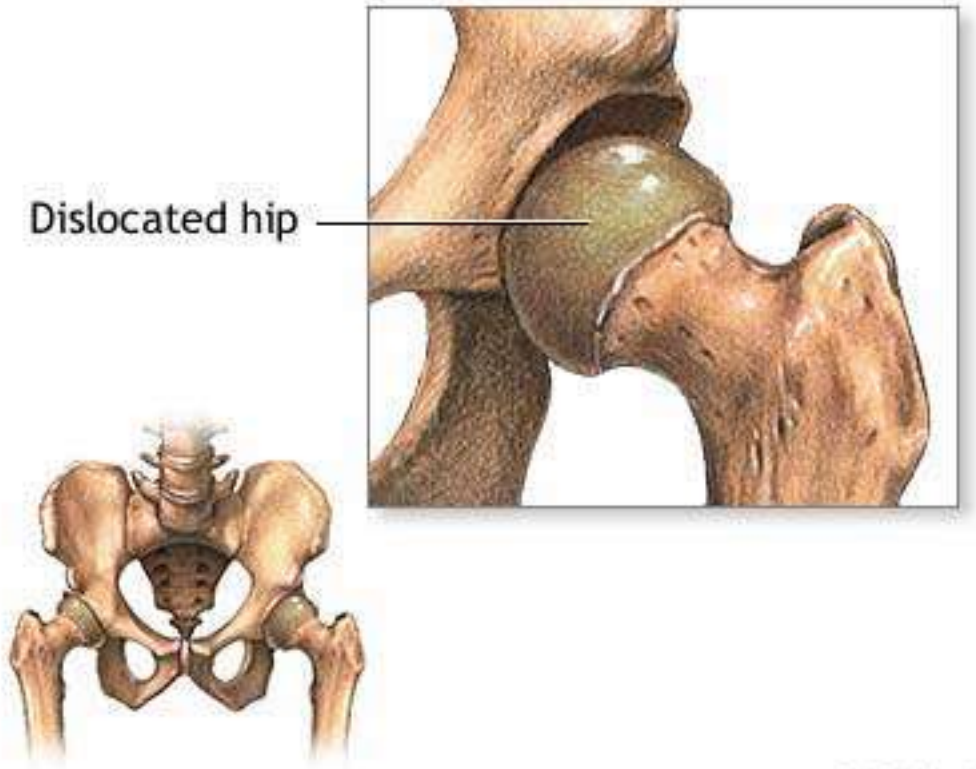
- Laboratory Tests: Ca, P, Mg, total protein, vit. D, CK-MM, LDH, aldolase, PTH, alcaline phosphatase
- X-ray evaluations
- Arthroscopy
- Computed Tomography
- Scheletal Scintigraphy
- MRI
- Biopsy
- USG of joins
- Osteodensitometry

Congenital dysplasia of the hip –

is the hypoplasia of an acetabulum, reduction its depth

and inadequate size of the femoral head





Clinical sings of Congenital dysplasia of the hip

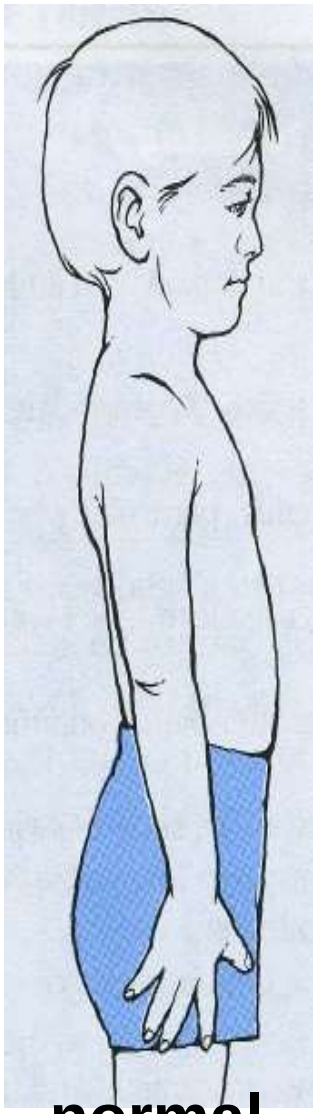
- Asymmetry of thigh skin folds
- Shortness of one of the extremities
- Knee joints are situated at different lev
- The level of abduction of the leg less th
- Ortalani's symptom – a click of

entrance will be felt as femoral head slips acetabulum

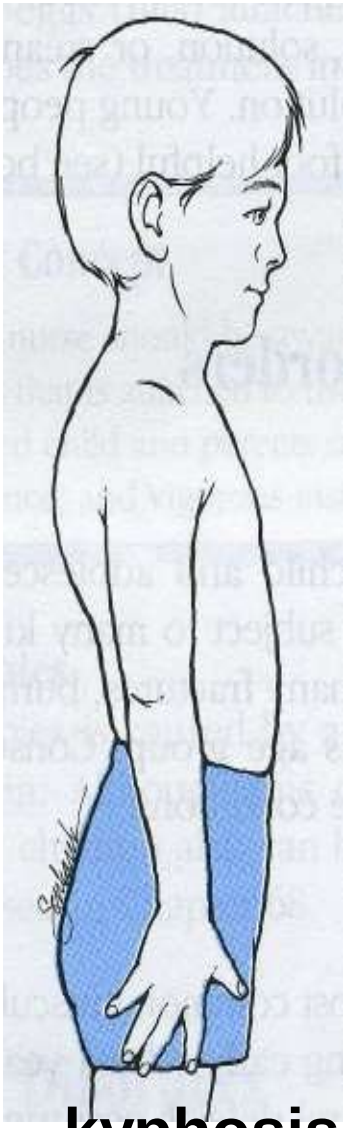
(when the thigh is gradually adducted)

- Late sign – "goose" gait (limping at walking)

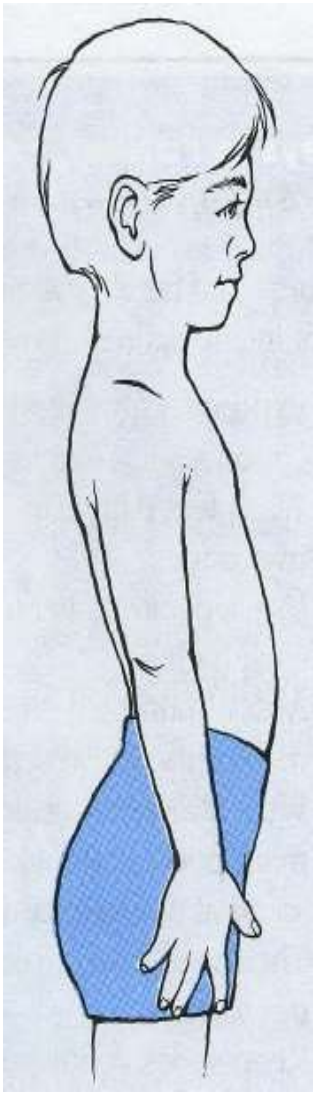
Semeiology of spinal cord affections



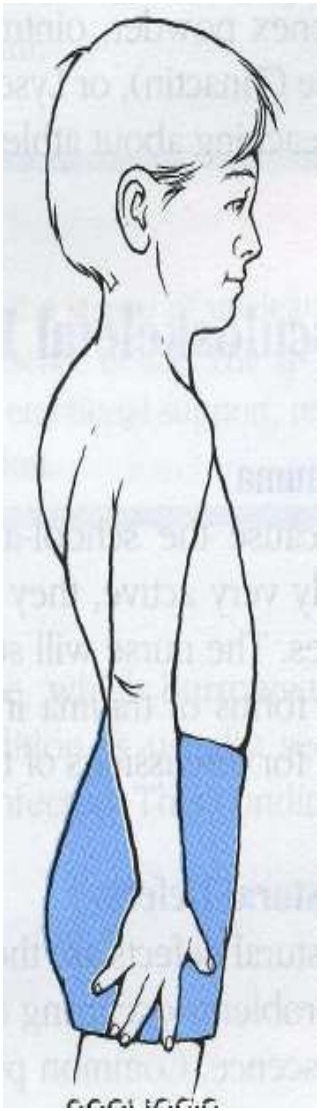
normal



kyphosis



lordosis



scoliosis

RICKETS IN CHILDREN

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Definition

Rickets is consequence of the vitamin D deficit and may occur due to calcium and phosphorus metabolic disorders.

- Blood analysis shows hypocalcemia and hypophosphatemia.
- Histology - Failure in mineralisation of the bone and cartilaginous tissues .
- Clinical - manifests as skeletal growth disorder.

Hystory

- Rickets (from Greek word meaning spinal column) was known since the first years of the human generation. It is described by Soran Efess (A.D) and by Galen (134-211 A.D).
- It is described in detail by a British anatomist and orthopedician, Glisson in 1650.

Incidence

- Rickets is frequently in premature children and the children fed only wheat floor.
- In Moldova diagnosis was confirm in 35.5%, X-Ray - 21.5%.

Physical and metabolic properties and food sources of vitamin D

Names and synonyms of Vitamin D	Characteristics	Biochemical action	Effects of deficiency	Effects of excess	Sources
Vitamin D3 (3-cholecalciferol), which is synthesized in the skin, **vitamin D2** (from plants or yeast) are biologically equivalent; 1 µg = 40 IU vitamin D.	Fat-soluble, stable to heat, acid, alkali, and oxidation; bile necessary for absorption; hydroxylation in the liver and kidney necessary for biologic activity	Necessary for GI absorption of calcium; increases absorption of phosphate; direct actions on bone, including mediating resorption	Rickets in growing children; osteomalacia; hypocalcemia can cause tetany and seizures	Hypercalcemia, which can cause emesis, anorexia, pancreatitis, hypertension, arrhythmias, CNS effects, polyuria, nephrolithiasis, renal failure	Exposure to sunlight (UV light); fish oils, fatty fish, egg yolks, and vitamin D–fortified formula, milk, cereals, bread

Causes of Rickets

Vitamin D Disorders

- Nutritional vitamin D deficiency
- Congenital vitamin D deficiency
- Secondary vitamin D deficiency
- Malabsorption
- Increased degradation
- Decreased liver 25-hydroxylase
- Vitamin D–dependent rickets types 1A and 1B
- Vitamin D–dependent rickets types 2A and 2B
- Chronic kidney disease

Causes of Rickets

Calcium Deficiency

- Low intake
- Diet
- Premature infants (rickets of prematurity)
- Malabsorption
- Primary disease
- Dietary inhibitors of calcium absorption

Phosphorus Deficiency

- Inadequate intake
- Premature infants (rickets of prematurity)
- Aluminum-containing antacids

Causes of Rickets

Renal Losses

- X-linked hypophosphatemic rickets
- Autosomal dominant hypophosphatemic rickets
- Autosomal recessive hypophosphatemic rickets types 1 and 2
- Hereditary hypophosphatemic rickets with hypercalciuria Overproduction of fibroblast growth factor-23
- Tumor-induced rickets
- McCune-Albright syndrome; Epidermal nevus syndrome
- Neurofibromatosis
- Fanconi syndrome
- Dent disease; Distal renal tubular acidosis

Risk Factors for Nutritional Rickets

Maternal Factors

Vitamin D deficiency

- Dark skin pigmentation
- Full body clothing cover
- High latitude during winter/spring season
- Other causes of restricted sun (UVB) exposure, e.g., predominant indoor living, disability, pollution, cloud cover
- Low–vitamin D diet

Low-calcium diet

- Poverty, malnutrition, special diets

Risk Factors for Nutritional Rickets

Infant/Childhood Factors

Neonatal vitamin D deficiency secondary to maternal deficiency/vitamin D deficiency

- Lack of infant supplementation with vitamin D
- Prolonged breastfeeding without appropriate complementary feeding from 6 mo
- High latitude during winter/spring season
- Dark skin pigmentation and/or restricted sun (UVB) exposure, e.g., predominant indoor living, disability, pollution, cloud cover
- Low–vitamin D diet

Low-calcium diet

- Poverty, malnutrition, special diets

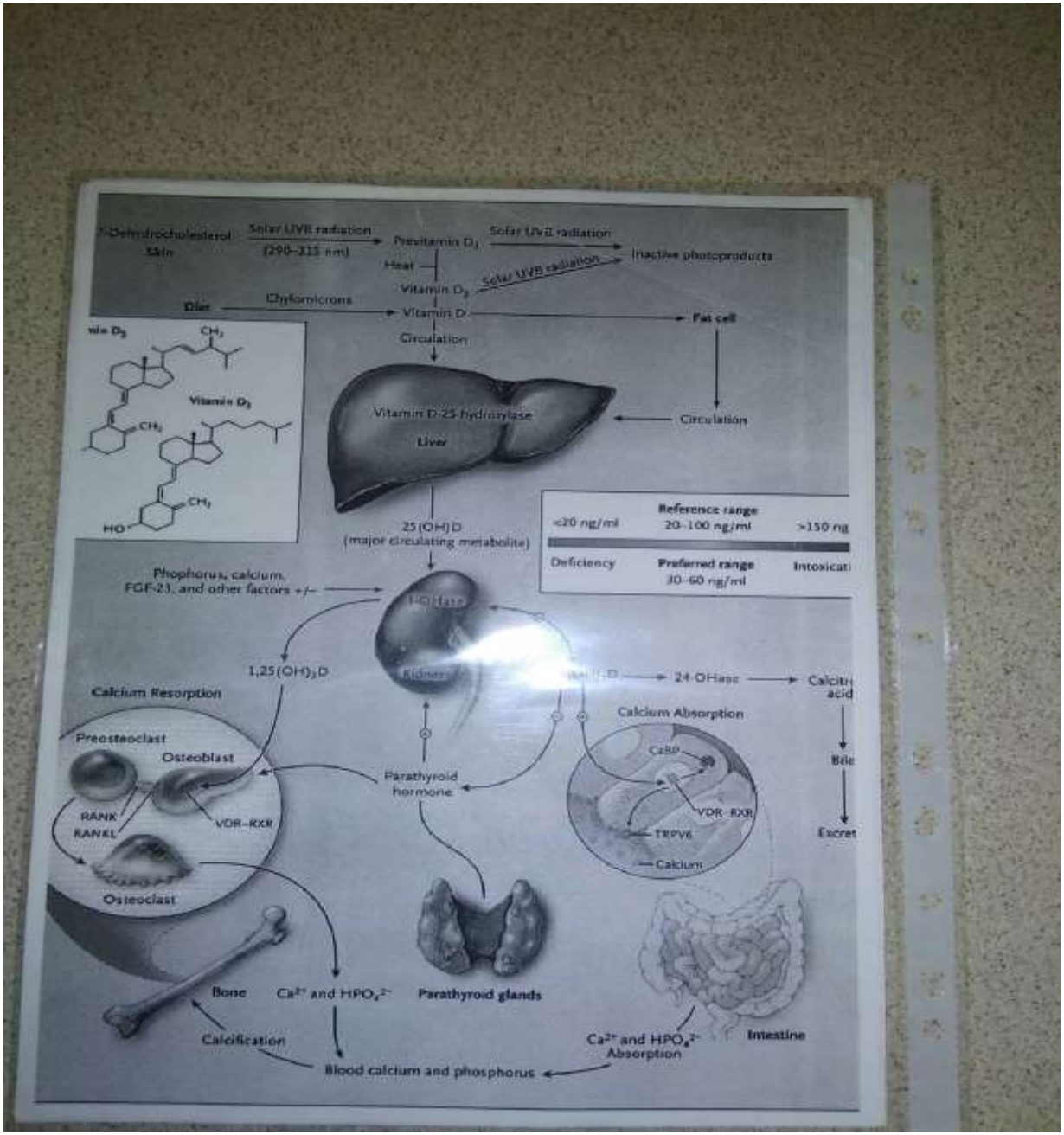
Risk factors

- Living in northern latitudes (>30°);
- Black children - inadequate skin penetration of sunlight;
- Decreased exposure to sunlight (polluted geographical areas, humid climate);
- Maternal vitamin D deficiency;
- Diets low in calcium, phosphorus and vitamin D, e.g. exclusive breast-feeding into late infancy, toddlers on unsupervised “dairy-free” diets;
- Macrobiotic, strict vegan diets;
- Phytates of cereals, stearic and palmitic acids decrease calcium absorption;
- Prolonged parenteral nutrition in infancy with an inadequate supply of intravenous calcium and phosphate;

- Intestinal malabsorption, defective production of 25(OH)D3 – liver disease. Increased metabolism of 25(OH)D3 – enzyme induction by anticonvulsants.

Defective production of 1,25(OH)2D3

- Hereditary type I vitamin D-resistant (or dependent) rickets (mutation which abolishes activity of renal hydroxylase);
- Familial (X-linked) hypophosphataemic rickets – renal tubular defect in phosphate transport;
- Chronic renal disease;
- Fanconi syndrome (renal loss of phosphate)
- Target organ resistance to 1,25(OH)2D3 - hereditary vitamin D-dependent rickets type II (due to mutations in vitamin D receptor gene).


7-Dehydrocholesterol
Skin
Solar UVB radiation
(290–315 nm)
Previtamin D_3
Solar UVB radiation
Inactive photoproducts
Heat
Vitamin D_3
Solar UVB radiation
Diet
Chylomicrons
Vitamin D
Fat cell
Circulation
Vitamin D_3
HO
Vitamin D-25-hydroxylase
Liver
Circulation
25(OH)D
(major circulating metabolite)
Reference range
<20 ng/ml
20–100 ng/ml
>150 ng
Deficiency
Preferred range
30–60 ng/ml
Intoxicati
Phosphorus, calcium,
FGF-23, and other factors +/–
1-OHase
Kidney
$1,25(OH)_2D$
24-OHase
Calcium Resorption
Calcium Absorption
Preosteoclast
Osteoblast
RANK
RANKL
VDR–RXR
Parathyroid
hormone
CaBP
VDR–RXR
TRPV6
Calcium
Excret
Osteoclast
Bone
Ca^{2+} and HPO_4^{2-}
Parathyroid glands
Intestine
Calcification
Ca^{2+} and HPO_4^{2-}
Absorption
Blood calcium and phosphorus

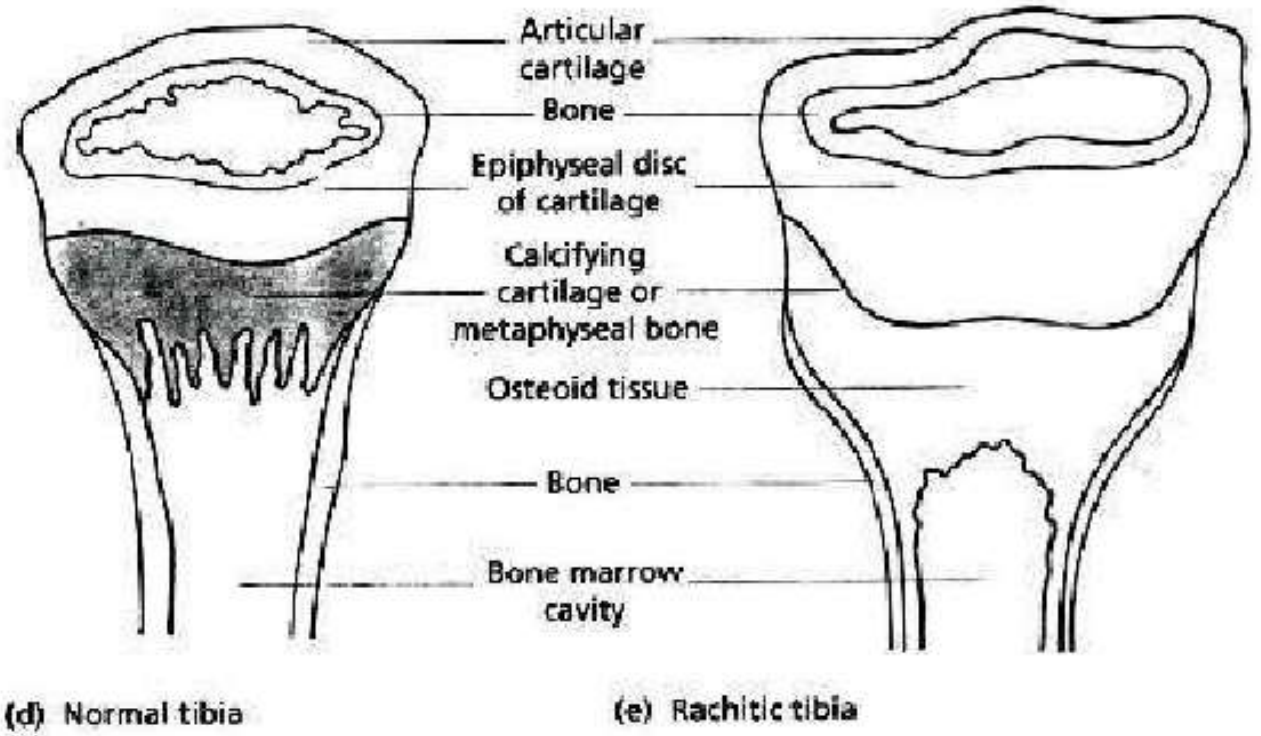
Etiology

- Rickets is due to partial deficiency, rarely complete deficiency of vitamin D.
- Vitamin D exist in 2 forms in the human body.
- Vitamin D2, exogenous form (calciferol), from ergosterol in the food
- Vitamin D3, endogenous form (cholecalciferol or provitamin stage 7-dehydrocholecalciferol, naturally present in human skin), activated by UV rays of 296-310nm wave length.
- Natural alimentation does not supply the daily requirement of 400-500IU of vitamin D in a baby.
- Breast milk contains 30-50 IU/liter, cow's milk 20-30 IU/l, egg yolk contains 20-50 IU/10g.
- 80% of the vitamin D is absorbed in the small intestine in the present of normal biliary secretion.
- Vitamin D reaches the blood through thoracic duct along with chilomicrons.

Pathogenesis of rickets

In Ricket cartilage cells fail to complete their normal cycle of proliferation and degeneration;

subsequent failure of capillary penetration occurs in a patchy manner(Fig)



Calcium regulation in the blood

- Vitamin D2 in the food (exogenous) + vitamin D3 (skin, endogenous) =>liver microsomal hydroxylate =>25(OH)D3
- In the renal cortical cells => activated from 1alpha-hydroxilase in 3 forms:

  24,25(OH)2 D3; 1,24,25 (OH)2 D3; 1,25 (OH)2 D3 end product considered a hormone.
- In placental macrophage of pregnancy women are present 1,25(OH)2 D3

Functions of vitamin D
Intestine

- Increases calcium binding protein
- Active transport in the jejunal cells
- Phosphorus ions absorption through specific phosphate carrier
- Alkaline phosphatase (AP) synthesis
- ATP-ase sensibility to calcium ions

Bones

- Mineralization of the bone and osteoblasts differentiation in presence of adequate calcium and phosphorus
- Deposition and reabsorption of calcium and phosphorus, normal calcification
- Skeletal growth and mineralization involve vitamin D-PTH-endocrine axis, growth hormone via somatomedins, thyroid hormones, insulin, androgens and estrogens in puberty

Kidney

- 1,25(OH)2D3 increase tubular reabsorption of calcium and phosphorus
- In rickets PTH blocks phosphorus reabsorption in kidney, elevated serum phosphatase due to increse osteoblastic activity
- Hypophosphatemia blocks PTH secretion and promotes 1,25(OH)2D3 synthesis, the most active metabolite of vitamin D

Muscles

- Vitamin D increase the muscular protein and the ATP in myocyts
- Improve tonicity and the normal contraction of the muscles

Parathyroid glands

- 1,25(OH)2D3 has direct feedback to PTH synthesis
- Low plasma calcium=> PTH secretion restore Ca from bone demineralization
- Secretion of PTH stimulate synthesis of 1,25(OH)2D3, increase calcium intestinal absorption, renal calcium reabsorption
- Calcitonin (secretion of C cells of thyroid gland) increase bone calcium deposition

Other effects of vitamin D

- Cellular metabolism: citric acid oxidation
- Formation of soluble complex of citrate and Ca in the blood
- Skin differentiations in the local treatment of Psoriasis
- Pulmonary differentiation (increases the surfactant in preterm infants)
- Immunomodulatory action in autoimmune disorders

Biochemical stages of rickets

Stage 1:

Low serum Ca level,

normal serum P;

normal serum PTH,

little raise AP,

Ca and P tubular reabsorption are normal,

no amino acid loss in the urine.

Biochemical stages of rickets

Stage 2: Raised PTH in the serum,

serum Ca is normalized by bone demineralization.

Change in the ratio of Ca:P (N=2:1), in this stage become 3:1 or 4:1,

high serum AP.

Raised Ca tubular reabsorption and decrease phosphate tubular reabsorption.

As a result => hyper-aminoaciduria. Phosphates are lost in the urine, alkaline pH.

X-ray findings: Osteoporosis and metaphyseal-epiphyseal changes.

Biochemical stages of rickets

Stage 3: Severe deficiency of vit. D for a long duration.

Hypocalcemia,

hypophosphatemia,

serum elevated of AP, PTH;

hyperaminoaciduria,

Radiological changes more expressive.

Laboratory Findings

Disorder	Ca	P	PTH	25-(OH)D	1,25-(OH)2 D	ALP	Urine Ca	Urine P
Vitamin D deficiency	N, ↓	↓	↑	↓	↓, N, ↑	↑	↓	↑

Classification

Calcium deficiency rickets can be classified in to 3 degree - I, II, III,

Depending on the duration, evolution and the complication:

1. degree I, II, III; evolution acute, subacute, recurrent.
2. Depending on vitamin D insufficiency:
   A) Diet
   B) Infections
   C) Food diversification
   D) Habitual
   E) No prophylaxis
   F) Prophylaxis with low dose
   G) Phenobarbital induced

Complications

- Rickets tetany
- Seizures
- Respiratory disorders
- Heart disorders
- Skeletal deformation
- Frequent illness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Muscular hypotonia

Increased lability of joints
«Pocket knife» symptom

Clinical manifestation of rickets

Muscular hypotony

Tibia convexity

Clinical Features of Rickets

General

- Failure to thrive (malnutrition)
- Listlessness
- Protruding abdomen
- Muscle weakness (especially proximal)
- Hypocalcemic dilated cardiomyopathy
- Fractures (pathologic, minimal trauma)
- Increased intracranial pressure

Clinical Features of Rickets

Head

- Craniotabes
- Frontal bossing
- Delayed fontanel closure (usually closed by 2 yr)
- Delayed dentition: No incisors by age 10 mo; No molars by age 18 mo
- Caries
- Craniosynostosis

Chest

- Rachitic rosary
- Harrison groove
- Respiratory infections and atelectasis

Clinical Features of Rickets

Back

- Scoliosis; Kyphosis; Lordosis

Extremities

- Enlargement of wrists and ankles
- Valgus or varus deformities
- Windswept deformity (valgus deformity of one leg with varus deformity of other leg)
- Anterior bowing of tibia and femur
- Coxa vara; Leg pain

Hypocalcemic Symptoms

- Tetany; Seizures
- Stridor caused by laryngeal spasm

Clinical manifestations

Rickets may develop in any age of an infant, more frequent at 3-6mo, early in premature infants.

- The first signs of hypocalcaemia are CNS changes - excitation, restlessness, excessive sweating during sleep and feeding, tremors of the chin and extremities.
- Skin and muscle changes - pallor, occipital alopecia, fragile nails and hair, muscular weakness, motor retardation.
- Complications - apnea, stridor, low calcium level with neuromuscular irritability (tetany).
- CNS changes are sometimes interpreted as CNS trauma and the administration of the Phenobarbital which interfere in metabolism of vitamin D and after 1-2wk of treatment with Phenobarbital the clinical stage worsens.

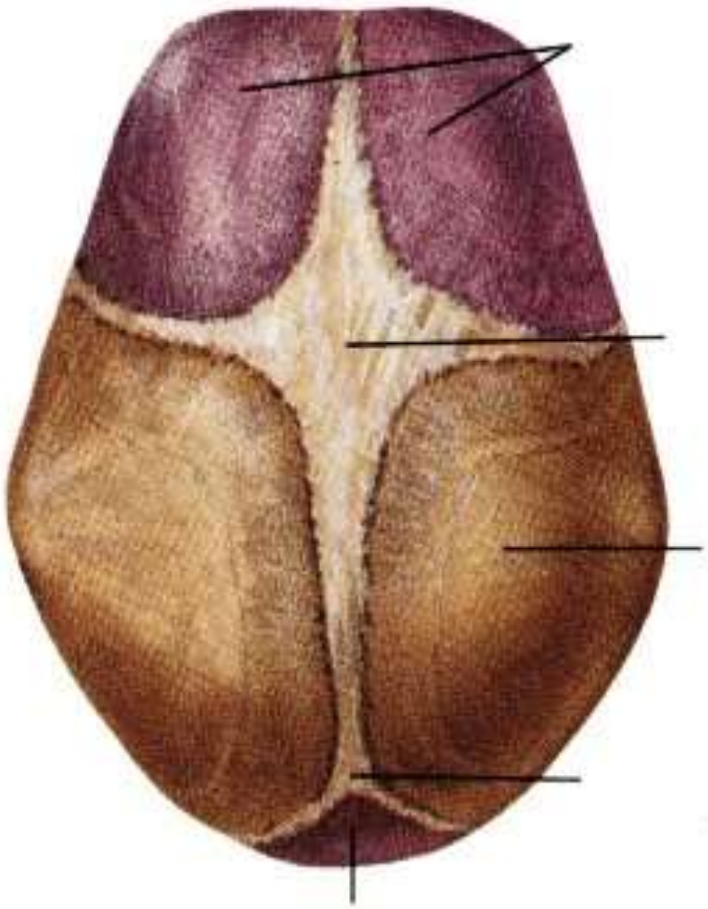
Acute signs

Florid (acute) rickets clinical signs:

- Craniotabes – osteomalacia, acute sign of rickets, detected by pressing firmly over the occipital or posterior parietal bones, ping-pong ball sensation will be felt. Large anterior fontanella, with hyperflexible borders, cranial deformation with asymmetric occipital flattening.

Subacute signs

- Frontal and temporal bossing
- False closure of sutures (increase protein matrix), in the X-ray craniostenosis is absent.
- Maxilla in the form of trapezium, abnormal dentition.
- Late teeth eruption, enamel defects in the temporary and permanent dentition.
- Enlargement of costo-chondral junctions - “rachitic rosary”
- Chest, sternum deformation, softened lower rib cage at the site of attachment of the diaphragm - Harrison groove.



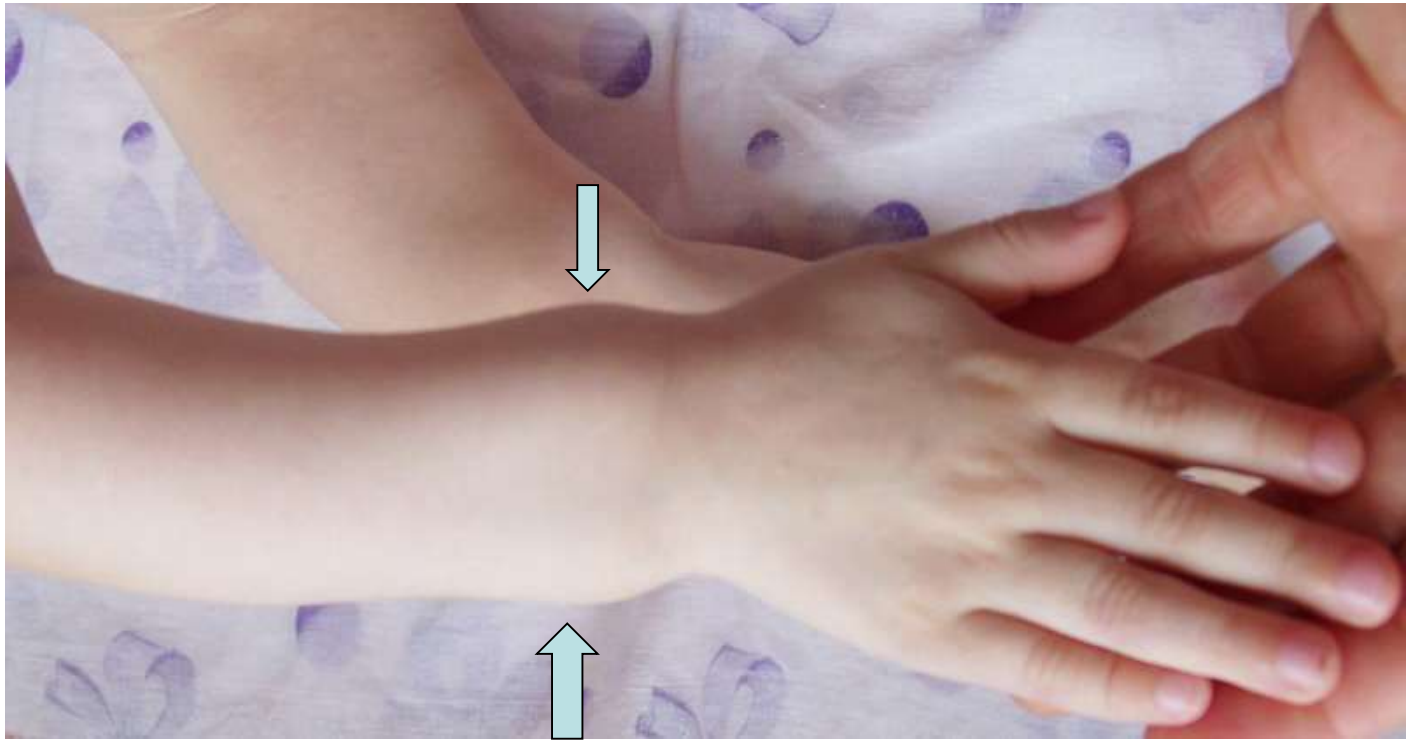
boxlike appearance

Harrison fissure

Rickets rosary

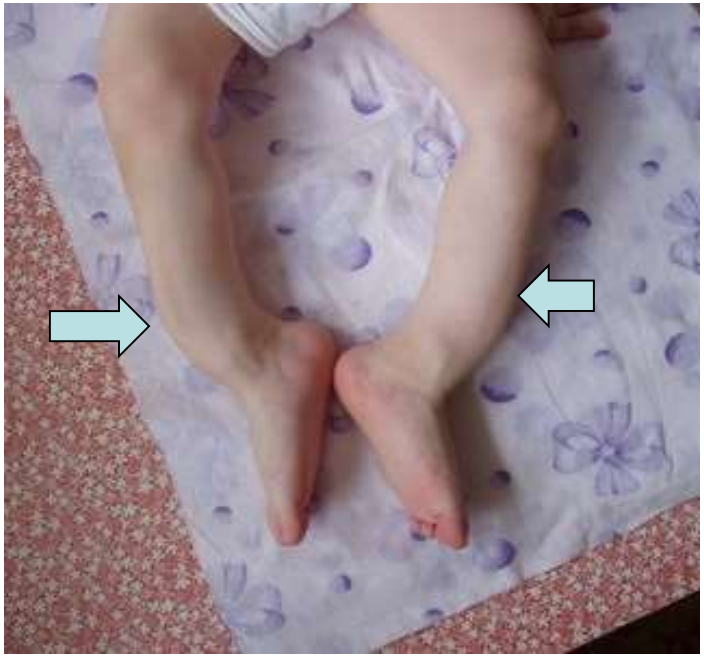
Subacute rickets signs

- Spinal column - scoliosis, lordosis, kyphosis.
- Pelvis deformity, entrance is narrowed (add to cesarean section in females)
- Extremities - thickening wrist and ankles, tibia anterior convexity, bowlegs or knock knees legs.
- Deformities of the spine, pelvis and legs result in reduced stature, rachitic dwarfism.
- Delayed motor development (head holding, sitting, standing, walking).



Thickening of the wrists

Deformation of the legs



“O”- shaped legs

“X” – shaped legs

Lordosis of vertebral column lumbar part

“O”-shaped deformation of the legs

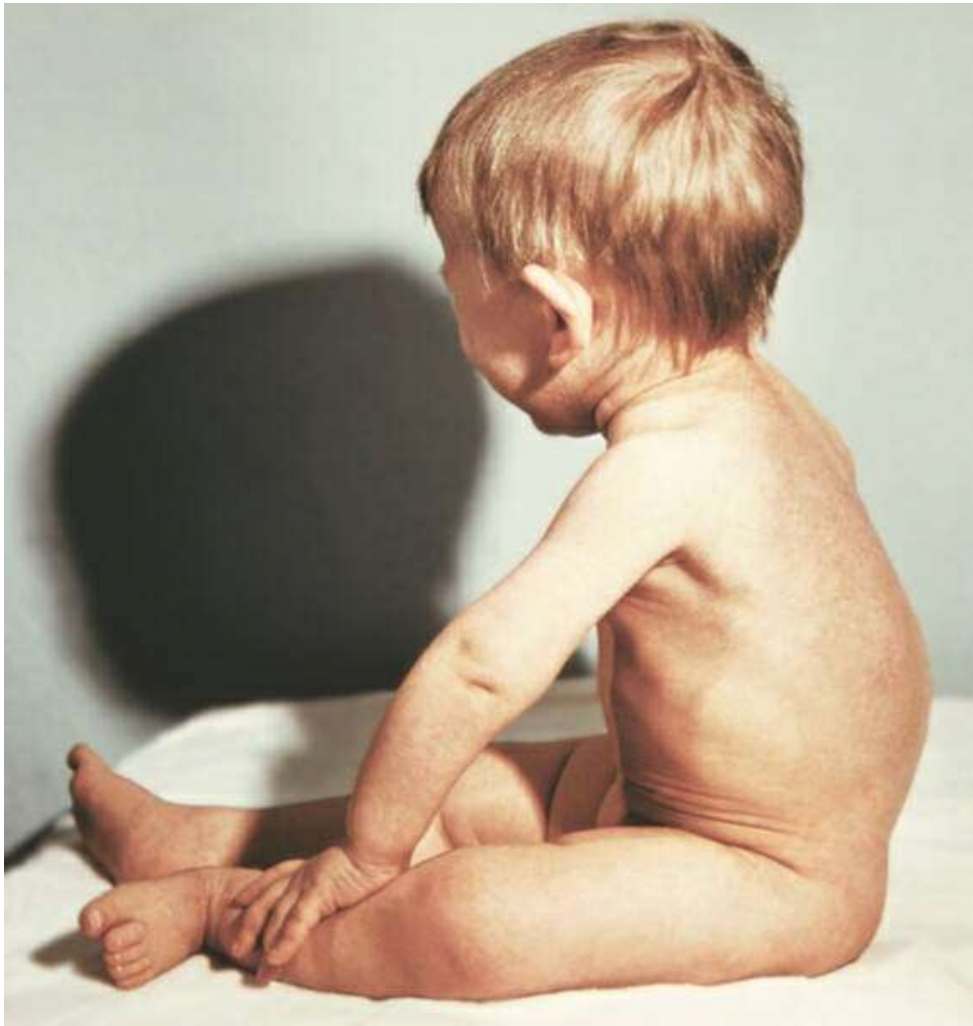
Changes of osseous system in rickets

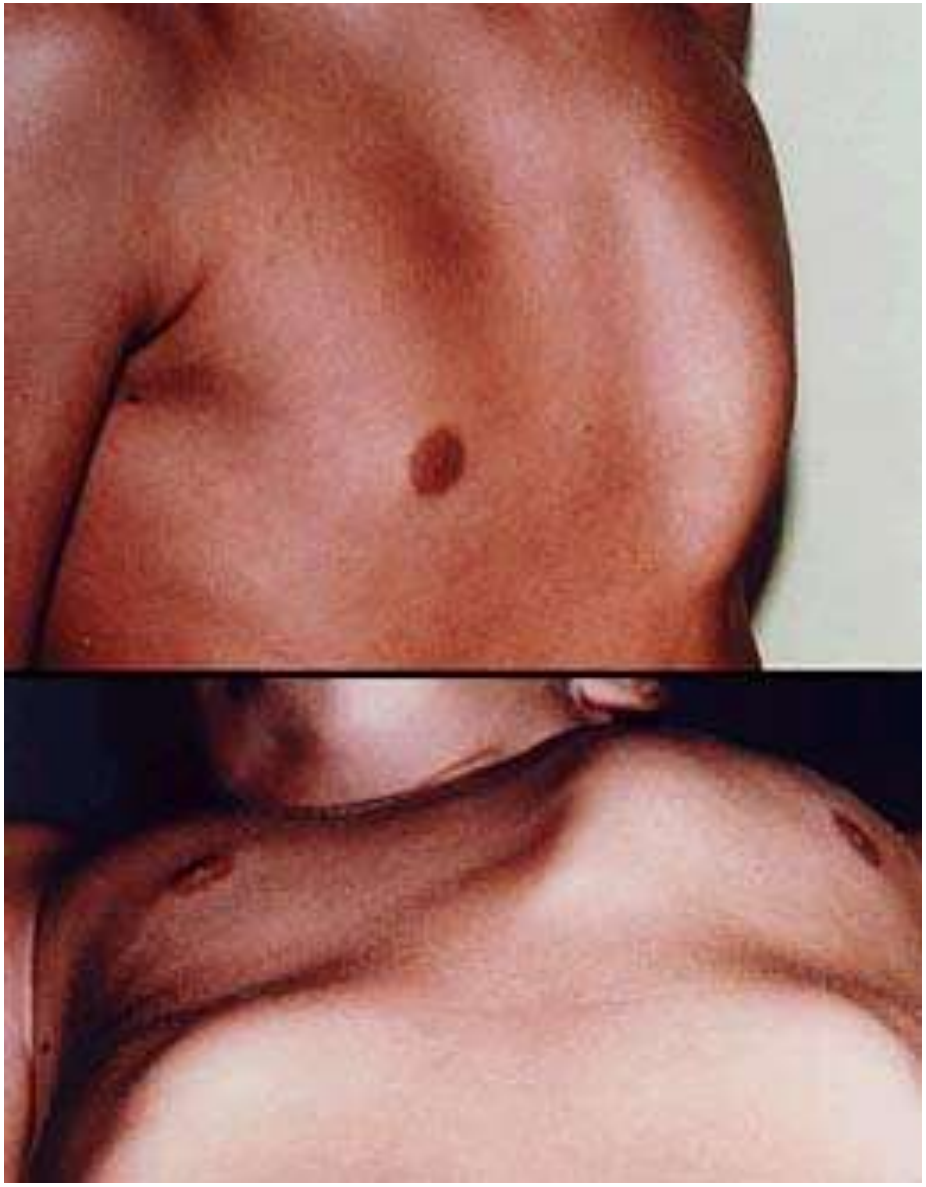
Deformation of vertebral column

Kyphosis in the lower part of thoracic vertebrae.
Kyphosis or lordosis in lumbar part.
Scoliosis in thoracic part.

Pelvic bones:

-flat pelvis,
-narrowing of pelvic cavity





Chest deformities

Laboratory data

1. Serum calcium level (N=2.2-2.6mmol/l). At the level <2.0mmol/l seizures sets in.
2. Phosphorus normal (1.5-1.8mmol/l). Normal ratio of Ca:P= 2:1; in rickets become 3:1; 4:1.
3. Serum 25(OH)D3 (N=28+2.1ng/ml); and 1,25(OH)2D3 (N=0.035+0.003ng/ml)
4. Serum alkaline phosphatase is elevated >500mmol/l.
5. Thyrocalcitonin can be appreciated (N=23.6+3.3pM/l)

Serum parathyroid hormone (N=598+5.0pM/l)

In urine: Aminoaciduria >1.0mg/kg/day

- Urinary excretion of 3’5’ cyclic AMP
- Decreased calcium excretion (N=50-150mg/24h)

Radiological findings

Only in difficult diagnostic cases.

1. X-ray of the wrist: concave (cupping) ends of ulna and radius in contrast to normally sharply, large rachitic metaphysis and a widened epiphyseal plate.
2. Osteoporosis of clavicle, costal bones, humerus.
3. Greenstick fractures.
4. Thinning of the cortex, diaphysis and the cranial bones.

Evolution

The evolution is slow with spontaneous healing at the age of 2-3 years.

If vitamin D are administered the normal bony structure is restored in 2-3mo.

Severe chest, spine and pelvis deformities may permanent persist.

Differential diagnosis

1. Vitamin D-dependent rickets type I and type II
2. Malabsorption disorders.
3. Hereditary Fanconi syndrome - multiple defects of proximal renal tubules, familial X-linked hypophosphatemia, renal tubular acidosis, osteogenesis imperfecta

Vitamin D-resistant rickets

- Type I called 1-alpha hydroxylase gene deficiency, result in inability to hydroxylate calcidiol in 1,25(OH)2D3 (calcitriol)
- Clinical and biochemical evidence of rickets starting in infancy, identified as unique form of vitamin D resistant rickets
- Calcitriol therapy 1-2mcg/day until healed bone, maintain dose varies 0,25-1mcg/day

Vitamin D-resistant rickets

- Type 2 vitamin D-dependent rickets, hereditary autosomal-recessive disorder, with end-organ resistance to calcitriol
- Rickets develop in first 2yr, peculiar syndrome is alopecia, marker of severity
- Additional ectodermal anomalies: multiple milia, epidermal cysts, oligodontia
- Treatment: Calcitriol 2mcg/day, calcium 1g/day, increased gradually to restore normal biochemical parameters

X-linked familial hypophosphatemia

- Autosomal recessive bone disease with tubular phosphorus reabsorption defect and reduced synthesis 1,25(OH)2D3
- Clinical manifestation of waddling gait, bowing legs, coxa vara, genu varus, genu valgum, short stature, enamel defects
- X-ray cupping of distal and proximal metaphysis of arm and legs

Treatment of familial hypophosphatemia

- Infants intake of sodium phosphate 0.5-1.0 g/24h, older children 1-4g/24h + vitamin D2 2000/kg/24h or 1,25(OH)2 D3 20-50ng/kg/24h
- Treatment used since patients become

Osteogenesis imperfecta

- Four genetic syndromes account in osteogenesis imperfecta: type I and IV autosomal dominant; type II and III autosomal recessive
- Clinical manifestation are common in all types: bone fragility, fractures, deformity of long bones and spine, short stature
- Calcium and calcitonin therapy increase skeletal mass and decrease fractures

Fanconi syndrome

- Rickets associated with multiple defects of the proximal renal tubule; de Toni-Debre-Fanconi syndrome, genetic disorder of metabolism or primary idiopathic
- Dysfunction in proximal tubule membrane with lost of bicarbonate, aminoaciduria, glycosuria, phosphateuria resulting in metabolic acidosis, hypophosphatemia, impaired conversion of vitamin D=>rickets

Prophylaxis in rickets

Specific antenatal prophylactic dose administration: 500-1000IU/day of vitamin D3 solution at the 28-th week of pregnancy. The total dose administered is 135000-180000IU. In term infants prophylactic intake of vitamin D3 500IU/d started at 10 days of age during the first 2 years of life; in premature the dose may increase to 1000IU/day.

Prophilaxis in rickets

WHO recommendation for rickets prophylaxis in a children coming from unfavorable conditions and who have difficult access to hospitals is 200000IU vitamin D3 i/muscular,

On the 7 day, 2, 4, 6 months - total dose 800000IU. In case of the necessary prolongation 500IU/day till 24mo are given.

Specific treatment in richets

The treatment is with vitamin D3 depending on the grade.

In I degree - 2000-4000 IU/day for 4-6 weeks, totally 120000-180000 IU.

In II degree - 4000-6000 IU/day for 4-6 weeks, totally 180000-230000 IU.

In III degree - 8000-12000 IU/day for 6-8 weeks, totally 400000-700000 IU.

Specific treatment in richets

- Along with vitamin D, calcium is also administered (40 mg/kg/day for a term baby, 80 mg/kg/day for a premature baby); also indicate vitamin B&C drugs.
- From the 7-th day of the treatment massage can be started.
- Intramuscular administration of 1% ATP solution in case of myopathy 1ml/day is preferred.

Rickets complications

1. Rickets tetany in result of low concentration of serum calcium (<2mmol/l), failure of the PTH compensation and muscular irritability occur.
2. Hypervitaminosis D occur after high oral dosing, extensive skin exposure to sunlight.

Clinical manifestation

Manifest tetany

- Spontaneous spasm: flexion at the elbow, extension of 2-5-th digits, extension and adduction of the thumb.
- Painful extension and adduction in the tibia tarsal joint.
- Rarely contractures in the eyelids and lips muscles.
- Laryngeal or bronchial spasm, manifesting as sudden dyspnea, apnea or cyanosis.

Latent tetany

The symptoms are not evident, but they can be performed.

Chvostek sign - percussion on the facial nerve leading to contraction of the superior lip, nasal wings, hemi or bilateral facial muscle contraction.

Trousseau sign - blood pressure cuff around the mid arm induce carp spasm.

Erb sign - <5mA galvanic current induced the nerve impulses.

The diagnosis of rickets tetany is based on the clinical manifestation of rickets, low levels of serum calcium, phosphorus, PTH; high serum alkaline phosphatase.

Treatment

- 1-2% of calcium chloride in milk - 4-6g/day for the first 2 days; after that 1-3g/day continued for 1-2wk. Calcium chloride in more concentrated may cause gastric ulceration. Calcium lactate may be added to milk in 10-12g/d for 10 days.
- Oxygen inhalation is indicated in convulsive seizures. Started treatment with vitamin D 5000-10000IU/d for 6-8 weeks, continued calcium intake. When the rickets is healed, the dose of vitamin D decrease to the usual prophylactic one.

Hypervitaminosis D

- Symptoms develop in hypersensitivity to vitamin D children or after 1-3mo of high doses intakes of vitamin D; they include hypotonia, anorexia, vomiting, irritability, constipation, polydipsia, polyuria, sleep disorder, dehydration. High serum level of acetone, nitrogen
- Ca>2.9mmol/l are found. Increase calcium concentration in urine may provoke incontinence, renal damage and calcification.

Treatment

- Preventing calcium rich food, cheese and cow's milk
- Intake mashed fruits and vegetables, juices, hydrating fluids - Ringer solution, water.
- Vitamin A, B, E according to age. In severe intoxication administration of Phenobarbital for 2-3 weeks or prednisone 1mg/kg 5-7 days reduces the calcium absorption and increases the calcium excretion. In the case of acidosis 4% sodium hydrocarbonate 5ml/kg is given.

Rickets Can be Prevent!