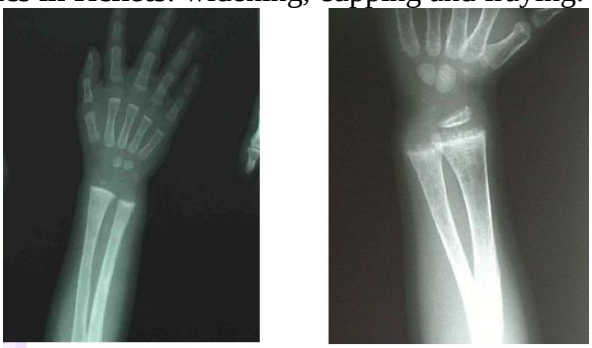
- **Rickets مرض الكُساح:**
  - It is a defective mineralization of the growing bone. In adults, this is known as osteomalacia (impaired mineralization of bone matrix).
  - **Rickets occurs due to:**
    - ✓ Vitamin D deficiency (the most common cause especially in infancy and early childhood).
    - ✓ Calcium deficiency (common between 3-6 years of age and known as adolescent rickets).
    - ✓ Deficiency of phosphate.
- **Types of vitamin D:**
  - **D2 (ergocalciferol):** found in plants.
  - **D3 (cholecalciferol):** found in animal sources especially salmon fish, eggs and cheese. It is also produced through exposure to sunlight for 5-30 minutes between 10 am and 3 pm (according to US weather) and temperature has to be less than 34 C ☺
- **Recommended daily needs of vitamin D:**
  - **< 1 year:** 400 IU/day
  - **1-18 years:** 600 IU/day
  - **> 18 years:** 1500 IU/day

  Notice that breast milk dependent infants must be supplied with vitamin D because breast milk only provides 25-40 IU of vitamin D per liter!
- **Metabolism of vitamin D:**
  - Liver and kidneys are involved in hydroxylation and activation of vitamin D. Therefore, any diseases in these two organs can result in vitamin D deficiency.
- **What are the functions of calcium?**

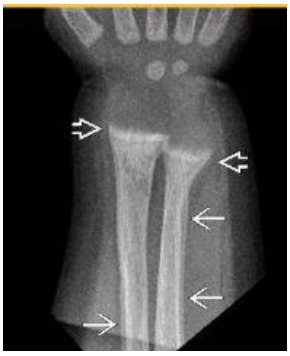
Intracellular	Extracellular
• Induction of electrical impulses inside cells. • Muscle contraction. • Degranulation and regulation of hormone release. • Co-factor in enzyme activities.	• Co-factor for coagulation cascade. • Maintains intracellular calcium concentrations. • Deposition of calcium (from diet) in bones.

- **What are the functions of vitamin D?**
  - **Intestine**: absorption of calcium and phosphate.
  - **Kidneys**: reabsorption of calcium and phosphate.
  - **Bones**: stimulating mineralization.
- **What are the functions of parathyroid hormone (PTH)?**
  - **Maintaining calcium level in blood (How?):**
    - ✓ Kidneys:
      - ❖ Increasing the excretion of phosphate.
      - ❖ Increasing the reabsorption of calcium in DCT.
    - ✓ Bones: increasing bone resorption.
    - ✓ Intestine: increasing calcium reabsorption by stimulating vitamin D production.
- **Calcitonin:**
  - It is produced by parafollicular cells in the thyroid gland.
  - **Function is independent of PTH and vitamin D:**
    - ✓ Increasing osteoblastic activity and decreasing osteoclastic activity.

- **Hypocalcemia:**
  - **This will stimulate PTH secretion and decrease calcitonin secretion thus:**
    - ✓ Increasing reabsorption of calcium and excretion of phosphate.
    - ✓ Increasing osteoclatic activity.
    - ✓ Activating vitamin D to aid in absorption of calcium from small intestine.
- **Growth plate in a normal bone:**
  - It is composed of a layer of cartilage in the two ends of the bone. These cartilage cells degenerate with deposition of calcium to produce ossification and formation of new bone thus growth occurs.
  - **There are four zones in the growth plate:**
    - ✓ Resting cartilage.
    - ✓ Proliferating cartilage.
    - ✓ Degenerating cartilage.
    - ✓ Ossification zone.
  - **Growth plate in rickets**: there will be no apoptosis of the hypertrophic chondrocytes (notice that this apoptosis is normally caused by phosphate ions).
  - **X-ray characteristics in rickets**: widening, cupping and fraying.

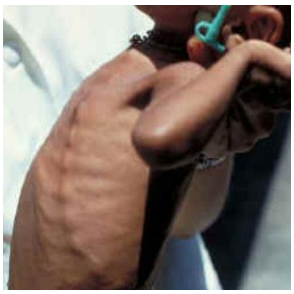


    - ✓ Healing rickets is characterized by "line of provisional calcification" which will start to appear.

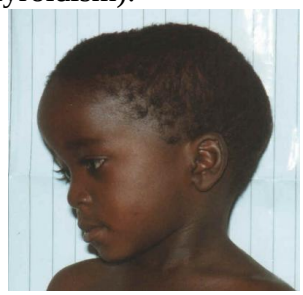
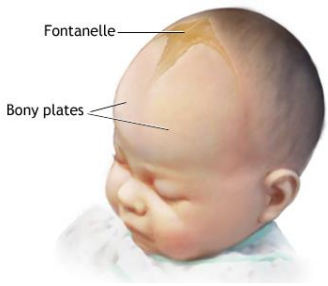


    - ✓ In healed rickets, bone density will return to normal but with slight cupping.
- **Biochemical manifestations in rickets:**
  - Increased serum alkaline phosphatase due to increased osteoblastic activity during formation of excessive osteoid.
  - Decreased serum inorganic phosphate.
  - Serum calcium is normal (but decreases with advanced rickets).
  - Decreased vitamin D and its metabolites.
- **What are the risk factors for rickets?**
  - Rapid growth rate which occurs between 6-36 months of age.
  - Being a vegetarian or exclusive breast-feeding for the first 6 months of life.
  - Dark skin (because melanin interferes with sunlight exposure).
  - Geographic location.
  - Genes: hereditary rickets (?)

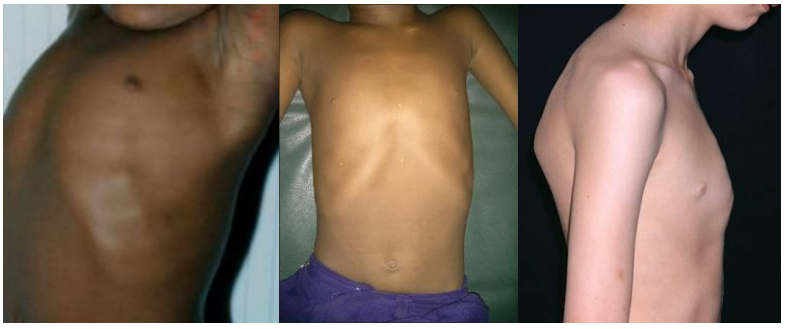
- **Symptoms of rickets:**
  - Sweating, restlessness and irritability.
  - Increased sympathetic activity.
  - Recurrent respiratory tract infections.
  - Muscle weakness and hypotonia.
  - Constipation.
  - Bone deformities.
  - Abdominal distention.
- **Early signs of rickets:**
  - Sweating ☺
  - Widening of anterior fontanel for age.
  - Craniotabes (see the image below): softening of the skull that usually occurs around the suture lines.
  - Rosary (see the image below): enlargement of osteochondral junction.
  - Radiological finding of active rickets.
  - Rise of serum alkaline phosphatase enzyme.

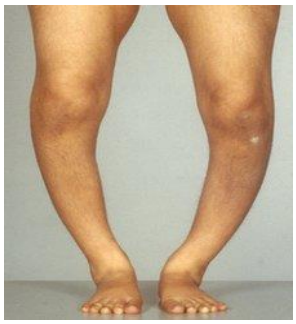
- **Advanced rickets:**
  - Bossing of the skull: due to increased proliferation of cartilage at occipital and parietal eminences making the skull looks like a box.
  - Enlargement of head circumference.
  - Delayed closure of anterior fontanel which remains widely opened (pay attention that posterior fontanel normally closes soon after birth and if it remains open you must suspect hypothyroidism).

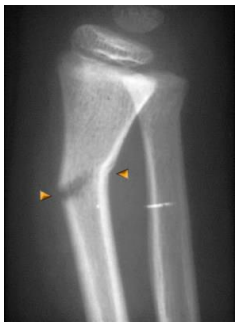



  - Rosary beads.
  - Longitudinal sulcus: longitudinal groove running along costochondral junction due to compression of rib cage by atmospheric pressure at the weakest point.
  - Harrison's sulcus: transverse sulcus along the lower border of costal margin due to inward traction of ribs at sites of diaphragmatic insertion.
  - Pigeon chest.



- Liver and spleen may be palpable.
- Pelvis inlet and outlet will be narrowed due to protrusion of the promontory and coccyx respectively. Females will face difficulty with delivery thus CS will be your choice.
- Correctable kyphosis (in dorsal region) and lordosis (in lumbar region).
- Deformities of long bones due to weight bearing (bowlegs: genu varum).
- Greenstick fracture: incomplete fracture (just from one side of the bone).
- Rackitec dwarfism.





- **Prognosis of rickets**: usually good with treatment but severe skeletal deformities will require orthopedic correction ☹
- **Prevention of rickets:**
  - Exposure to UV light in sunshine or administration of vitamin D orally (400-800 IU/day).
  - In preterms or those with malnutrition or hypothyroidism, 1000-1500 IU/day of vitamin D is needed due to accelerated rate of growth.
- **Treatment of rickets:**
  - Oral vitamin D (1500-5000 IU/day) for 6-8 weeks.
  - **Shock therapy**: vitamin D (600000 IU) via IM injection. The dose is repeated after 2-4 weeks if there is no radiologic evidence of healing.
- **Vitamin D dependent rickets type I (autosomal recessive):**
  - Defective 1α-hydroxylase enzyme in kidneys.
- **Vitamin D dependent rickets type II (autosomal recessive):**
  - Intracellular vitamin D receptor deficiency associated with early onset of severe rickets and alopecia.
- **Hereditary hypophosphatemic vitamin D resistant rickets (X-linked dominant):** affecting girls more; tubular defect in phosphate retention resulting in excessive urinary phosphate losses.
- **Tetany:**
  - Neuromuscular excitability due to decreased ionized serum calcium.
  - **There are two main types of tetany:**
    - ✓ Manifest tetany.
    - ✓ Latent tetany: there are no manifestations except with mechanical or electric stimulation of the nerve:
      - ❖ *Chvostek sign*: tapping anterior to external auditory meatus leading to contraction of orbicularis ori muscle and twitch of the upper lip of entire mouth.
      - ❖ *Peroneal sign*: tapping the head of the fibula leading to dorsiflexion and abduction of the foot.
      - ❖ *Trousseau sign*: blood pressure cuff on the arm is inflated above the systolic blood pressure for 5 minutes leading to carpopedal spasm.
  - **Treatment:**
    - ✓ Calcium 5-10 ml of 10% solution IV very slowly.
    - ✓ Followed by oral calcium lactate for 1 week.
    - ✓ And treatment of the underlying cause.