

## Hypocalcemia

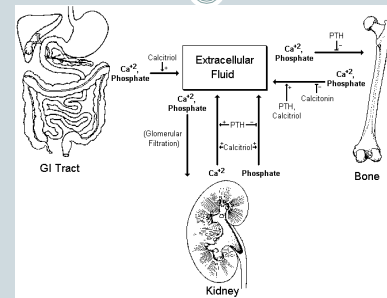
## Normal value

- Serum calcium

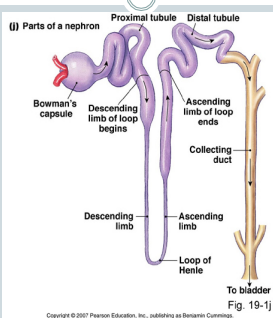
	Total mg/dl	Ionized mg/dl
Cord blood	9.0 ~ 11.5	5.0 ~ 6.0
New born (1 <sup>st</sup> 24 hrs)	9.0 ~ 10.6	4.3 ~ 5.1
24 ~ 48 hrs	7.0 ~ 12.0	4.0 ~ 4.7
Child	8.8 ~ 10.8	4.8 ~ 4.92
There after	8.4 ~ 10.2	4.8 ~ 4.92

## Physiologic functions

- Blood coagulation
- Cellular communication
- Exocytosis / endocytosis
- Muscles contraction
- Neuromuscular transmission

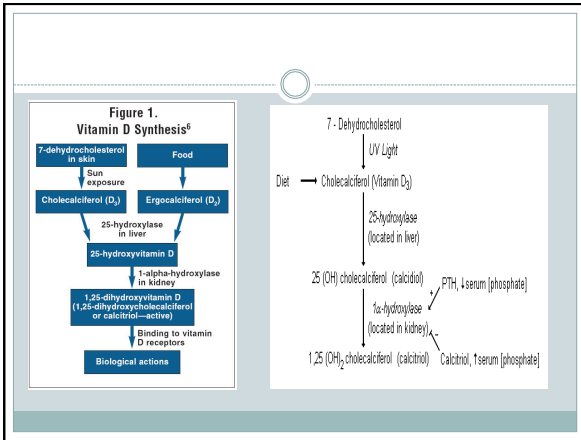


## Nephron – a functional unit of kidney



## Influencing factors in Calcium and Phosphate Balance

- Calcitriol (1,25 dihydroxycholecalciferol - active form of vitamin D3)
- Parathyroid hormone (PTH)



- 1,25(OH)<sub>2</sub>D increases intestinal calcium absorption and helps maintain normal serum calcium levels.
- When dietary calcium is low and, therefore, intestinal calcium absorption is low, 1,25(OH)<sub>2</sub>D also binds to osteoblasts, thereby increasing osteoclast activity and causing the release of skeletal calcium

### Role of Calcitriol

- Stimulates GI absorption of both calcium and phosphate
- Stimulates renal reabsorption of both calcium and phosphate
- Stimulates bone resorption

Net effect of calcitriol →  $\uparrow$  serum calcium  
 $\uparrow$  serum phosphate

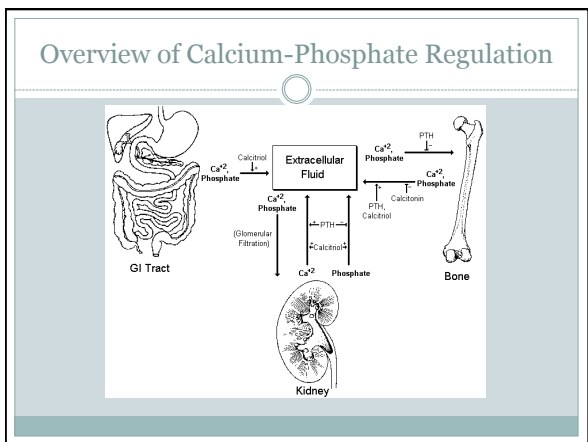
### Role of PTH

- Stimulates renal reabsorption of calcium
- Inhibits renal reabsorption of phosphate
- Stimulates bone resorption
- Inhibits bone formation and mineralization
- Stimulates synthesis of calcitriol

Net effect of PTH →  $\uparrow$  serum calcium  
 $\downarrow$  serum phosphate

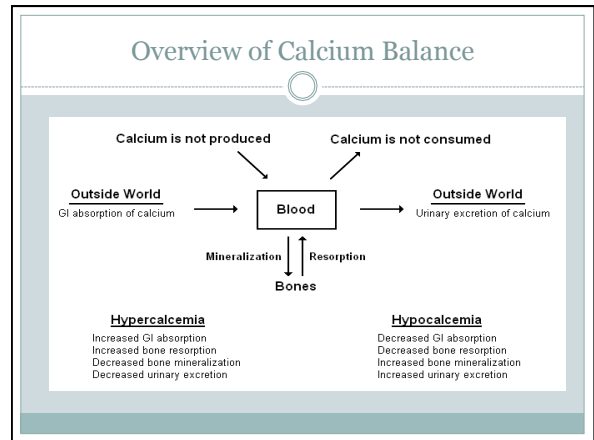
### Regulation of PTH

- Low serum [Ca<sup>2+</sup>] → Increased PTH secretion
- High serum [Ca<sup>2+</sup>] → Decreased PTH secretion



### Different Forms of Calcium

- Calcium in the plasma:
  - 45% in ionized form (the physiologically active form)
  - 45% bound to proteins (predominantly albumin)
  - 10% complexed with anions (citrate, sulfate, phosphate)
- To estimate the physiologic levels of ionized calcium in states of hypoalbuminemia:
- $[Ca^{+2}]_{Corrected} = [Ca^{+2}]_{Measured} + [0.8 \times (\text{decrease in albumin concentration below normal in g/dl})]$



### Etiologies of Hypocalcemia

<p><b>Decreased GI Absorption</b></p> <ul style="list-style-type: none"> <li>Poor dietary intake of calcium</li> <li>Impaired absorption of calcium</li> <li>Vitamin D deficiency                             <ul style="list-style-type: none"> <li>Poor dietary intake of vitamin D</li> <li>Malabsorption syndromes</li> </ul> </li> <li>Decreased conversion of vit. D to calcitriol                             <ul style="list-style-type: none"> <li>Liver failure</li> <li>Renal failure</li> </ul> </li> <li>Low PTH</li> <li>Hyperphosphatemia</li> </ul> <p><b>Decreased Bone Resorption/Increased Mineralization</b></p> <ul style="list-style-type: none"> <li>Low PTH (hypoparathyroidism)</li> <li>PTH resistance (pseudohypoparathyroidism)</li> <li>Vitamin D deficiency / low calcitriol</li> <li>Hungry bones syndrome</li> <li>Osteoblastic metastases</li> </ul>	<p><b>Increased Urinary Excretion</b></p> <ul style="list-style-type: none"> <li>Low PTH                             <ul style="list-style-type: none"> <li>thyroidectomy</li> <li>Autoimmune hypoparathyroidism</li> </ul> </li> <li>PTH resistance</li> <li>Vitamin D deficiency / low calcitriol</li> </ul>
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### Hypocalcemia

- Serum calcium < 8 mg/dl or
- Ionized calcium < 4 mg/dl

### Causes ( O P Ghai)

- **Vit D deficiency**
  - Malnutrition
  - Malabsorption
  - Abnormal metabolism
  - Prolong phenytoin therapy
- **Increase losses**
  - Idiopathic hypercalciuria
  - Renal tubular acidosis
  - Frusemide therapy
  - Prolonged corticosteroid therapy

- **Metabolic**
  - Hypoparathyroidism (Low PTH)
  - Pseudohypoparathyroidism (PTH resistance )
  - Hypomagnesemia
  - hyperphosphatemia
- **Others**
  - Hypoproteinemia
  - Acute pancreatitis
  - Prematurity
  - IDM
  - High phosphate milk ingestion

### Clinical features

- **Tetany ( a classic manifestation)**
  - Carpopedal spasms – wrist flexed, fingers extended, thumbs adducted over palms / feet extended and adducted, toes planter flexed
  - Laryngospasm – spasmodic closure of the vocal cord producing expiratory stridor
  - Seizures – brief and recurrent, usually generalized
- **Jitteriness**
- **Paresthesias – perioral, hands and feet**

**Illustration of**  
**Respirating carpopedal spasm**

In the hand, carpopedal spasm is characterized by the flexion of the wrist, extension of the fingers, and adduction of the thumb over the palm. The feet are characterized by the extension of the foot, plantar flexion, and adduction of the toes over the plantar surface.

- **Trousseau sign**
  - A blood pressure cuff is inflated slightly above the systolic pressure for more than 3 minutes; carpopedal spasm occurs if hypocalcemia is present as a result of ischemia of motor nerves
- **Chvostek sign**
- Tapping the facial nerve anterior to the external auditory meatus elicits a twitch of the upper lip or entire mouth
- **ECG**
  - Prolong QT interval, prolong ST interval, peaked T wave, heart block

**TROUSSEAU'S SIGN**

**Illustration:** Inflating a sphygmomanometer cuff above systolic blood pressure for several minutes.

**Positive response:** Muscular contraction including flexion of the wrist and metacarpophalangeal joints, hyperextension of the fingers, and flexion of the thumb on the palm, suggestive of neuromuscular excitability caused by hypocalcemia.

Figure 2. Illustration of the elicitation of Trousseau sign. Adapted with permission from Bates: The Clinical Examination of Medicine, 10th Edition, © 2001 by Elsevier Saunders, Philadelphia, PA, 2001.

### Management

- **Symptomatic hypocalcaemia**
  - 100 – 200 mg / kg calcium gluconate (1 – 2 ml / kg of 10% calcium gluconate; 9 – 18 mg of elemental calcium) in 5 – 10 mins under cardiac monitoring (can be repeated every 8 to 6 hours)
- **Calcium supplementation**
  - Calcium carbonate, calcium gluconate, calcium glubionate – 50 mg/kg/day in 3 - 4 divided doses

- **In certain situations like**
  - Pancreatitis – complete correction of hypocalcaemia should be avoided because resolution of primary problem will release Ca from complexed calcium
  - Hypocalcaemia with acidemia – first correct hypocalcaemia if possible because acidemia increases ionized calcium concentration by displacing calcium from albumin. Correction of acidemia will decrease ionized calcium.

## Hypercalcaemia

### • Causes

- Parathormone excess
  - Parathyroid adenoma
  - Parathyroid hyperplasia
  - Transient neonatal hyperparathyroidism
- Vit D excess; hypervitaminosis A
- Sarcoidosis
- William's syndrome
- Subcutaneous fat necrosis
- Thyrotoxicosis
- Prolonged immobilization

## Clinical features

- Urinary stones with colic and hematuria
- Nephrocalcinosis – deposition of Ca in renal parenchyma
- Bony changes – fractures, deformity etc
- Severe hypercalcaemia - coma

## Management

- Forced saline diuresis with frusemide or peritoneal dialysis
- Biphosphonates – modality of treatment in children
- Pamidronate and etidronate – in hypercalcaemia due to malignancy, immobilization and hyperparathyroidism
- Surgical