

HYPOCALCEMIA



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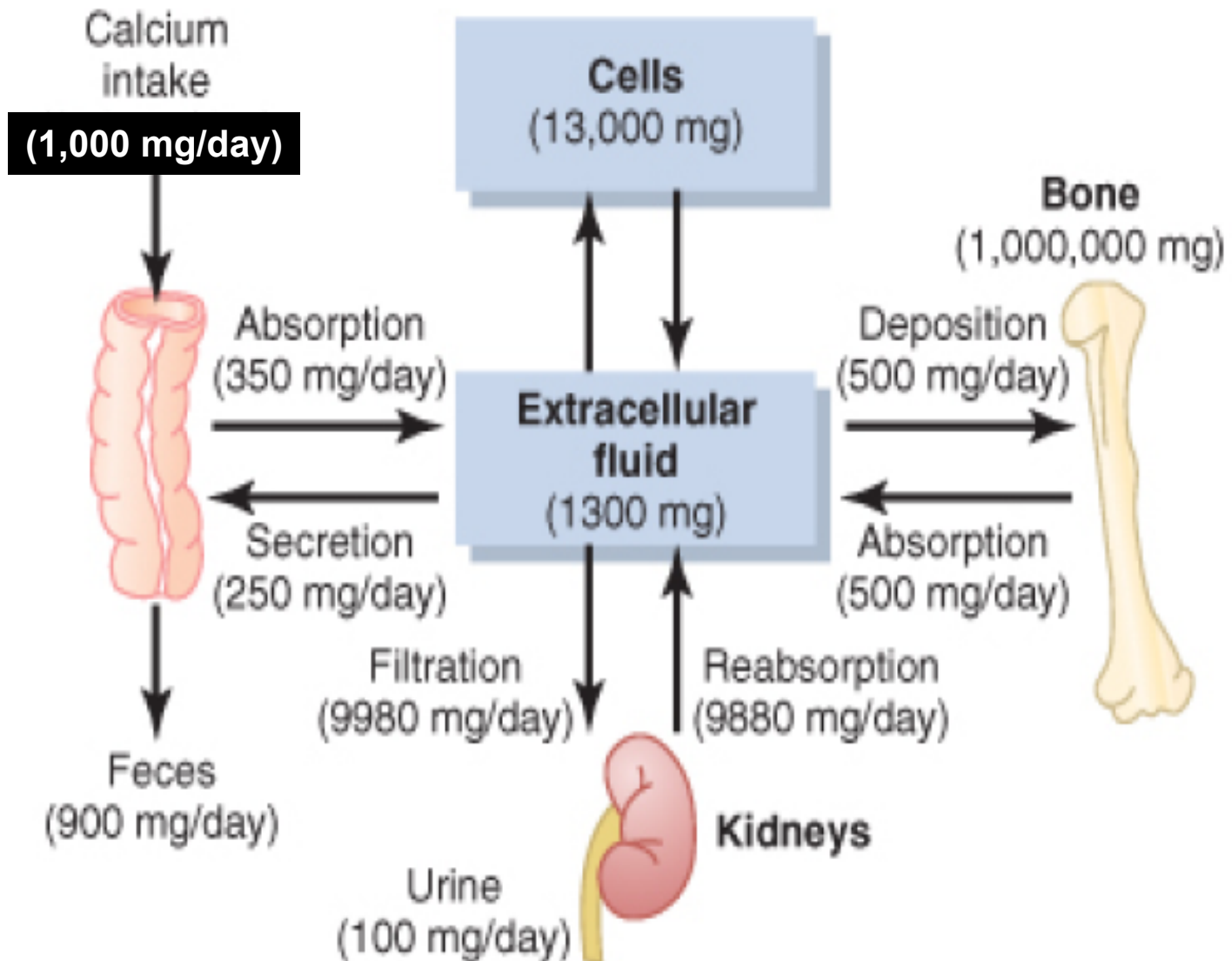
Why do we need calcium

- Calcium messenger system – regulates cell function
- Activates cellular enzyme cascades
- Smooth muscle and myocardial contraction
- Nerve impulse conduction
- Secretory activity of exocrine glands

Physiology of Calcium

- 98% of the body calcium is in the skeleton
- Only 2% is in circulation and only half of this is free calcium (ionized Ca^{++})
- Ionized Ca^{++} is only physiologically active
- 1% is bound to proteins

Calcium Metabolism



Calcium Homeostasis

Parathyroid Hormone

1,25 DHC or Vitamin D₃

Calcitonin

Calcium Homeostasis

PTH

- 4 PT glands
- 84 AA hormone
- Increase serum Ca

Calcitriol

- Active bone formation
- Main effect is on the Gut
- Increase Ca absorption from gut

Calcitonin

- Para follicular C of Thyroid
- 34 AA hormone
- Opposing the effect of PTH

Calcium

**Reference Range:
8.8 – 10.3 mg/L**

Hypocalcemia

- A serum calcium **<8.4 mg/dL** with a normal serum albumin
- or
- An ionized calcium **<4.2 mg/dL** defines hypocalcemia

Pseudo Hypocalcemia

Pseudohypocalcemia describes the situation in which the total calcium is reduced due to hypoalbuminemia, but the **corrected [Ca]** and ionized calcium remain within the normal ranges.

Corrected serum calcium = Measured serum calcium + (0.8)x (4 - Measured serum albumin).

Thus, in a patient with a serum $[Ca^{2+}]$ of 7.8 mg/dL and a serum albumin of 2 g/dL, the corrected serum $[Ca^{2+}]$ is $7.8 + (0.8) \times (4 - 2) = 9.4$ mg/dL.



Etiology

Causes of Hypocalcemia

- 1. Decreased intake or absorption**
 - a) Malabsorption
 - b) small bowel bypass, short bowel
 - c) vitamin D deficit- decreased absorption

- 2. Increased loss**
 - a) alcoholism
 - b) chronic kidney disease
 - c) diuretic therapy

Causes continued-

3. Endocrine causes-

a) hypoparathyroidism (genetic, acquired)

b) post-parathyroidectomy

c) pseudo hypoparathyroidism

d) calcitonin secretion with medullary carcinoma of thyroid.

e) familial hypocalcemia

Causes continued-

4. Associated diseases

- a) Pancreatitis**
- b) Rhabdomyolysis**
- c) Septic shock**

5. Physiologic causes

- a) Associated with decreased serum albumin.**
- b) Decrease end – organ response to vitamin D**
- c) Hyperphosphatemia**
- d) Induced by aminoglycoside antibiotics, loop – diuretic, foscarnet**

Clinical Feature

Clinical manifestations vary with the *degree of hypocalcemia* and *rate of onset*.


1- **Acute, severe hypocalcemia** may cause

i) laryngospasm,

ii) confusion,

iii) seizures,

iv) vascular collapse with bradycardia and decompensated heart failure.

2- **Acute, moderate hypocalcemia** may cause increased excitability of nerves and muscles,  circumoral or distal paresthesias and tetany.

Latent Tetany

- **Trousseau's sign** is the development of carpal spasm when a blood pressure cuff is inflated above systolic pressure for 3 minutes.
- **Chvostek sign** refers to twitching of the facial muscles when the facial nerve is tapped anterior to the ear.
- The presence of these signs is known as **latent tetany**.



ASSESSMENT TIP

Eliciting Chvostek's sign

Begin by telling the patient to relax his facial muscles. Then stand directly in front of him, and tap the facial nerve either just anterior to the earlobe and below the zygomatic arch or between the zygomatic arch and the corner of his mouth. A positive response varies from twitching of the lip at the corner of the mouth to spasm of all facial muscles, depending on the severity of hypocalcemia.



Trousseau sign:

(very uncomfortable and painful)

- **Trousseau's sign** is the development of carpal spasm when a blood pressure cuff is inflated above systolic pressure for 3 minutes.



Response-

- * Flexion at the wrist
- * Flexion at the MP joints
- * Extension of the IP joints
- * Adduction thumbs/fingers

Symptoms and signs may be associated with underlying cause

- **Vitamin D deficiency:** bone pain ,fractures or proximal myopathy.
- **Hypoparathyroidism** - mental retardation, personality changes, extra-pyramidal signs, cataract and papilloedema.
- *Hypocalcemia during development of permanent teeth:* enamel hypoplasia

Enamel dysplasia

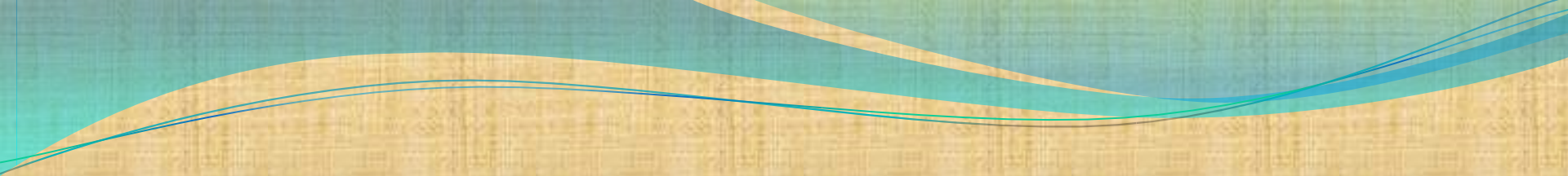


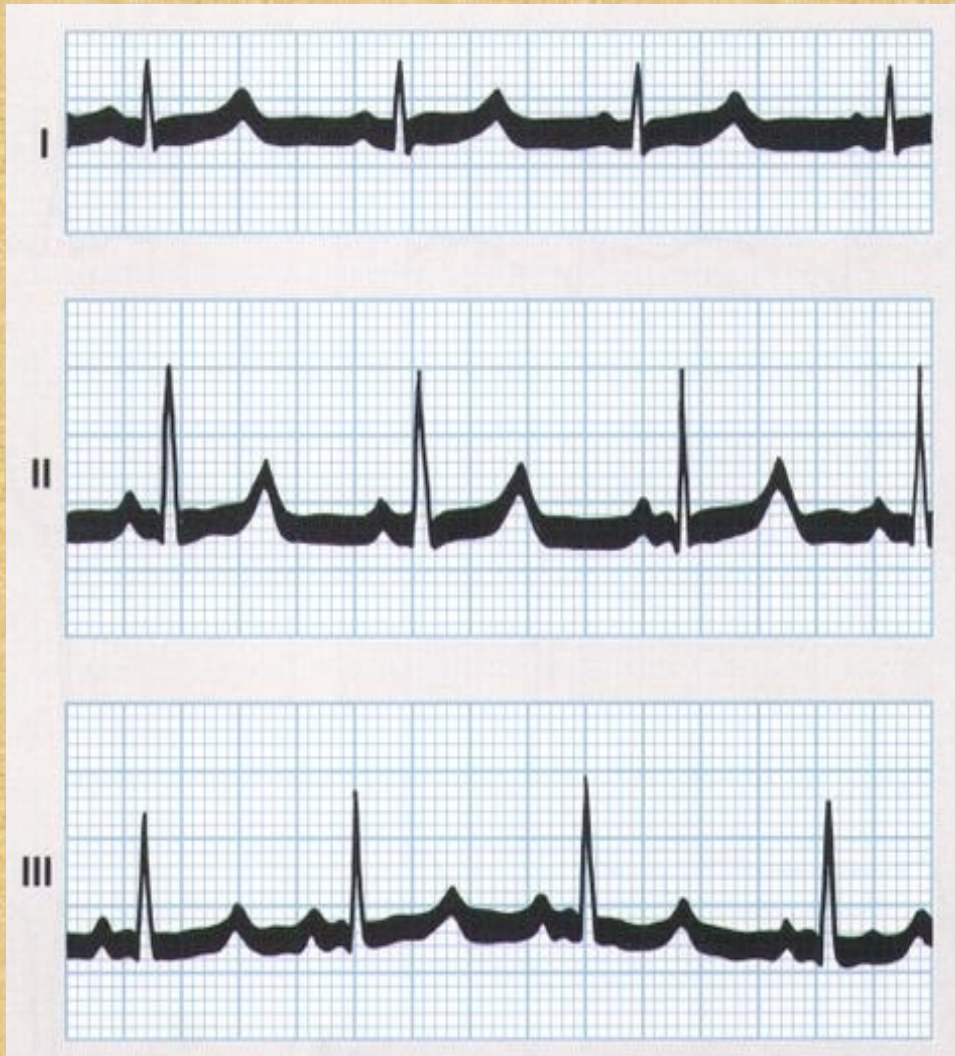
Features of hypocalcaemia 'SPASMODIC'¹²

- **S**pasms (carpopedal spasms = Trousseau's sign)
- **P**erioral paraesthesiae
- **A**nxious, irritable, irrational
- **S**eizures
- **M**uscle tone ↑ in smooth muscle—hence colic, wheeze, and dysphagia
- **O**rientation impaired (time, place and person) and confusion
- **D**ermatitis (eg atopic/exfoliative)
- **I**mpetigo herpetiformis (↓Ca²⁺ and pustules in pregnancy—rare and serious)
- **C**hvostek's sign; **c**horeoathetosis; **c**ataract; **c**ardiomyopathy (long QT interval on ECG)

Investigation

- **Total and ionized calcium**
- **Serum Albumin** should be measured if there is an abnormality in serum calcium levels to rule out pseudohypocalcemia.
- **Serum PTH** that is **low** or inappropriately normal in the setting of hypocalcemia is indicative of **hypoparathyroidism**.
- A high PTH is often found with **vitamin D deficiency**, PTH resistance, and hyperphosphatemia.
- **Serum phosphorus** is often helpful in identifying vitamin D deficiency (low calcium, low phosphorus) or intravascular chelation of calcium (low calcium, high phosphorus).

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- **Vitamin D level**
 - **Magnesium** deficiency should always be ruled out during management of hypocalcemia
 - **Kidney function test**
 - The **ECG** may show a prolonged QT interval and bradycardia
 - **Karyotyping and family screening**



- Long QT interval with normal T waves



Treatment of Hypocalcemia

Acute Hypocalcemia

- **Acute management of symptomatic hypocalcemia** requires prompt and aggressive therapy.
- **Calcium supplementation.** IV calcium is reserved for severe or symptomatic hypocalcemia .
- Administered as **calcium chloride or calcium gluconate.**
- Calcium gluconate - favoured due to reduced risk of tissue toxicity with extravasation.

Acute Hypocalcemia

- Symptomatic hypocalcemia may be treated with IV calcium gluconate (bolus of 1–2 g IV over 10–20 min followed by infusion of 10 ampoules of 10% calcium gluconate diluted in 1 L D5W infused at 30–100 mL/h).
- **Hypomagnesemia**, if present, must be treated first in order to effectively correct the hypocalcemia.

Chronic management

Management of chronic hypocalcemia requires a high oral calcium intake, usually with vitamin D supplementation.

Oral calcium supplements.

Calcium carbonate (40% elemental calcium) or calcium acetate (25% elemental calcium) can be given with the goal administration of 1 to 2 g of *elemental* calcium per orally.

Calcium supplementation should be given apart from meals to minimize binding with phosphorus and maximize enteric absorption.

Serum levels should be checked once to twice per week to guide on going therapy.

Vitamin D

- Simple dietary deficiency can be corrected by the use of ergocalciferol 400 to 1,000 IU/d.
- However, in conjunction with other hypocalcemic disorders, larger doses may be required.
- A 6- to 8-week regimen of 60,000 IU should be given weekly in those with underlying impairments in vitamin D metabolism (i.e., renal insufficiency)

Complication

- **Development of Hypercalcemia.** In the event that Hypercalcemia develops, vitamin D and calcium supplements should be stopped.
- Once serum calcium falls to normal, both forms of supplementation should be restarted at lower doses.
- Hypercalcemia due to calcitriol usually resolves within 1 week

Thanks

