Electrolyte imbalance (Potassium & Calcium)

Objectives:

- Understand the basic physiologic principles of potassium hemostasis.
- Know the application of physiologic and clinical principles in approaching hyperkalemia.
- Know the application of physiologic and clinical principles in approaching hypokalemia.
- Understand the basic principles of Calcium hemostasis.
- Know the application of physiologic and clinical principles in approaching hypercalcemia.

Team Members: Allulu Alsulayhim, Ghada Alskait, Trad Alwakeel, Lulwah Alshiha.

Team Leader: Nora AlSahli

Doctor: Riyadh AlSehli

Revised By Doctor: Maha AlGhamdi

Resources: 435 team + Davidson + Step up.

- Editing file
- Feedback

Basic knowledge about potassium

★ Where does K come from?

- → The main source of K is our diet.
- → **Can be found in:** Fruits, potatoes, beans, and grains. You are eating lots of K if u r MacDonald's fan.
- → High-fat diets usually contain low amounts of potassium.
- → Average daily intake approximately 50 to 100 mmol.

Table 1 Potassium content of selected foods

Foods and drinks	Potassium content (mmol)
1 small banana (85 g)	8.6
Blueberries (100 g)	1.9
White mushrooms (75 g)	8.1
Broccoli, cooked (75 g)	5.8
Green beans, cooked (75 g)	3.9
Onions, cooked (75 g)	1.5
French fries (150 g)	17.7
Parboiled rice (150 g)	2.2
Spaghetti, without egg (150 g)	2.3
Orange juice (200 ml)	7.9
Milk, full fat (200 ml)	7.7
Coca Cola (200 ml)	0.1
Potato crisps (20 g)	5.1
Milk chocolate bar (20 g)	2.4
White chocolate (20 g)	1.8
Wine gums (20 g)	1.8

This is actually what they use in the clinic especially patients with kidney disease and we usually advise our patient to avoid certain foods which are high in potassium like "bananas, French fries, dates and orange juice"

★ How do we lose K:

- → **Renal excretion**: PRIMARY mechanism! Very efficient gets rid of almost 90% of our potassium intake until GFR < 30 ml/min. Normal GFR is > 60 ml/min
- → **Intestinal excretion:** Only handles 10 % of the daily K load. Efficiency can be enhanced in renal failure but it is variable from one person to another. When GFR is compromised it can reach 15-20% in patients with chronic kidney disease.

★ Where does K live in the body:

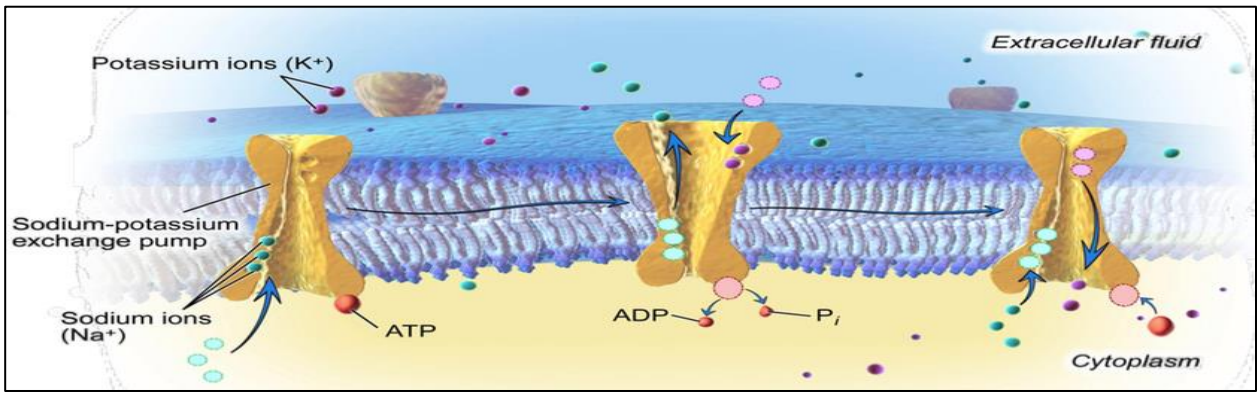
- → Potassium is a cation found majorly inside the cell (intracellular). While sodium is the major extracellular cation.
- → Total body potassium is approximately 50 mmol/kg body weight.
- → **Intracellular**: 100- 150 mmol/L (98 % of total body **K**).
- → **Extracellular**: 3.4 – 5.5 mmol/L (2% of total body **K**) this is what they measure in the lab when we ask for potassium level in the blood.

★ What keeps the intracellular K high?

- → **Insulin + Beta agonists** (catecholamine): Enhance pump function. So, Hypokalemia is a side effect of insulin.

★ What keeps Extracellular K low?

- → The Na/K ATPase pump زي لما يجونك ضيوف (البومب فقط تظف لك المكان الين تشتغل الكلية وتتخلص من البوتاسيوم, فجأة والمكان محيوس تقوم تاخذ حوستك وتخبيها بالادراج)
- → Renal clearance: requires normal GFR and normal aldosterone axis (Adrenal deficiency, Aldosterone resistance). The most important clinically.
- → Intestinal excretion.
- → **Beta blocker**: Inhibit pump function. Clinically important.



K\Na pump is very important & vital: it keeps the balance between intracellular K & Na. This pump is critical to establish the resting membrane potential which is -90 millivolt by creating charge difference. If you understand this u will understand a lot of K complications.
Now we know that the potassium is very high inside the cell and low outside it and vice versa with sodium what should happen when considering the law of physics is that they should move from the high concentration to low concentration until they reach equilibrium. But does that really happen? NO because we have ATP- dependent or active transport channel the Na/K ATPase which pushes 3 molecules of Na against the conc. gradient and pulls 2 molecules of K which keeps the charge inside the cell negative and maintains the resting membrane potential.

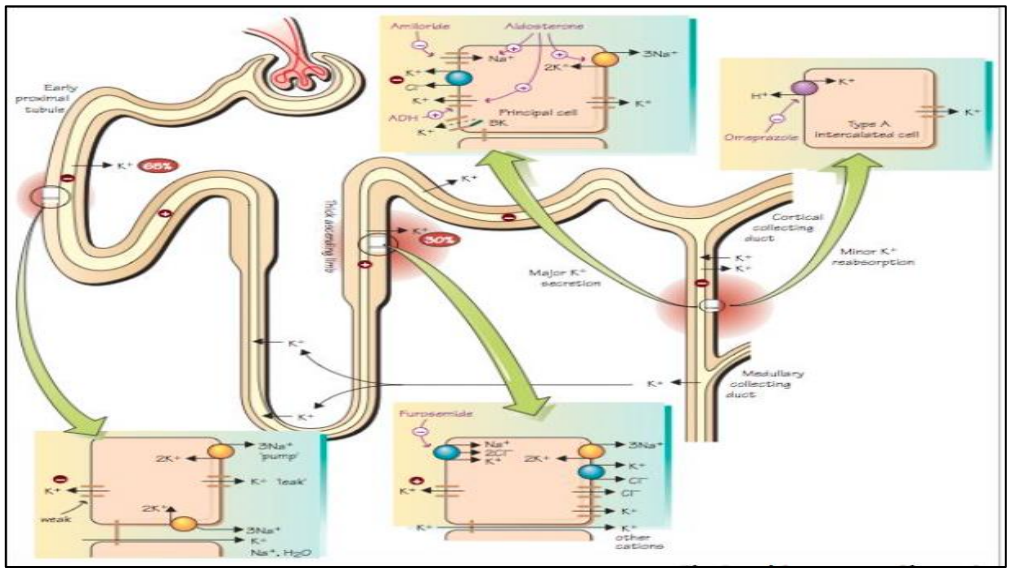
★ What happens when we eat K? (Normal physiology of Potassium):

- → Oral K intake →Absorbed in the intestine →enters portal circulation →Increased ECF K stimulates **insulin** release → Insulin facilitates K entry into intracellular compartment by stimulating cell membrane **Na/K ATPase pump**. Which is not enough and further stimulation of the renal system is required.
- → The transient rise in serum K stimulates renal and intestinal clearance of extra K. Aldosterone system starts working. So, one of the triggers for it is hyperkalemia

★ In order to Keep Serum K in normal range, we need:

1. Normally functioning Na/K ATPase pump.
2. Intact renal response.

There are two important components in the kidney that must intact in order for the system to work efficiently which are Normal GFR (normal blood flow to the kidney) and normal renal tubules (functioning aldosterone produced in normal amounts and with sensitive receptors)



Most of our K regulatin happens in the collecting ducts because secretion happens here. Always remember that the Kidney can Reabsorb (Na, uric acid, bicarbonate, Glucose) BUT it never never reabsorb **K** why? Because we want to keep it at a very low level in the blood.

Site of action of aldosterone is collecting ducts it enhances the secretion of K (VERY important in maintaining potassium balance).

Why is K important?

- → Maintains **electrical gradient** across cell membranes i.e.: resting membrane potential.
- → Essential for generation of **action potential**.
- → Essential for **intracellular metabolism** e.g protein synthesis.

★ What happens if K level is abnormal?

★ Most of the symptoms are from muscles either skeletal or cardiac

- → Skeletal muscle dysfunction: weakness and paralysis
- → Cardiac cell irritability: arrhythmia in advanced stages can lead to cardiac arrest.

- **What is the effect of hyperkalemia on Action Potential?**

Hyperkalemia reduces the negativity of the resting membrane potential. For example, it changes it from -80 mV to -70 mV. When the resting membrane potential drops like this, it becomes very close to the threshold potential which increases the cell excitability.

★ Can you eat too much K?

- → If GFR is normal, renal clearance of **K** has a huge adaptive capacity.
- → **K** intake is restricted only if:
  - GFR is reduced.
  - Existing aldosterone axis dysfunction.
  - Na/K ATPase is not efficient (blocked by drugs, Insulin ↓).

★ Hyperkalemia vs Hypokalemia:

Hyperkalemia [K]>5.5	Hypokalemia [K]<3.4
★ Causes: (the most common cause of hyperkalemia in clinical practice is tubular dehydration) [K] secretion depends on how much water and Na deliver to the distal tubule. **1- NA/K ATPase dysfunction:** ▪ B blockers ▪ Digoxin ▪ ↓Insulin (Type 1 diabetes) **2- Massive Cell breakdown:** ▪ Rhabdomyolysis (massive necrosis of the muscle and muscle breakdown, typically happens in crush injury) ▪ Tumor lysis syndrome (those with malignant hematological tumors who are treated with chemo → it causes lysis of the malignant cells	**★ Causes:** **1- Decreased Oral intake:** ▪ Malnutrition ▪ Eating disorders **2- GI losses:** ▪ Diarrhea. ▪ Laxative abuse. Usually in patients with eating disorders. ▪ Intestinal fistulas. ▪ Decreased potassium absorption in intestinal disorders. ▪ Vomiting and nasogastric drainage (volume depletion and metabolic alkalosis also result).

→ a huge amount of intracellular K will be released in the circulation.)
- Hemolysis
- Burns.

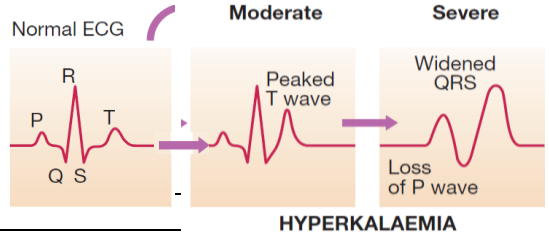
3- Impaired renal function Acute kidney injury, Chronic renal failure whatever the reason was as long as the GFR is reduced.

4- Aldosterone axis dysfunction:
- Adrenal deficiency. (Addison disease)
- Aldosterone resistance. Inherited diseases or some medications like: Spironolactone (Block aldosterone receptors), ACE inhibitors.

Some patient with advance DM will have aldosterone resistance

#Redistribution: translocation of potassium from intracellular to extracellular space:
- Acidosis (not organic acidosis)
- GI bleeding.
- Insulin deficiency: Insulin stimulates the Na+ -K+ ATPase and causes K+ to shift into cells. Therefore, insulin deficiency and hypertonicity (high glucose) promote K+ shifts from ICF to ECF
- Rapid administration of β-blocker.

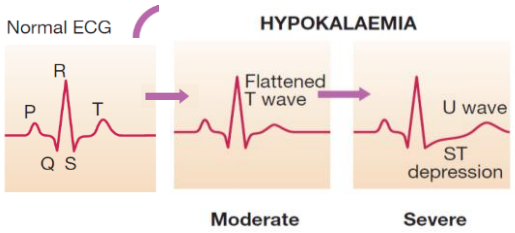
★ Clinical features:
- Arrhythmias—The **most important** effect of hyperkalemia is on the heart. Check an ECG immediately in a hyperkalemic patient. With increasing potassium, ECG changes progress through tall, peaked T waves, QRS widening, PR interval prolongation, loss of P waves, and finally a sine-wave pattern.



3- Renal losses:
- Diuretics. (specially loop diuretics)
- Primary and secondary hyperaldosteronism like in Conn's syndrome[1], Adrenal adenoma secreting aldosterone.
- Excessive glucocorticoids.
- Bartter syndrome[2].

4- Rapid transcellular shift:
- Insulin therapy which cause over stimulation of Na K pump.
- Periodic paralysis (occasional episodes of muscle weakness)
- Epinephrine (β2-agonists) which cause over stimulation of Na K pump. (Salbutamol)
- Certain antibiotics especially Bactrim and amphotericin B.

★ Clinical feature:
- Arrhythmias—prolongs normal cardiac conduction.
- Flattening of T waves on EKG. U waves appear if severe.



- Muscular weakness, fatigue, paralysis, and muscle cramps.
- Decreased deep tendon reflexes.
- Paralytic ileus.
- Polyuria and polydipsia.
- Nausea/vomiting.

[1] Disease of the adrenal glands involving excess production of Aldosterone.
[2] chronic volume depletion secondary to an autosomal-recessive defect in salt reabsorption in the thick ascending limb of the loop of Henle leads to hyperplasia of juxtaglomerular apparatus, which leads to increased renin levels
[2] chronic volume depletion secondary to an autosomal-recessive defect in salt reabsorption in the thick ascending limb of the loop of Henle leads to hyperplasia of juxtaglomerular apparatus, which leads to increased renin levels and secondary aldosterone elevations

- Muscle weakness and (rarely) flaccid paralysis.
- Decreased deep tendon reflexes.
- Respiratory failure.
- Nausea/vomiting, intestinal colic, diarrhea.

★ Treatment

How to lower K level? Our main goal is to push the K outside the body but this can take time

1. Push K into cells: "Shift potassium into the intracellular compartment."
   a. **Insulin**
   b. Sodium bicarbonate:
      • Increases pH level, which shifts K+ into cells.
      • An emergency measure in severe hyperkalemia.
   c. Beta agonists. "very large doses" this stimulates the Na/K ATPase (Salbutamol)
2. **Remove the K load:**
   a. Through the kidney:
      i. • Loop diuretics (furosemide). The loop diuretics is the most efficient, can lower K levels in 1 hour.
      ii. • Dialysis.
   b. Through the gut: Laxatives, K chelation (Ca resonium) this can be efficient but it takes time about 6 hours.
3. **Remember to make the heart less irritable!**
   I.V Ca gluconate Only indicated when there is ECG changes. Or K more than 7.
   (IV calcium was found to decrease the threshold potential in the heart)

- Exacerbates digitalis toxicity.

★ Treatment

How to raise K level?

1st Identify the cause through history and physical.

1- Stop the loss.

2- Replace lost K with K (PO or IV* if rapid correction is urgently needed)

*you can't give a lot of K through peripheral line because it very irritant to the vein can cause thrombophlebitis.

We can't differentiate between hypo or hyperkalemia clinically, most of the patient will present with muscle weakness and fatigue.

Basic knowledge about calcium

★ Where does Ca come from?

→ Total body Ca = **1000 g**. = 1kg

→ Diet: 1000 – 1500 mg /day in average.
→ The normal serum calcium (Ca2+) range is 8.5 to 10.5 mg/dL.

★ Where Does Ca live?

→ The vast majority of total body calcium **(99%)** is present in the **skeleton**.
→ Non-bone calcium represents 1% of total body calcium:
  - Ionic complexes (9%) (Ionized) (calcium phosphate, calcium carbonate, and calcium oxalate)
  - Protein-bound complexes (40%) (Non-Ionized)
  - Free ions (51%) (Non-Ionized)

Ionized form is the active form and the one we actually measure in the serum. What we measure in the serum represent very little amount of total ca, therefore ca range is very narrow (2.1-2.5mmol/L)

★ Why Ca is important (Calcium functions)?

Bone calcium (99%)	Non bone calcium (1%)
▪ Skeletal strength. ▪ Dynamic store. (When body needs calcium, bone will donate to your circulation. When body has excess Ca, the bone will store it. Can be physiologic or pathologic)	▪ Extra- and intracellular signaling. ▪ Nerve impulse transmission. ▪ Muscle contraction. ▪ Cell excitability.

★ What keeps Ca in balance?

1. Total intake.
2. Rate of intestinal absorption.
3. Intestinal excretion.
4. Renal reabsorption. Remember we said the kidney never reabsorbs potassium? here it's different the kidney can both reabsorb and excrete calcium.
5. Renal excretion.
6. Bone turnover.

→ All these parameters are controlled by:

- PTH
- Active Vitamin D
- Serum Ionized Ca level through feedback to the parathyroid gland to secrete more PTH

★ Affect of Albumin and pH in calcium: (Step-up)

Albumin	Changes in pH alter the ratio of calcium binding
Calcium in plasma exists as: 1. Protein-bound form: most calcium ions are bound to albumin, so the total calcium	An increase in pH increases the binding of calcium to albumin. Therefore, in alkalemic states (especially acute respiratory alkalosis), total

concentration fluctuates with the protein (albumin) concentration. 2. Free ionized form: physiologically active fraction; under tight hormonal control (PTH), independent of albumin levels. In **hypoalbuminemia** the total calcium is low, but ionized calcium is normal.	calcium is normal, but ionized calcium is low and the patient frequently manifests the signs and symptoms of hypocalcemia.

★ Hormonal control of calcium:

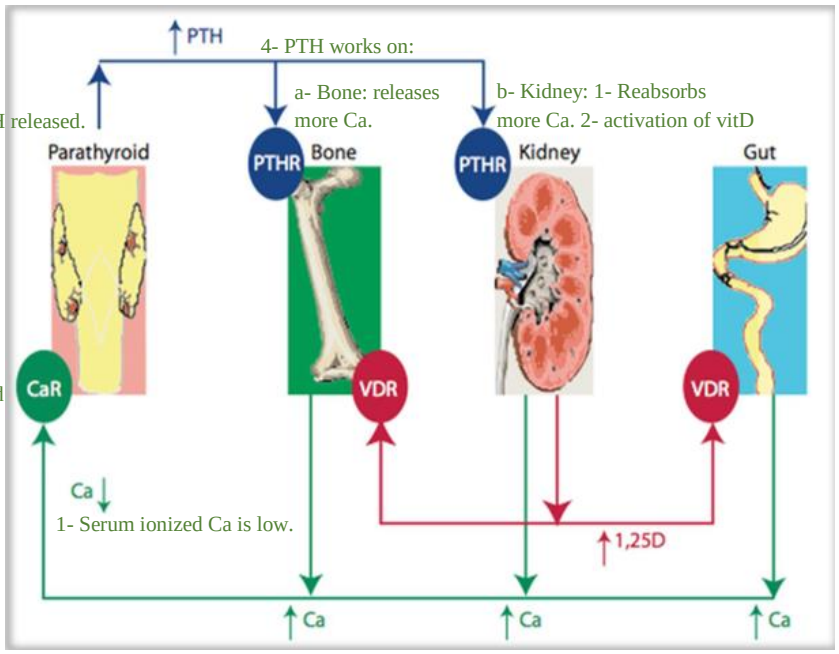
1- PTH is a hyper-calcemic hormone: ↑ plasma Ca + and ↓ plasma PO4 by acting on:

A. **Bone:** ↑ bone resorption.
B. **Kidney:** ↑ Ca.
C. **Activates Vitamin D in the kidney.** (final activation step)

2- Vitamin D is also hyper-calcemic: ↑ plasma Ca + and ↑ plasma PO_4 by acting on:

A. **Bone:** ↑ bone resorption
B. **Kidney:** ↑ Ca^{2+} reabsorption, ↓PO_4 reabsorption.
C. **Intestinal:** ↑ Ca absorption, ↑ PO_4 reabsorption.

* Hormonal mechanisms maintain narrow physiologic range of 10%.



★ What can go wrong?

1. Oral intake.
2. Intestinal absorption IBD, malabsorption, bowel resection all can decrease intestinal absorption.
3. Renal reabsorption.

4. Renal excretion.
5. Intestinal excretion.
6. Bone turnover.
7. Active Vitamin D.

★ Hypercalcemia Vs Hypocalcemia

Hypercalcemia	Hypocalcemia
★ Cases: **1- Increased Intestinal absorption:** ★ Increased Ca intake (milk alkali syndrome) /Vitamin D intake. ★ Sarcoidosis. **2- Increased renal reabsorption:** ★ Secondary to Hyperparathyroidism. ★ Thiazide diuretics. Only causes mild hypercalcemia. **3- Increased bone resorption:** ★ Osteoclastic bone metastasis (bone lysis usually associated with malignancy) ★ Immobilization. ★ Paget disease of the bone. **4- High PTH:** ★ Primary hyperparathyroidism hyperplasia or parathyroid adenoma ★ Multiple Endocrine Neoplasia (rare) **5- High Vit.D:** ★ Vit D Intoxication ★ **Clinical features: initial** symptoms are excessive thirst (polydipsia) and polyuria (nephrogenic diabetes insipidus) **Renal "STONES":** ★ Nephrolithiasis. ★ Nephrocalcinosis (Ca deposits in the interstitium of the Kidney).	**★ Causes** **1- Low intestinal absorption:** ▪ Decreased intake, malabsorption. ▪ Small bowel resection. ▪ Vitamin D deficiency. **2- Low renal absorption:** ▪ Hypoparathyroidism. ▪ Loop diuretics with very high doses. ▪ Tubular defects. ▪ Renal failure. **3- Bone remodeling:** Excessive osteoblastic activity. Hungry bone syndrome (Patients with advanced chronic kidney disease or dialysis patients usually have: ▪ No activation of Vit D → Vit D deficiency→ hypocalcemia ▪ Low GFR→ hyperphosphatemia All activate parathyroid gland to produce PTH, so for many years their bones are exposed to high levels of PTH→ the bones are always in resorption state 'demineralized' and "Hungry" for Ca. When they undergo parathyroidectomy → (PTH) is gone from their circulation → the bone will cause sudden & sharp uptake of Ca (hyper-shift of Ca from the circulation) resulting in severe hypocalcemia) عشان كذا قبل العملية نعطيهم فيتامين دال ونحاول نرفع الكالسيوم لأننا متوقعين نزوله بعد العملية. **4- Low PTH:** ▪ Hypoparathyroidism.

- Nephrogenic diabetes insipidus.
- **Dehydration.**

Skeleton "BONES":

- Bone pain.
- Osteoporosis.
- Osteitis fibrosa cystica in hyperprathyroidism (subperiosteal resorption, bone cyst).

Gastrointestinal "Abdominal Moans":

- Nausea, Vomiting.
- Anorexia, Weight loss.
- Constipation.
- Abdominal pain.
- **Pancreatitis**.
- Peptic ulcer disease.

Neuromuscular "psychic groans":

- Impaired concentration and memory
- Confusion, stupor, coma
- Lethargy and fatigue
- Muscle weakness
- Corneal calcification (band keratopathy)
- Irritability, depression and severe anxiety, psychosis.

Cardiovascular: clinically not important EXCEPT for vascular

- Hypertension
- Shortend QT interval on ECG
- Cardiac arrhythmias
- Vascular calcifications.

Other:

- Itching
- Keratitis, Conjuctivitis

#Mnemonic:
"Painful **bones**, renal **stones**, abdominal **groans**, and psychic **moans**"

5- Low Vit D:

- Renal failure.

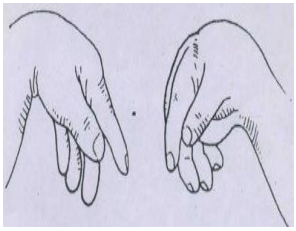
Clinical features: د. رياض: اللي تهمني بالأحمر

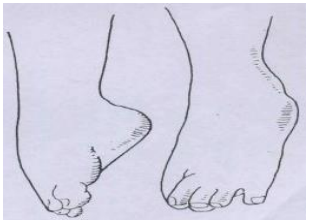
Neuropsychiatric:

- **Seizure** Usual most important presentation.
- Dementia
- Extrapyramidal
- Papillidema
- Cataract

Neuromuscular :

- Paresthesia (numbness) usually pre oral.
- **Spasm** (Tetany): Typically Carpopedal spasm.





Patient with sever hypocalcemia may come with spasm spontaneously, if not you can use the following two tests to elicit the sign:

- Chvostek sign: tapping a facial nerve leads to contraction (twitching) of facial muscles.

- Trousseau sign: Inflate BP cuff to a pressure higher than the patient's systolic BP for 3 minutes (occludes blood flow in forearm). This elicits carpal spasms.

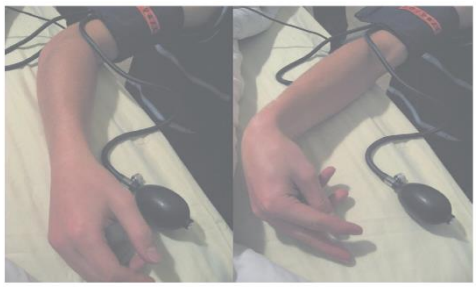


Figure 47.1 Trousseau's sign. Inflation of the sphygmomanometer cuff causes carpal muscle spasm in an individual with hypocalcaemia.

	○ Hyperactive deep tendon reflexes. **Cardiovascular:** ▪ ***Prolonged QT interval***, hypocalcemia should always be in the differential diagnosis for a prolonged QT interval. ▪ Heart failure ▪ Hypotension **Autonomic:** ▪ Biliary colic ▪ Bronchospasm ▪ Diaphoresis

Summary

Electrolyte Imbalance (Potassium and Calcium):		
Potassium (K)	**Basic Information**	• K represents 50 mmol/kg body weight and it comes from our diet. • Majorly intracellular (98% of total body K). • 2% is extracellular and very imp for myocyte function and it is the only one we can measure. → Main importance: Maintains **electrical gradient** across cell membranes i.e.: resting membrane potential. • In order to keep serum K in range we need: 1. Functional Na/K ATPase pump. 2. Intact renal response (primary excretion). • K intake restricted if: 1. GFR is reduced. 2. Existing aldosterone axis dysfunction. 3. Na/K ATPase is not efficient (blocked by drugs or Insulin ↓)
	Hyperkalemia	**Causes:** 1. NA/K ATPase dysfunction. 2. Massive cell breakdown. 3. Impaired renal function. 4. Aldosterone axis dysfunction. **Clinical feature:** • Arrhythmias, on ECG: tall, **peaked T waves**, QRS widening, PR interval prolongation, loss of P waves, and finally a sine-wave pattern. (because hyperkalemia will drop the cardiac threshold, so any action potential can stimulate it) **Treatment (goal: reduce K level)** • Reduce cardiac muscle irritability **with IV Ca gluconate "membrane**

		stabilizer" (only if EKG changes)
		• Push K into cells through:
		  ○ Insulin
		  ○ Sodium bicarbonate (if pt has acidosis)
		  ○ Beta agonists (Salbutamol 'requires high dose')
		• Remove K load:
		  ○ Through kidney loop diuretics (furosemide)
		  ○ Through gut: Laxatives, K chelation (Ca resonium)

	Hypokalemia	**Causes:** • GI losses: diarrhea – laxatives • Renal losses: diuretics – hyperaldosteronism • Insufficient dietary intake: malnutrition – eating disorders • Rapid transcellular shift: insulin - epinephrine **Clinical feature:** ▪ Arrhythmias, on ECG: prolonged normal cardiac conduction and flattening of T waves. U waves appear if severe.
Calcium (Ca)	**Basic Information**	▪ 99%of Ca in skeleton (skeletal strength – dynamic store) and 1% is non-bone Ca (cell signaling – nerve impulse transmission - muscle contraction) • **Ca balance is kept by:** total intake - rate of intestinal absorption and excretion - renal reabsorption and excretion - bone turnover • **All parameters above are controlled by:** PTH (bone – kidney)– Active VitD (bone – kidney – gut) – Serum Ionized Ca level • Hormonal mechanisms (PTH – VitD both increase Ca) maintain narrow physiologic range of 10%
	Hypercalcemia	**Causes:** • Increased Intestinal absorption: increased Ca/VitD intake • Increased renal reabsorption: Secondary to Hyperparathyroidism - Thiazide diuretics • Increased bone resorption: osteoclastic bone metastasis - immobilization • High PTH: primary hyperparathyroidism - Multiple Endocrine Neoplasia • High VitD: VitD intoxication • Causes not related to ca intake: Granuloma (which produces Ca) – Sarcoidosis. **Clinical features:** • Cardiovascular: **ECG shows shortened QT interval** • Neuromuscular: muscle weakness – fatigue – lethargy – impaired memory • Stones: Nephrocalcinosis - Nephrogenic diabetes insipidus - Dehydration • Bones: pain • GIT: abdominal pain - peptic ulcer – **pancreatitis** - constipation – nausea – vomiting.

	Hypocalcemia	**Causes:** • Low intestinal absorption: decreased intake - malabsorption - small bowel resection - VitD deficiency • Low renal absorption: hypoparathyroidism - loop diuretics (because it decreases Ca reabsorption) - tubular defects - renal failure • Bone remodeling: hungry bone syndrome • Low PTH: hypoparathyroidism • Low VitD: renal failure **Clinical features:** • **Cardiovascular: Prolonged QT interval** – HF – HTN • **Increased neuromuscular irritability:** paresthesia - spasm (tetany): (Chvostek sign - Trousseau sign) • **Neuropsychiatric:** seizure - dementia - extrapyramidal - papilledema – cataract.

Questions

1. What is the mechanism behind using insulin in treatment of hyperkalemia?

A. Increase renal loss of K

B. Trans shift of K

C. Cell lysis

D. Help cardiac membrane from damage

2. A 65-year-old diabetic man with a creatinine of 1.6 was started on an angiotensin-converting enzyme inhibitor for hypertension and presents to the emergency room with weakness. His other medications include atorvastatin for hypercholesterolemia, metoprolol and spironolactone for congestive heart failure, insulin for diabetes, and aspirin. Laboratory studies include:

K: 7.2 mEq/L

Creatinine: 1.8 mg/dL

Glucose: 250 mg/dL

CK: 400 IU/L

A. Worsening renal function

B. Uncontrolled diabetes

C. Statin-induced rhabdomyolysis

D. Drug-induced effect on the renin-angiotensin-aldosterone system

3. A 21-year-old woman complains of urinary frequency, nocturia, constipation and polydipsia. Her symptoms started 2 weeks ago and prior to this she would urinate twice a day and never at night. She has also noticed general malaise and some pain in her left flank. A urine dipstick is normal. The most appropriate investigation is:

A. Serum phosphate
B. Serum calcium
C. Parathyroid hormone (PTH)
D. Plasma glucose

4. Which of the following is an indication for treatment with IV Ca gluconate for a patient with hyperkalemia?

A. Respiratory failure
B. Nausea/vomitting
C. Peaked T wave
D. Muscle weakness

5. Which of the following ECG changes can be found in a patient with hypercalcemia?

A. Peaked T wave
B. ST elevation
C. U wave
D. Prolonged Q-T interval

6. Which patient is at risk for hyperkalemia?

A. A patient with parathyroid cancer
B. Patient with Cushing's Syndrome
C. Patient with Addison's Disease
D. Patient with breast cancer

7. Which of the following is not a known cause of hypercalcemia?

A. Sarcoidosis
B. Use of thiazide diuretics
C. Loop diuretics
D. High intake of Vit D.

8. A 27-year-old alcoholic man presents with decreased appetite, mild generalized weakness, intermittent mild abdominal pain, perioral numbness, and some cramping of his hands and feet. His physical examination is initially normal. His laboratory returns with a sodium level of 140 mEq/L, potassium 4.0 mEq/L, calcium 6.9 mg/dL, albumin 3.5 g/dL, magnesium 0.7 mg/dL, and phosphorus 2.0 mg/dL. You go back to the patient and find that he has both a positive Trousseau and a positive Chvostek sign. Which of the following is the most likely cause of the hypocalcemia?

A. Poor dietary intake
B. Hypoalbuminemia
C. Pancreatitis
D. Decreased end-organ response to parathyroid hormone because of hypomagnesemia

Answers:

1. B
2. D
3. B
4. C
5. D
6. C
7. C
8. D