

# Body Cations: K and Ca

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Medicine 341

# POTASSIUM

- Where does K come from?
- How much K do we eat every day?
- How do we lose the K?
- Where does K in the body live?
- How does K move?
- Is K important?
- What keeps K in normal range ?
- What happens if K level is abnormal?
- What causes high K?
- What causes low K ?

# Where does K come from?

- Depending on diet, the normal daily intake can vary
- Fruits, potatoes, beans, and grains
- 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

# How do we loose K ?

- **Renal clearance**

- primary mechanism

- Very efficient until  $GFR < 30$  ml/min

- **Intestinal excretion**

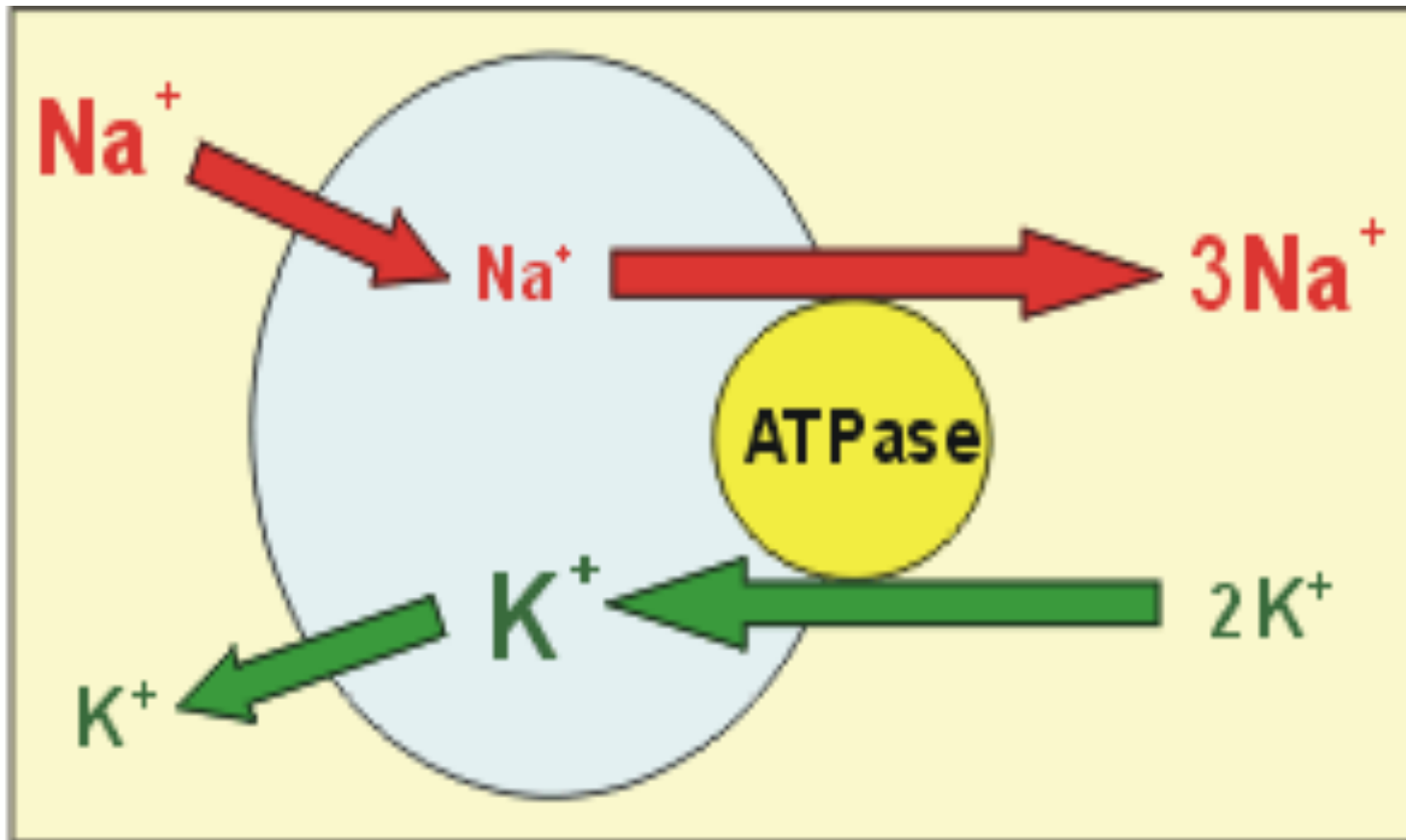
- Only handles 10 % of the daily K load

- Efficiency can be enhanced in renal failure but it is variable form one person to another

# Where does K live in the body ?

- Total body **K** is approximately 50 mmol/kg body weight
- **K** is the most abundant intracellular cation (100- 150 mmol/l) → 98 % of total body **K**
- Extracellular **K** concentration (3.4 – 5.5 mmol/L) → 2% of total body **K**

# What keeps the IC K high ?



- Insulin , Beta agonists enhance the pump function
- Beta Blockers inhibit the pump function

# What keeps EC K low?

- The Na/K ATPase pump
- Renal clearance : requires normal GFR and normal aldosterone axis
- Intestinal excretion

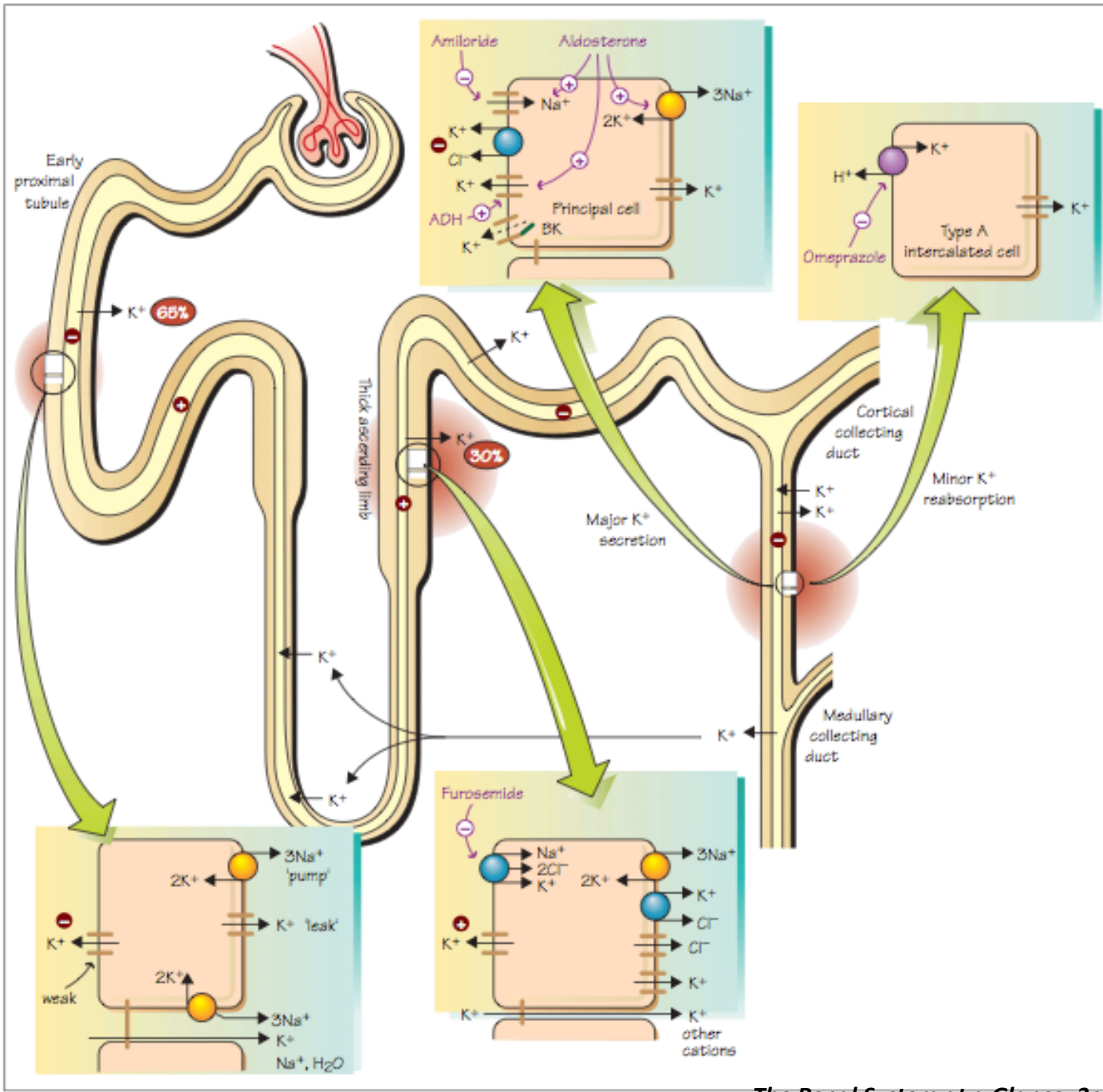


# What happens when we eat K ?

- Oral **K** intake is initially absorbed in the intestine and enters portal circulation
- increased ECF **K** stimulates insulin release
- insulin facilitates **K** entry into intracellular compartment by stimulating cell membrane Na/K ATPase pump
- The transient rise in serum K stimulates renal and intestinal clearance of extra K

# In order to Keep serum K in normal range, we need:

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



The Renal System at a Glance, 3e. By Chris O'Callaghan

# 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?

- Skeletal muscle dysfunction: weakness and paralysis
- Cardiac cell irritability: arrhythmia

## NA/K ATPase dysfunction

- B blockers
- Digoxin
- ↓Insulin

## Massive Cell breakdown

- Rhabdomyolysis
- Tumor lysis syndrome

**Hyperkalemia**  
**[K]>5.5**

## Impaired Renal function

## Aldosterone axis dysfunction

- Adrenal deficiency
- Aldosterone resistance

# 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 ↓ )

↓ Oral intake

- Malnutrition
- eating disorders

**Rapid transcellular shift**

- Insulin therapy
- Periodic paralysis

**Hypokalemia**

**[K] < 3.4**

↑ **Renal loss**

- Diuretics
- too much aldosterone

↑ **Intestinal loss**

- Diarrhea
- Laxative abuse



# How to raise K level?

- Stop the loss
- Replace lost K with K ( PO or IV if rapid correction is urgently needed)

# How to lower K level ?

- Reduce Cardiac muscle irritability with Ca gluconate (only if EKG changes)
- Push K into cells: Insulin , Beta agonists
- Remove the K load
  - Through the kidney: diuretics, dialysis
  - Through the gut : Laxatives, K chelation(Ca resonium), Patiromer, Zirconium

# Calcium balance

# Calcium

- Where does Ca come from?
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- How does Ca move?
- Why is Ca important?
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# Where does Ca come from?

- Diet : 1000 – 1500 mg /day in average
- Total body Ca = 1000 g

# 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
    - ✓ *free ions (51%)*
    - ✓ *protein-bound complexes (40%)*
    - ✓ *ionic complexes (9%) [calcium phosphate, calcium carbonate, and calcium oxalate]*
- } **Non Ionized**

# Why Ca is important ?

- Bone Ca
  - ✓ *skeletal strength*
  - ✓ *dynamic store*
  
- Non-Bone Ca
  - ✓ *extra- and intracellular signaling*
  - ✓ *nerve impulse transmission*
  - ✓ *muscle contraction*

# What keeps Ca in balance ?

- Total intake
- Rate of intestinal absorption
- Intestinal excretion
- Renal reabsorption
- Renal excretion
- Bone turnover

All these parameters are controlled by

1- PTH

2- Active Vitamin D

3- Serum Ionized Ca level

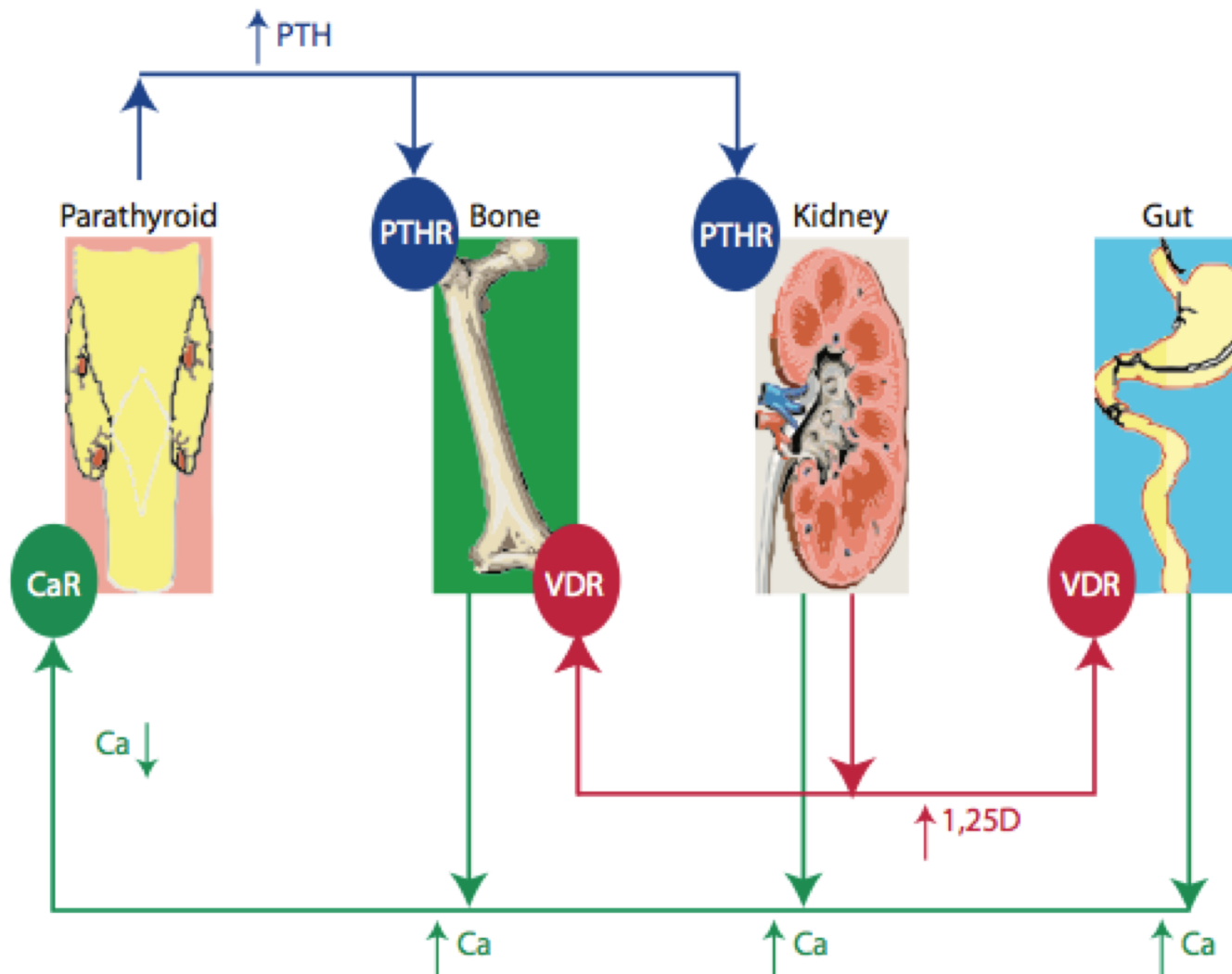


# PTH is a **hyper**-calcemic hormone

- ↑ Release of Ca from bones (bone resorption)
- ↑ Renal absorption of Ca
- Activates Vitamin D in the kidney

# Active Vitamin D is also **hyper**-calcemic

- ↑ Intestinal absorption of Ca
- ↑ Bone resorption



**Hormonal mechanisms maintain narrow physiologic range of 10%.**

# What can go wrong ?

- Oral intake
- Intestinal absorption
- Renal reabsorption
- Renal excretion
- Intestinal excretion
- Bone turnover

- PTH
- Active Vitamin D

**↑ Intestinal absorption**

- Increased intake
- Increased Vit D

**↑ Renal reabsorption**

- Hyperparathyroidism
- Thiazide diuretics

**Hypercalcemia**

**↑ Bone resorption**

- Osteoclastic bone metastasis
- Immobilization

**↑ PTH**

- Primary hyperparathyroidism
- Multiple Endocrine Neoplasia

**↑ Vit D**

- Intoxication

## Clinical Manifestations of Hypercalcemia

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### **Renal "stones"**

Nephrolithiasis  
Nephrogenic diabetes insipidus  
Dehydration  
Nephrocalcinosis

### **Skeleton "bones"**

Bone pain  
Arthritis  
Osteoporosis  
Osteitis fibrosa cystica in  
hyperparathyroidism (subperiosteal  
resorption, bone cysts)

### **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)

### **Cardiovascular**

Hypertension  
Shortened QT interval on  
electrocardiogram  
Cardiac arrhythmias  
Vascular calcification

### **Other**

Itching  
Keratitis, conjunctivitis

## ↓ Intestinal absorprtion

- Decreased intake
- mal-absorption
- Small bowel resection
- Vit D deficiency

## ↓ Renal reabsorprtion

- hypo-parathyroidism
- Loop diuretics
- Tubular defects

## Hypocalcemia

## Bone remodeling

- Hungry bone syndrome

## ↓ PTH

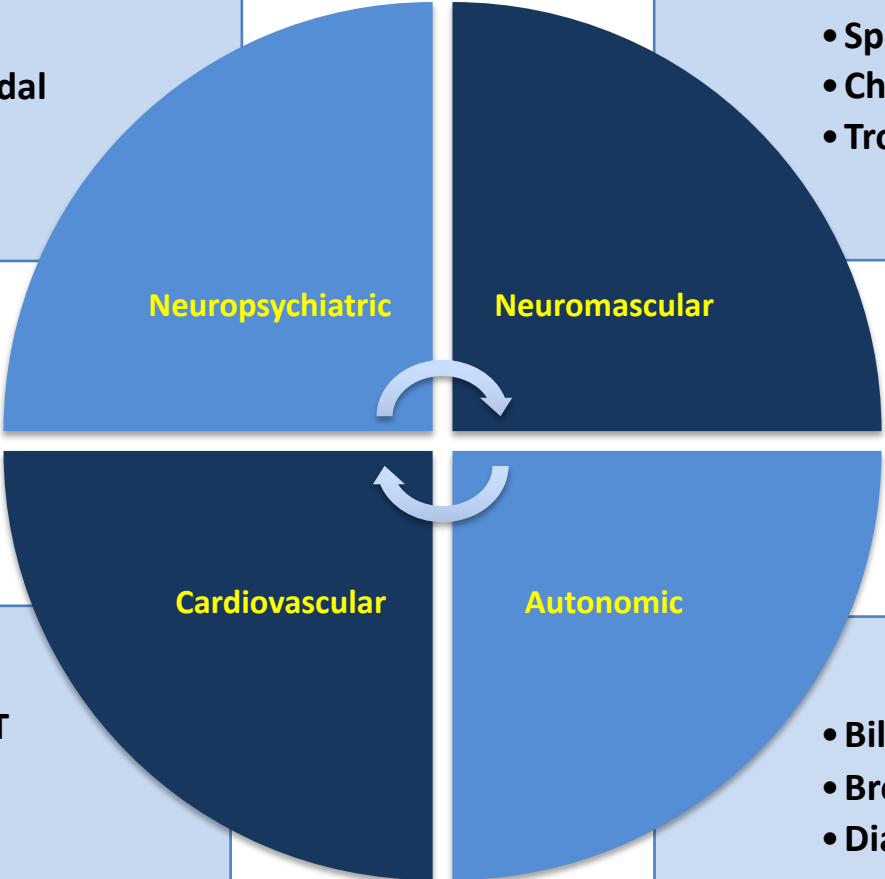
- hypoparathyroidism

## ↓ Vit D

- Renal failure

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- Seizure
- Dementia
- Extrapyrimalidal
- Papillidema
- Cataract



- Parasthesia
- Spasm
- Chvostek's sign
- Trousseau's sign

- Prolonged QT interval
- Heart failure
- Hypotension

- Biliary colic
- Bronchospasm
- Diaphoresis



**Thank You**