Body Cations: K and Ca

Dr. Riyadh Al Sehli, MBBS, FRCPC
Transplant Nephrologist
Medicine 341

POTASSIUM

- Where does **K** come form?
- How much **K** do we eat every day?
- How do we loose 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 form?

- 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

Pediatr Nephrol (2011) 26:377–384

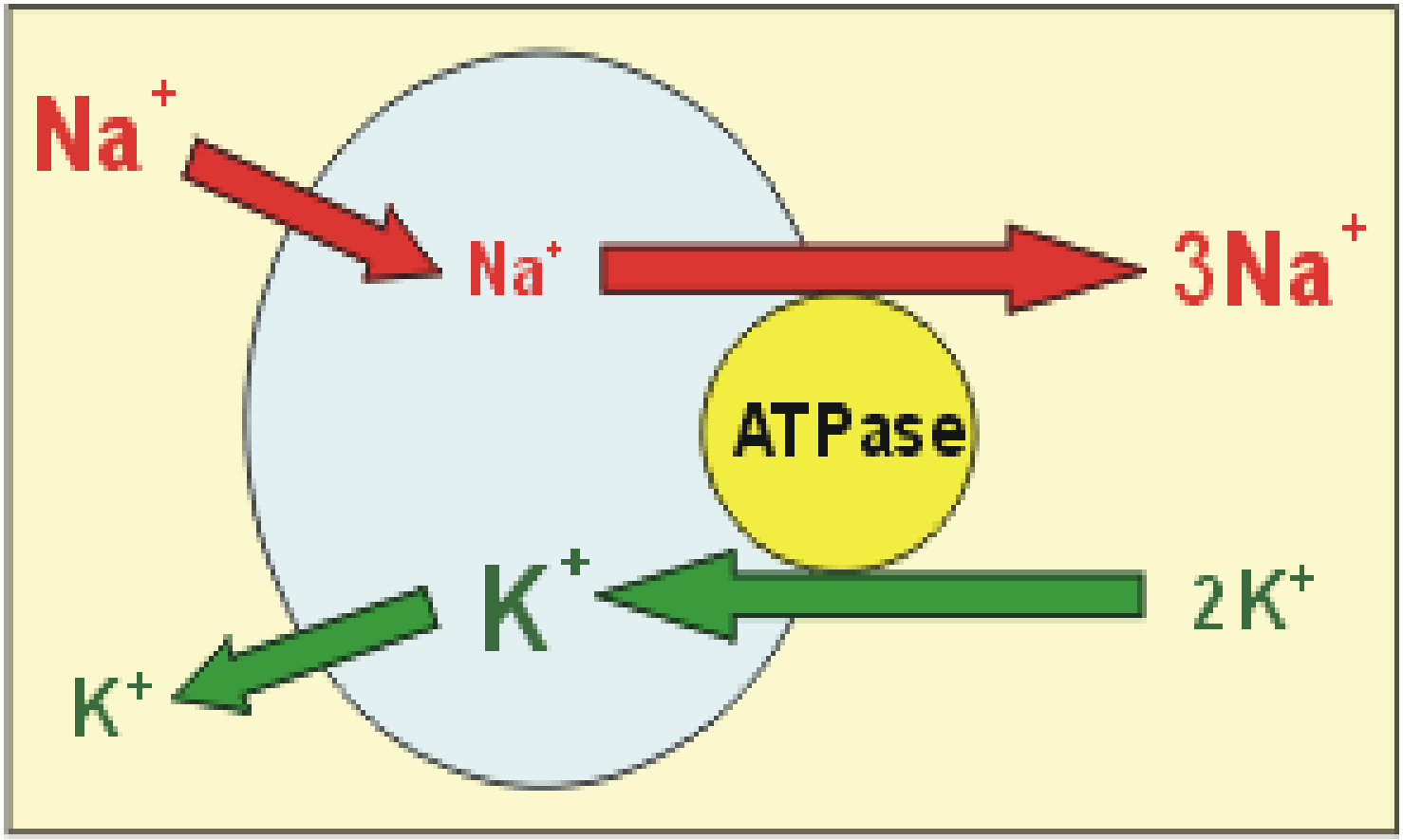
How do we loose **K** ?

- **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?

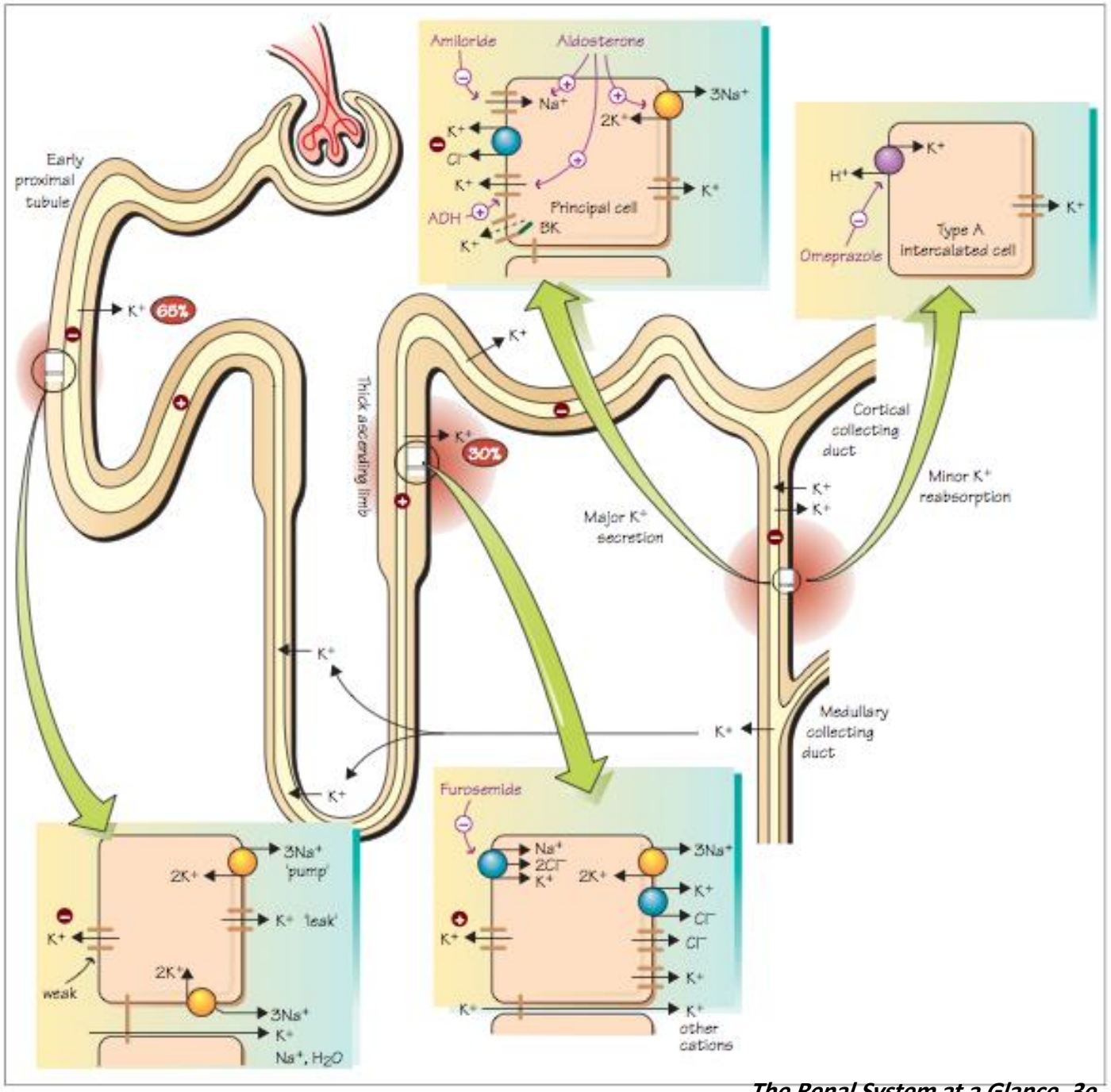
- The Na/K ATPase pump
- Renal clearance : requires normal GFR and normal aldosterone axis
- Intestinal excretion

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.

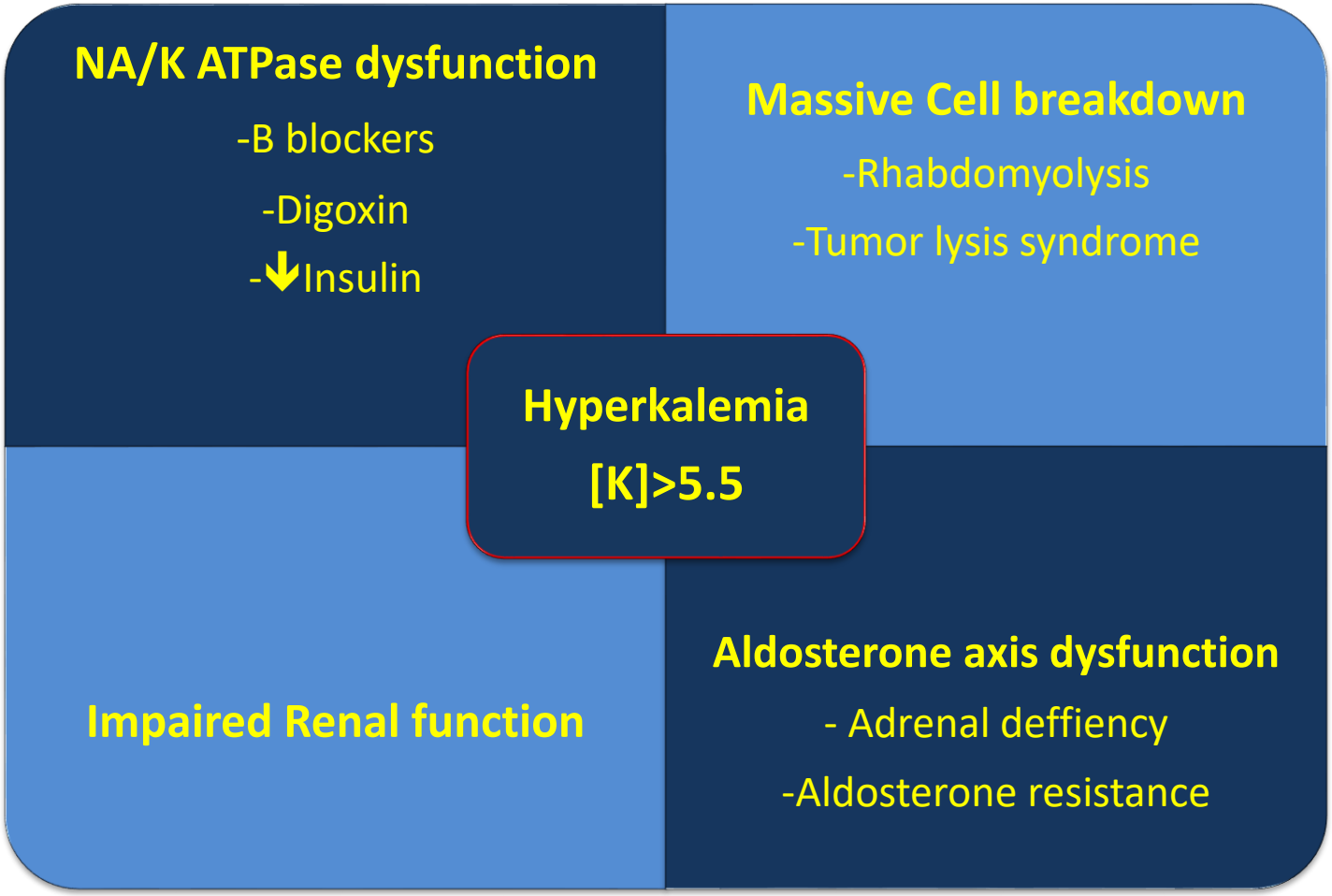
➢ *resting membrane potential essential for generation of action potential*

- Essential for intracellular metabolism e.g protein synthesis

What happens of **K** level is abnormal?

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

Pediatr Nephrol (2011) 26:377–384


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 deffiency
-Aldosterone resistance

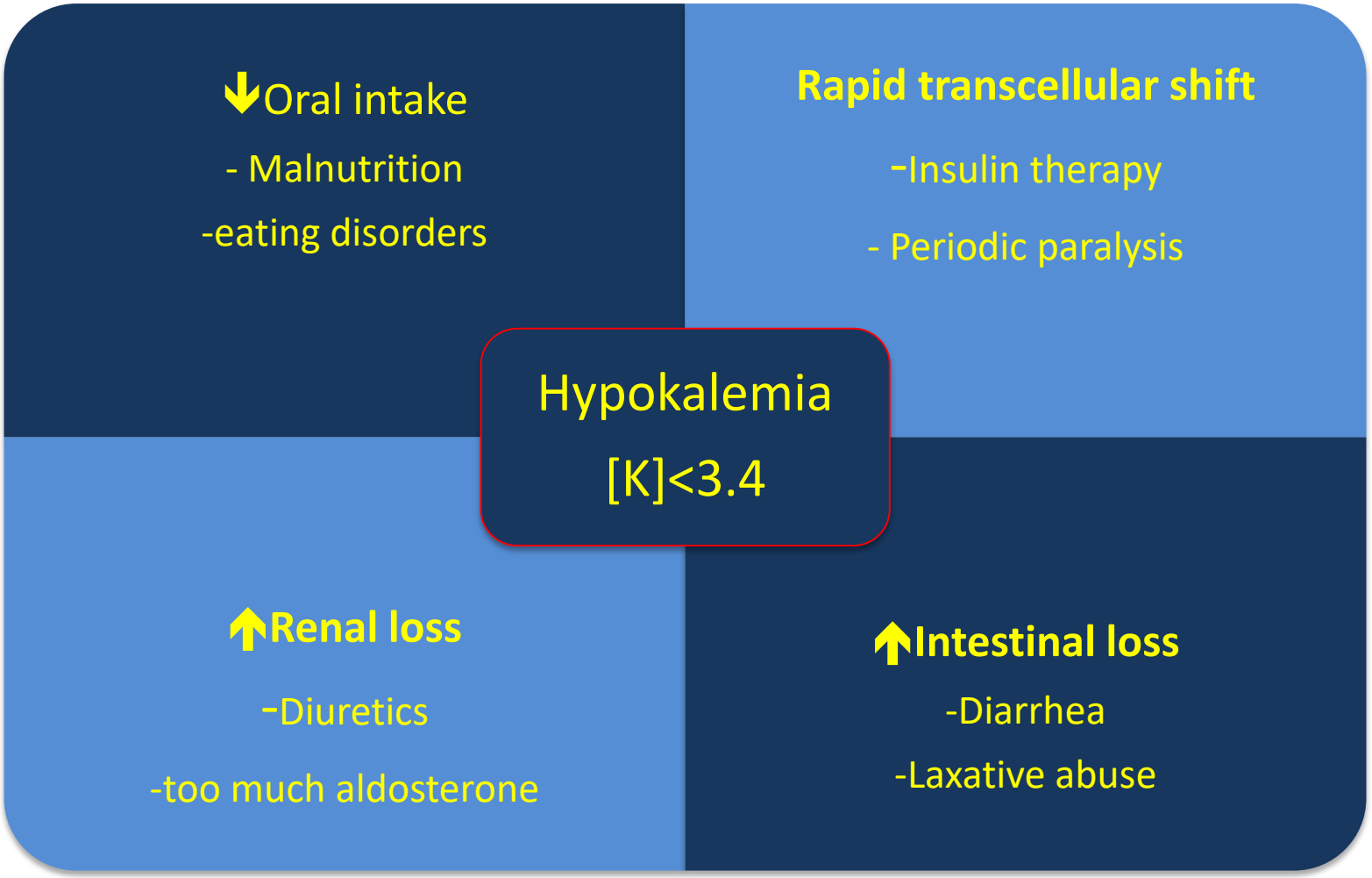
Can you eat too much **K** ?

- 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)

Calcium balance

Calcium

- Where does **Ca** come form?
- How much **Ca** do we eat every day?
- How do we loose the **Ca**?
- Where does **Ca** in the body live?
- How does **Ca** move?
- Why is **Ca** important?
- What keeps **Ca** in normal range ?
- What happens if **Ca** level is abnormal?
- What causes high **Ca**?
- What causes low **Ca** ?

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%)*

Non Ionized

✓ *ionic complexes (9%) [calcium phosphate, calcium carbonate, and calcium oxalate]*

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

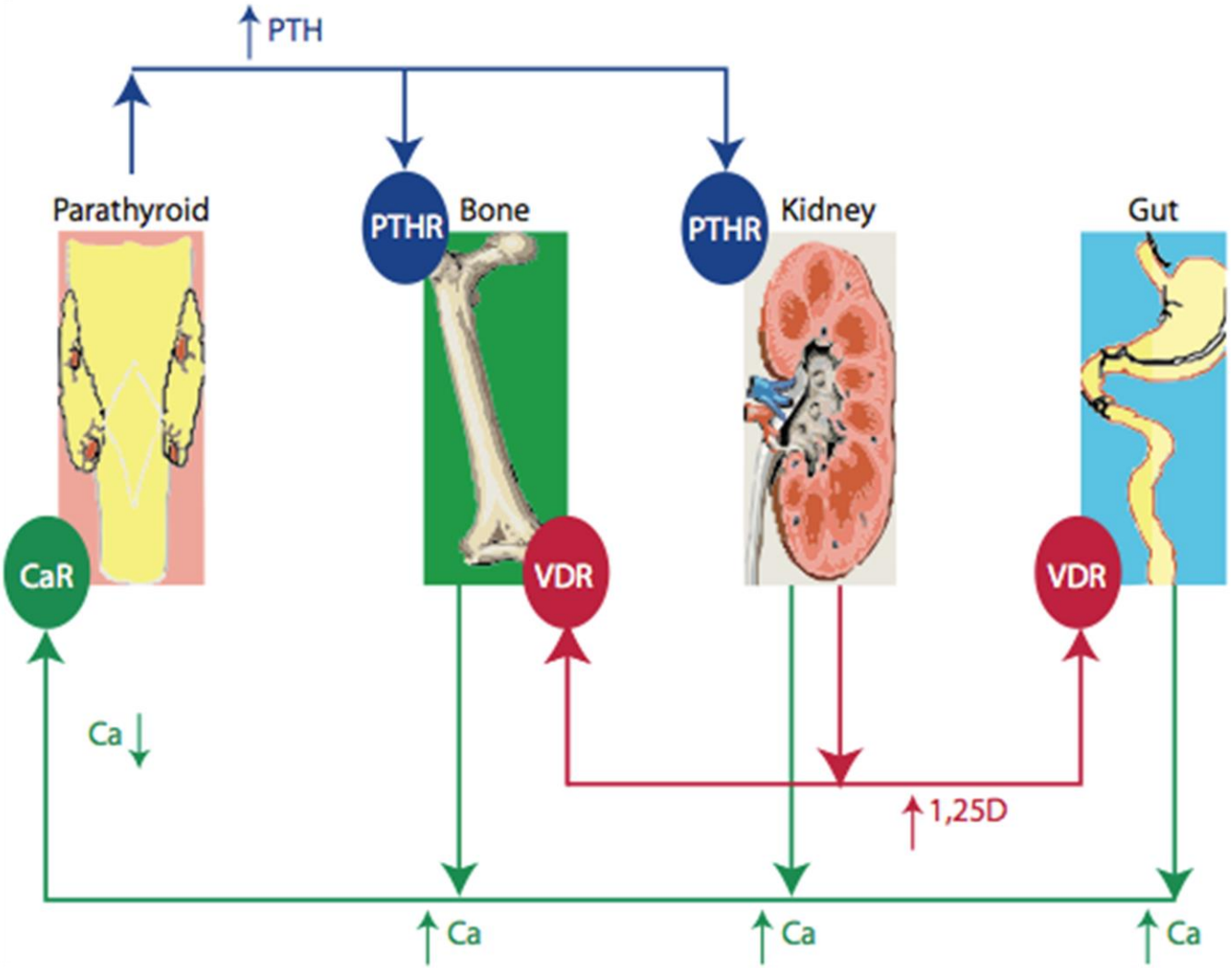
All these parameters are controlled by
1- PTH
2- Active Vitamin D
3-Serum Ionized Ca level

PTH is a **hyper**-calcemic hormone

- ↑Release of Ca form 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



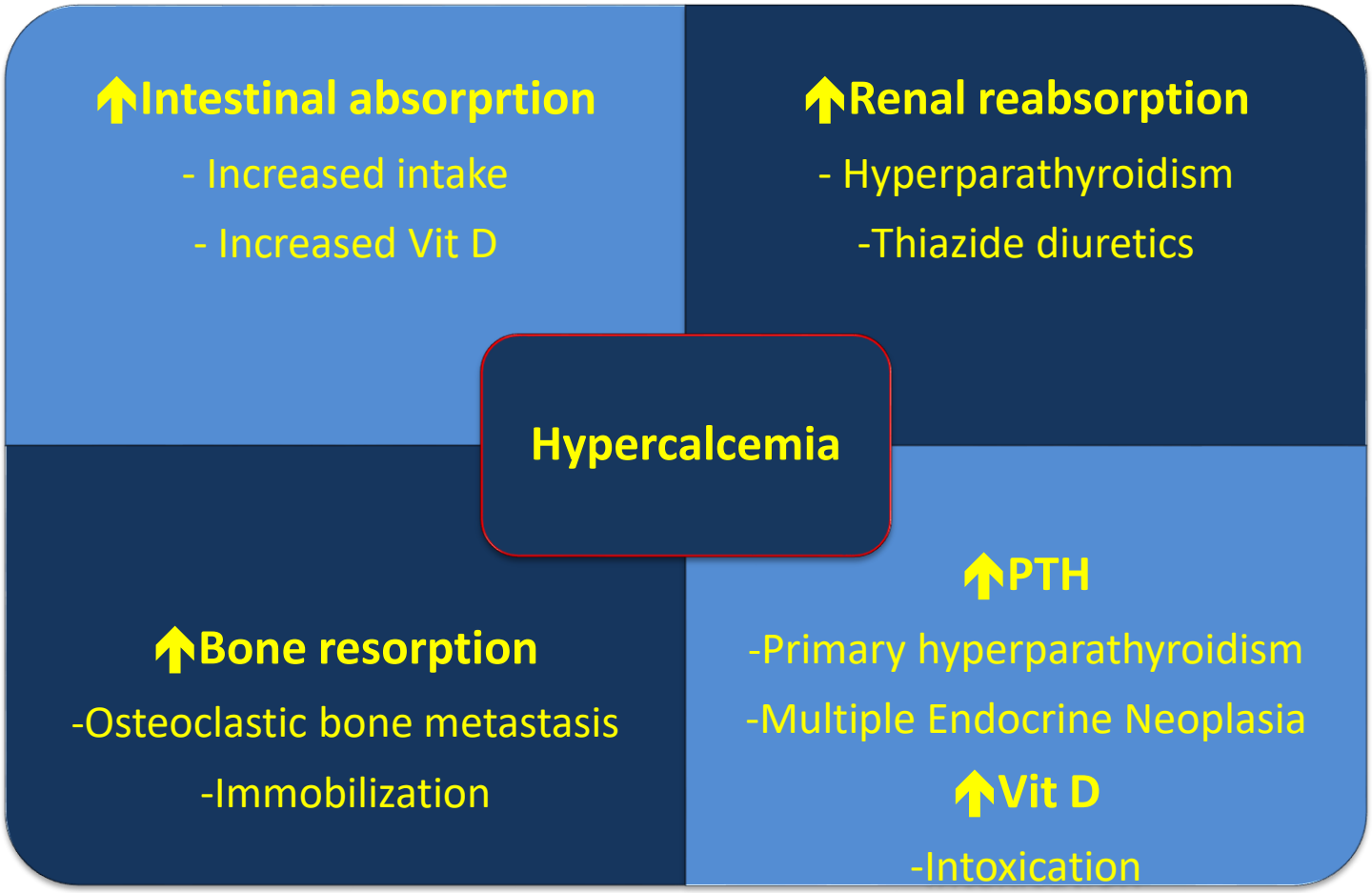
Hormonal mechanisms maintain narrow physiologic range of 10%.

Clin J Am Soc Nephrol 5: S23–S30, 2010

What can go wrong ?

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

- PTH
- Active Vitamin D


↑Intestinal absorprtion
- 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

Renal "stones"
Nephrolithiasis
Nephrogenic diabetes insipidus
Dehydration
Nephrocalcinosis

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

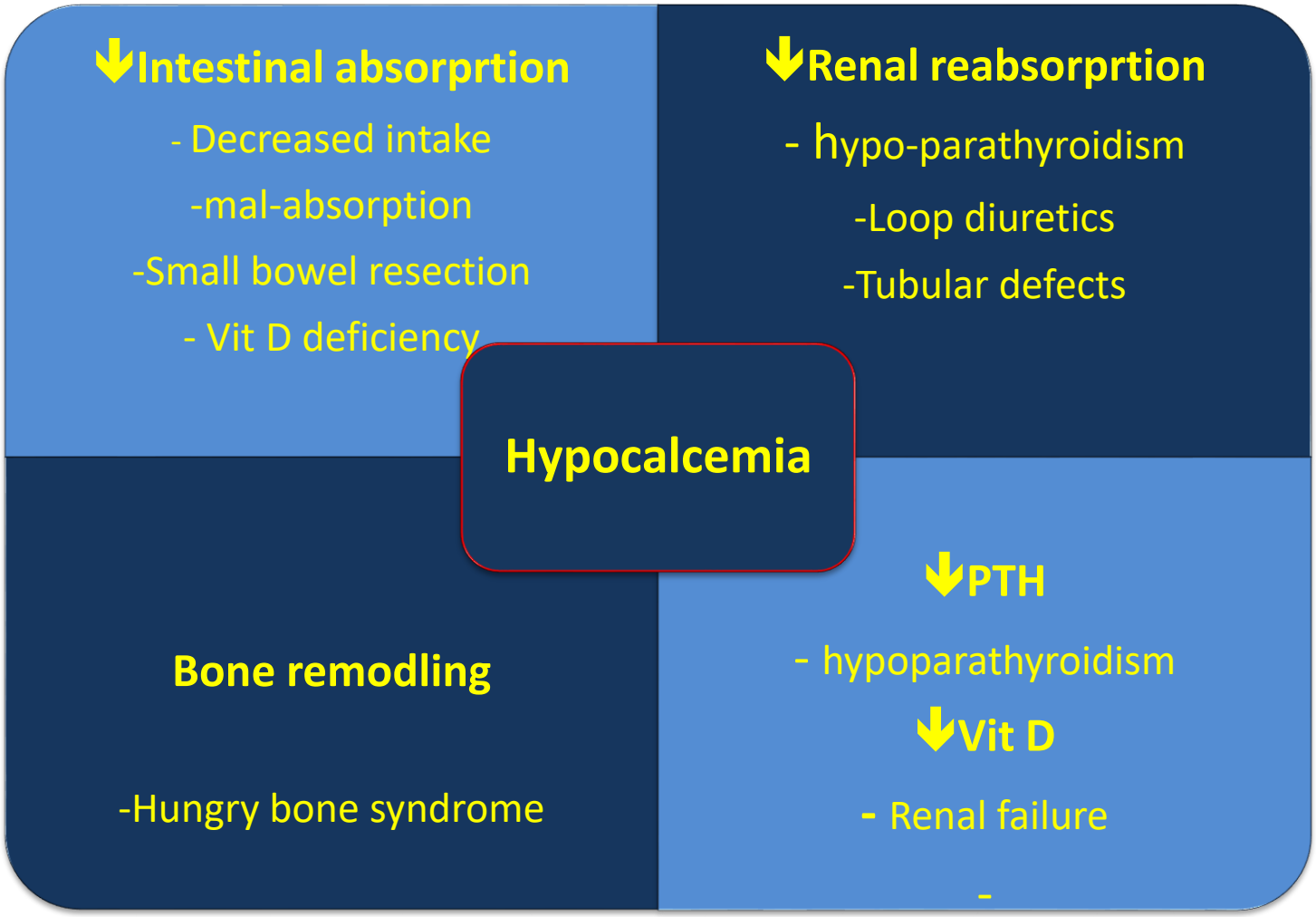
Gastrointestinal "abdominal moans"
Nausea, vomiting
Anorexia, weight loss
Constipation
Abdominal pain
Pancreatitis
Peptic ulcer disease

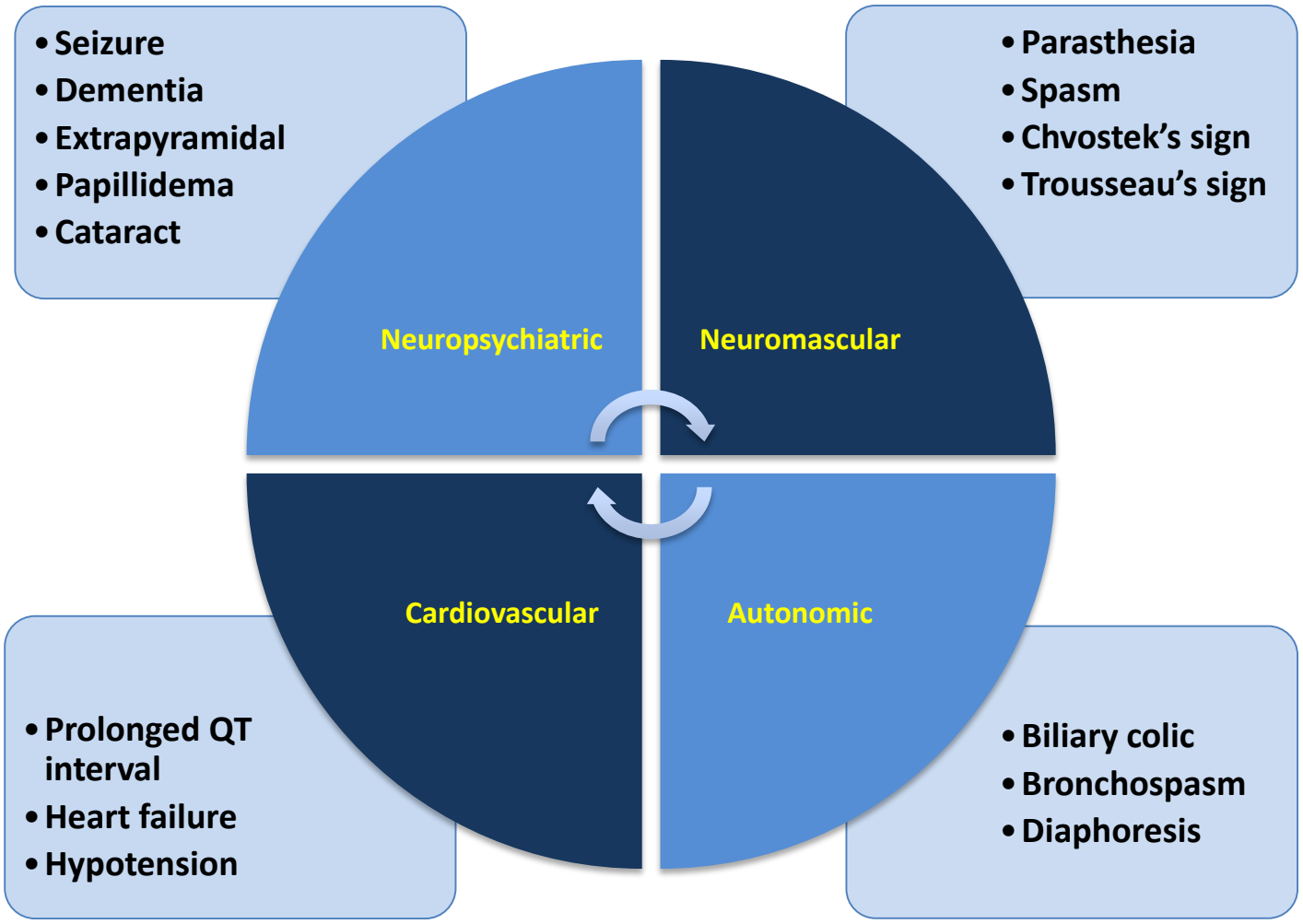
Neuromuscular "psychic groans"
Impaired concentration and memory
Confusion, stupor, coma
Lethargy and fatigue
Muscle weakness
Corneal calcification (band keratopathy)

Cardiovascular
Hypertension
Shortened QT interval on electrocardiogram
Cardiac arrhythmias
Vascular calcification

Other
Itching
Keratitis, conjunctivitis

Am Fam Physician 2003;67:1959-66


↓Intestinal absorprtion
- Decreased intake
-mal-absorption
-Small bowel resection
- Vit D deficiency
↓Renal reabsorprtion
- hypo-parathyroidism
-Loop diuretics
-Tubular defects
Hypocalcemia
Bone remodling
-Hungry bone syndrome
↓PTH
- hypoparathyroidism
↓Vit D
- Renal failure
-


• Seizure
• Dementia
• Extrapyramidal
• Papillidema
• Cataract
• Parasthesia
• Spasm
• Chvostek's sign
• Trousseau's sign
Neuropsychiatric
Neuromascular
Cardiovascular
Autonomic
• Prolonged QT interval
• Heart failure
• Hypotension
• Biliary colic
• Bronchospasm
• Diaphoresis

Thank You