

K^+/Na^+ IMBALANCE SEMINAR

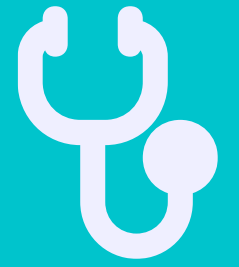
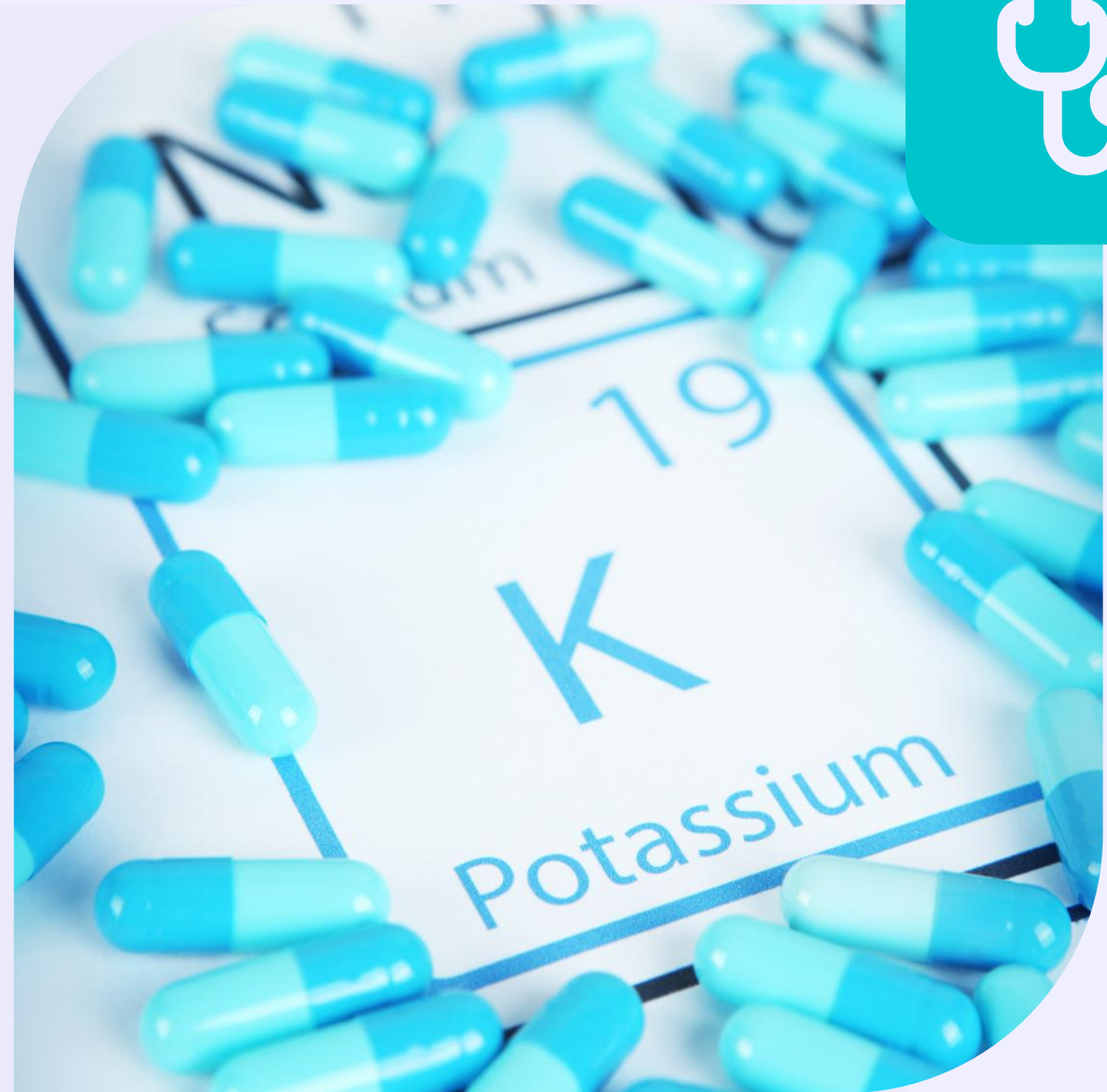


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Physiology of Na regulation

- primary cation in ECF which determines ECF osmolarity

- **NA BALANCE REGULATION**

1. Na balance (Na excretion = Na intake)
2. Positive Na balance: increase Na retention → Na in ECF → ECF expansion → increase blood volume and blood pressure
3. Negative Na balance: increase excretion of Na → decrease Na in ECF → ECF contraction → decrease blood volume and blood pressure
4. Na is filtered freely from the glomerulus and reabsorbed from the tubular system
5. Some of Na is excreted in urine

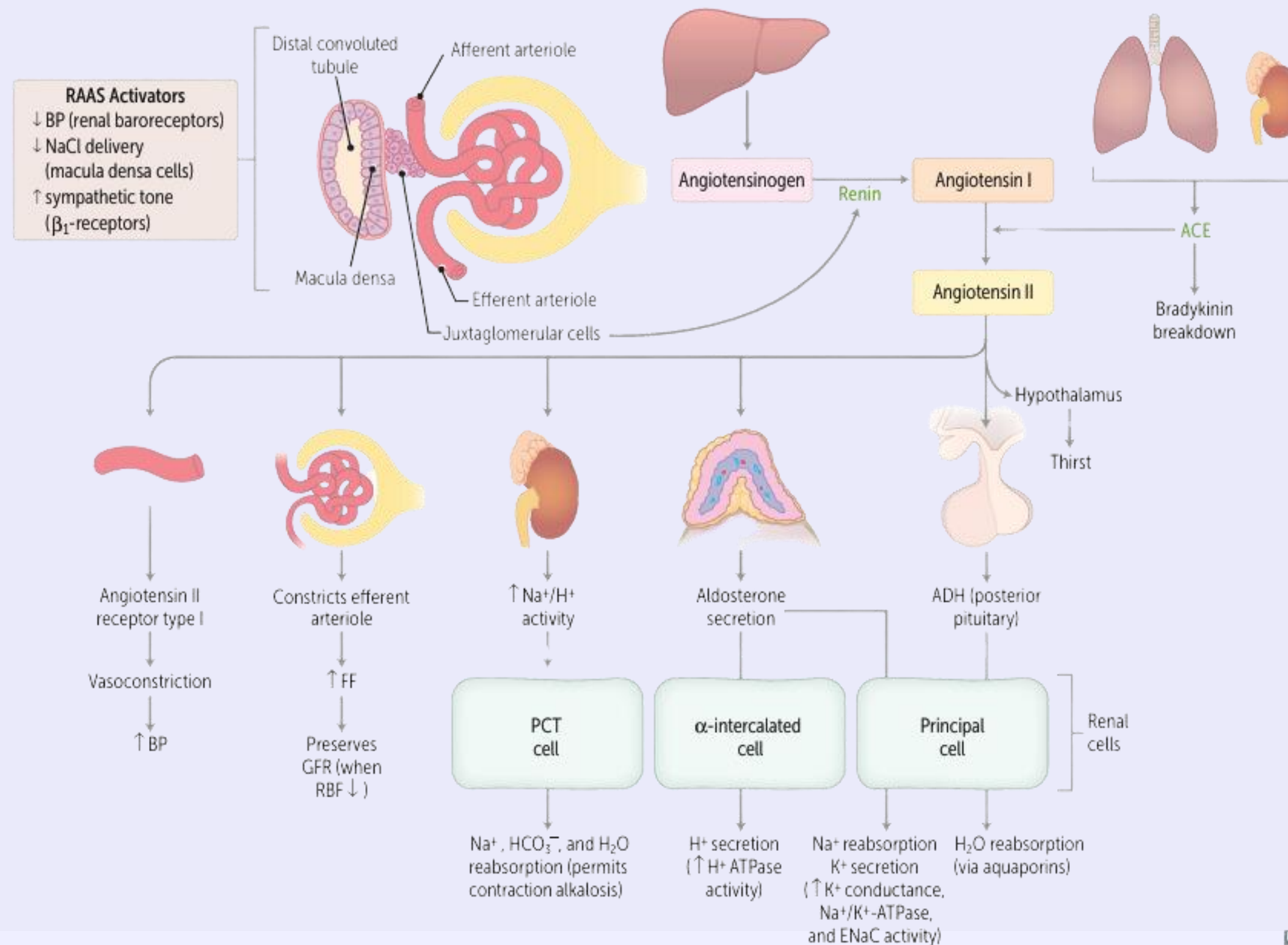
- **Effective arterial blood volume (EABV)**

1. Any change in ECF will cause a parallel change in EABV
2. Edema: fluid filtered into interstitial space → increase ECF → increase EABV (increase BP) → Na excretion altered by kidneys (attempts to restore normal EABV)

Physiology of Na regulation

- Renin angiotensin aldosterone system (RAAS)

Renin-angiotensin-aldosterone system

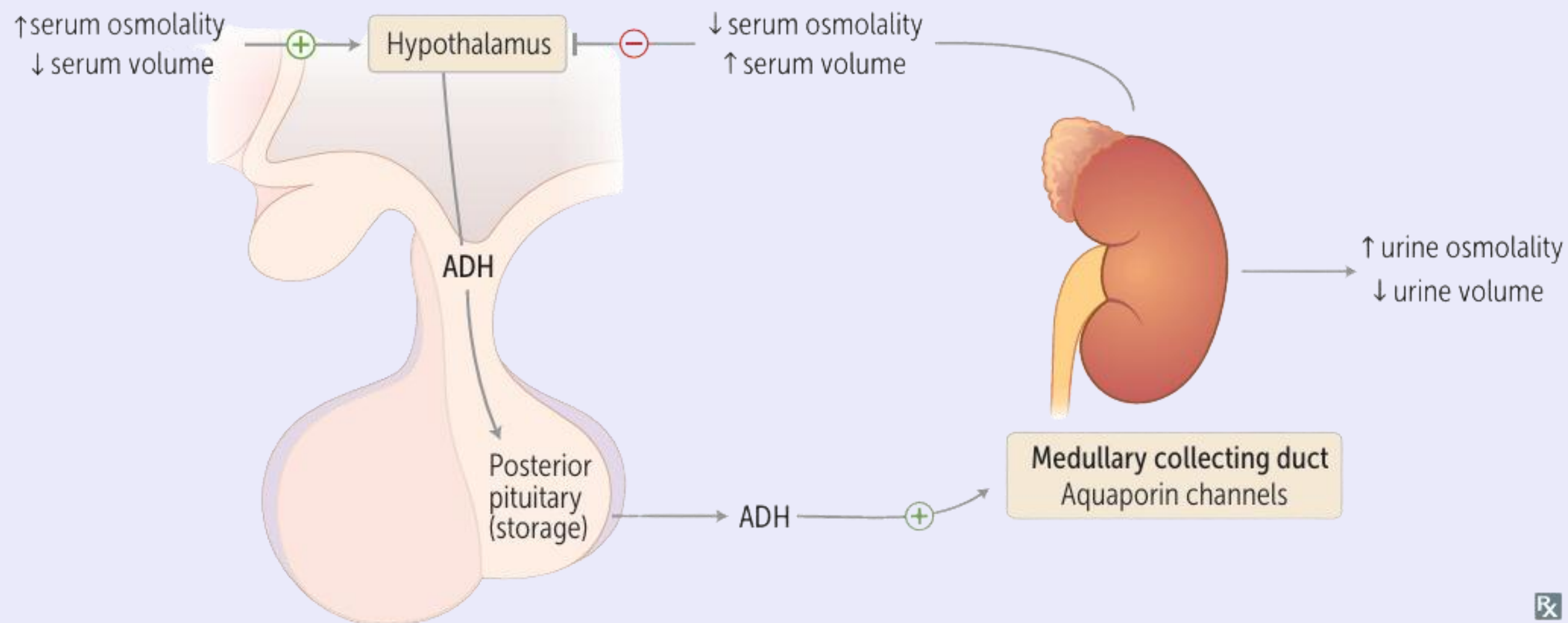


Physiology of Na regulation

- **Atrial Natriuretic peptide (ANP)**
 - Released from atrial myocytes in response to high blood volume and atrial pressure
 - ANP causes :
 1. vasodilation and + Na reabsorption at the renal collecting tubule
 2. Dilates afferent renal arterioles and constricts efferent arterioles, promoting diuresis and contributing to the "aldosterone escape" mechanism.
 3. Acts opposite to angiotensin 2
- **Brain Natriuretic peptide (BNP)**
 1. Released from ventricular myocytes in response to increased tension.
 2. Similar physiologic action to ANP
 3. Useful for diagnosing heart failure

Physiology of Na regulation

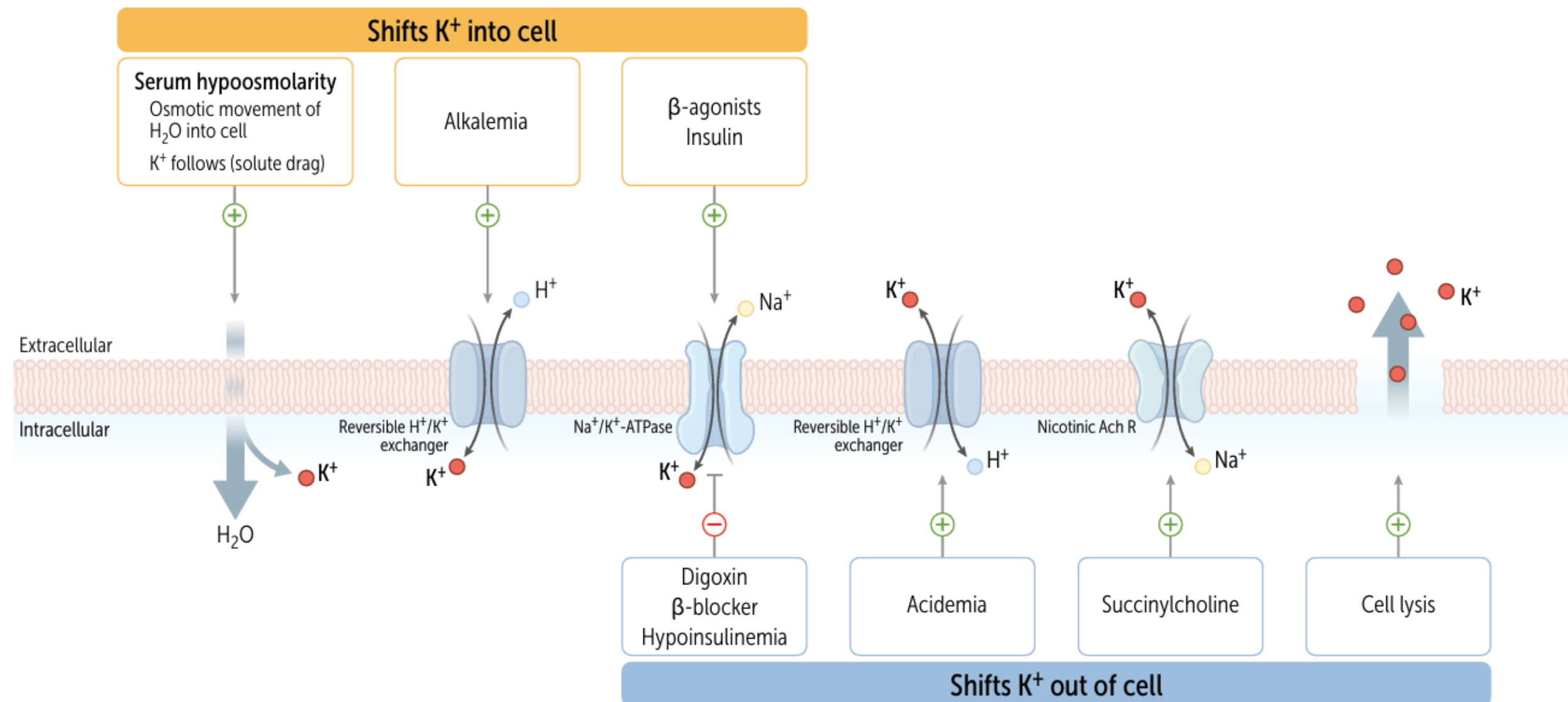
- **Anti diuretic hormone (ADH)**
- Source: supraoptic and paraventricular nuclei in posterior pituitary
- Regulates blood pressure (V1-receptors) and serum osmolality (V2-receptors)
- ADH induces reabsorption of water causing:
 1. increase in urine osmolarity
 2. Decrease in serum osmolarity



Physiology of K regulation

- Potassium (K^+): primary intracellular cation (98%)
- Regulates intracellular osmolarity
- Concentration gradient across cell membrane establishes resting membrane potential, essential for excitable cell function (e.g. myocardium)
- Potassium (K^+) is regulated mainly via Na^+/K^+ ATPase pump
- K is filtered freely from the glomerulus and reabsorbed from the tubular system
- Some of K is excreted in urine

Potassium shifts



Definition

- **Hyponatremia:** decreased $[\text{Na}^+]$ level in the blood
 - That happen by :
 - Losing Na^+ more than water
 - Gaining water more than Na^+
- **Normal level of $[\text{Na}^+]$** is between (135-145mosm/L)
- **Hyponatremia** is when the $[\text{Na}^+](< 135 \text{mosm/L})$

Causes&classifications of hyponatremia

❑ Hypotonic hyponatremia [Serum osmolality <280 mOsm/kg]

- **According to ECF volume status :**

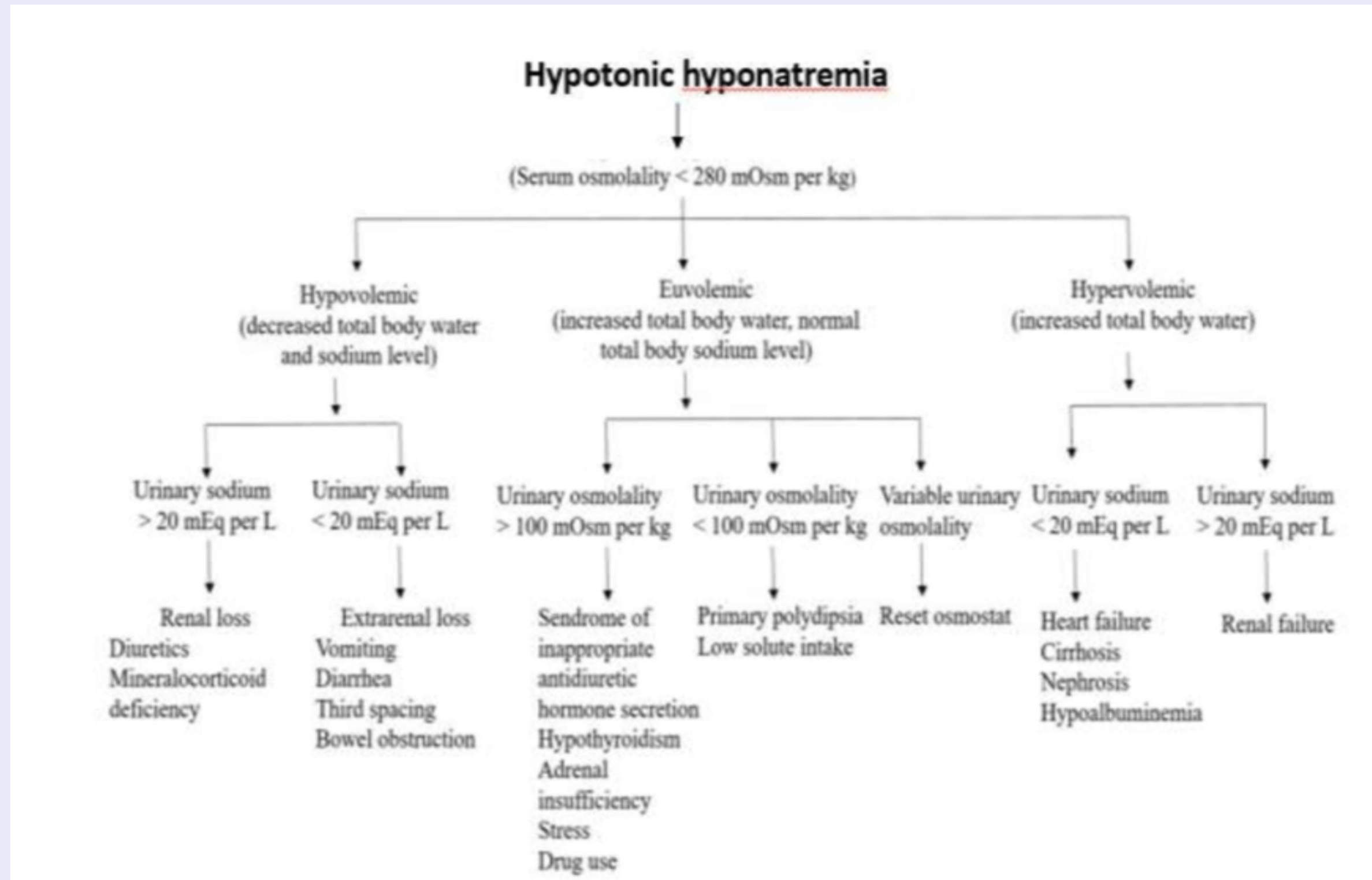
- hypervolemic hypotonic hyponatremia
- Euvolemic hypotonic hyponatremia
- Hypovolemic hypotonic hyponatremia

❑ Isotonic hyponatremia [Serum osmolality (280-295)mOsm/kg]

❑ Hypertonic hyponatremia [Serum osmolality >295 mOsm/kg]

❖ Hypotonic hyponatremia

Serum osmolality < 280 mOsm/kg



A-Hypervolemic hypotonic hyponatremia

❖ Overview:

large increase of Total body water( ECF) & small increase of Na⁺

❖ Causes:

- CHF
- Liver Cirrhosis
- Nephrotic syndrome
- Chronic kidney disease

A-Hypervolemic hypotonic hyponatremia

Mechanism :

- fluid leaks out blood vessels to the Interstitial space and lead to performing edema,ascitis,JVD

-decreased effective arterial blood volume → Stimulate Renine angiotensin aldosterone system:

- 1- ↑ ADH → Increase water retention
- 2- ↑ Aldosterone → Increased Na⁺retain → that lead to increase H₂O retention (water follow Na⁺)

- Low urine sodium due to water retaining states.

B- Euvolemic **hypotonic hyponatremia**

❖ Overview

Elevated total body fluids (but without evidence of expanding of ECF) & **normal level of Na⁺**

❖ Classifications according urine osmolality

1-High concentrated urine

Caused by : SIADH (Small cell lung cancer and carbamazepine)

Mechanism: Inappropriate ADH release lead to water retention & dilution of sodium, while ECF volume remains normal due to sodium excretion by urination (Aldosterone escape mechanism)

2- Diluted urine

Caused by :

1. Adrenal insufficiency
2. Hypothyroidism
3. Primary polydipsia (Drinking too much of water)
4. Potomania (Drinking too much of alcohol)

C- Hypovolemic hypotonic hyponatremia

❖ Overview

Small decrease of total body fluids(↓ ECF) & large decrease of Na⁺

Low urine sodium

❖ Causes

■ 1-Extra renal losses:

A-Diarrhea & Vomiting

B-diaphoresis

C-burns

C- Hypovolemic **hypotonic hyponatremia**

■ 2-Renal causes :

- A. Diuretics (eg:Thiazides)
- B. Meneralocorticoid deficiency (eg:Addison's disease)

■ 3-Cerebral salt wasting:

disruption in the hypothalamic pituitary axis **or** brain injury like(SAH,Brain tumors)

Increased natriuretic peptide ➡ that decreased renal sodium reabsorption & enhanced renal sodium excretion

Diagnosis & Treatment:

Subtype	Diagnosis	Treatment
Hypovolemic Hypotonic Hyponatremia	- Signs of dehydration: hypotension, tachycardia.- - Urine sodium: <20 mmol/L in non-renal loss, >20 mmol/L in renal loss.	- Volume resuscitation: Normal saline 0.9%.- - Treat underlying cause (discontinue diuretics, treat Addison's disease).
Euvolemic Hypotonic Hyponatremia	- No signs of dehydration or overload.- - Urine sodium: >20 mmol/L, high urine osmolality in SIADH.	- Fluid restriction: 1–1.5 L/day.- - Treat underlying cause (e.g., SIADH, adrenal insufficiency, hypothyroidism).
Hypervolemic Hypotonic Hyponatremia	- Signs of fluid overload: edema, ascites, JVD.- - Urine sodium: <20 mmol/L in heart failure, cirrhosis, nephrotic syndrome.	- Fluid and sodium restriction.- - Diuretics (e.g., furosemide).- - Treat underlying condition (e.g., heart failure, cirrhosis).

❖ Isotonic hyponatremia (pseudo hyponatremia)

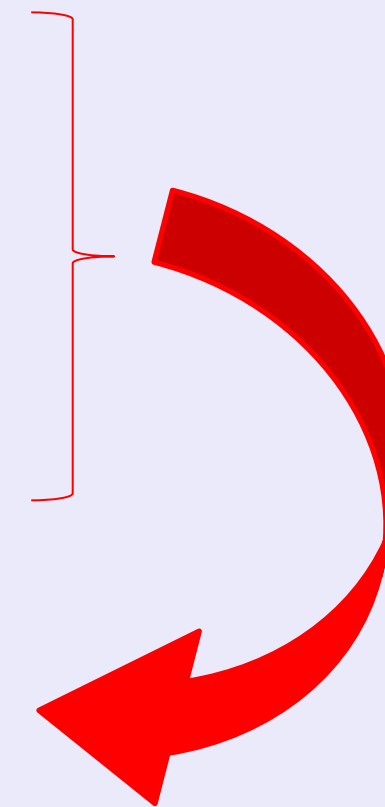
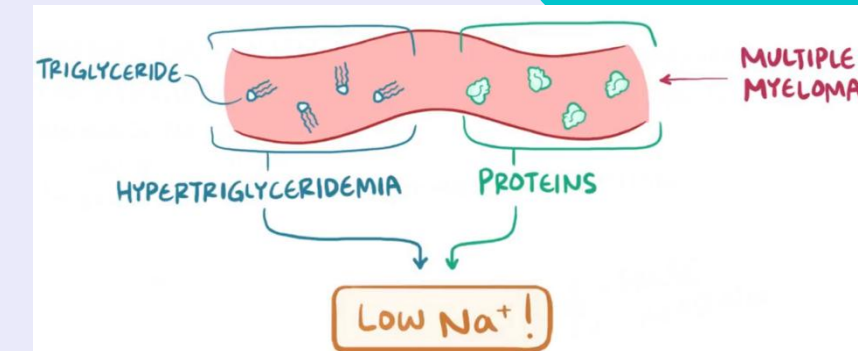
❖ Overview:

- Normal level of Na^+ & Normal H_2O volume
- Increased level of: Triglycerides(Hyper_lipidemia)
Protein (Multiple myeloma)

- These factors lead to false lab results

decreased Na^+ result

Mechanism: Traditional sodium measurement methods : These assume plasma is composed of 93% water and 7% non-water components. When lipids or proteins are excessively elevated, they dilute the plasma water portion, resulting in falsely low sodium readings.





Diagnosis:

1. Serum osmolality: normal osmolality (280-295mOsm/kg)
2. plasma lipid & protein levels: elevated triglycerides & immunoglobulin proteins

Treatment:

1. **Hyperlipidemia**: lifestyle modification or lipid lowering medications (eg: statins)
 2. **Hyperproteinemia** : Treat the underlying disease (eg: chemotherapy for multiple myeloma)
 3. **Avoid unnecessary sodium correction** : because plasma water sodium concentration is normal
- 



❖ Hypertonic hyponatremia

Caused by the presence of osmotic substances that increase plasma osmolality then cause an osmotic shift of water out of cells. The dilution of plasma sodium leads to hyponatremia

These substances include:

- Glucose-Hyperglycemia
 - Mannitol, sorbitol, glycerol, maltose
 - Radiocontrast agents
- 

Hypertonic hyponatremia

❖ Diagnosis:

- Elevated serum osmolality
- Elevated serum glucose

❖ Treatment

- 1-Treat the underlying causes :
 - **Hyperglycemia:**Use insulin therapy
 - **Mannitol or contrast agent exposure:**stop or adjust the use of the osmotic agent
- 2-sodium correction:
 - Gradually to avoid osmotic demyelination syndrome
- 3- monitoring for neurological symptoms

Clinical features

❖ Symptoms usually begin when the Na^+ level falls to $<120\text{mEq/L}$

❖ **Symptoms:-**

1- **Neurological symptoms:** caused by osmotic water shifts which lead to increased ICF volume —————> Brain edema

Headache, muscle twitching, hyperactive deep tendon reflex

Increased ICP, Coma, Seizure

2- **GI symptoms:** vomiting, watery diarrhea

3- **Cardio Vascular symptoms:** HTN due to increased ICP

4- **Increased salivation & lacrimation**

5 **Oliguria progressing to anuria** (maybe reversible)

Hypernatremia

- **General Features of hypernatremia**

- Defined as $\text{Na} > 145 \text{ mEq/L}$
- Hypernatremia is much less common than hyponatremia and nearly always indicates a water deficit.
- Hypernatremia is always associated with high osmolarity which stimulate thirst center in the brain making the patient thirsty so hypernatremia doesn't occur unless the patient experiences one of the following:
 1. The thirst center doesn't stimulate water intake or there is limited access to water
 2. The patient lost a lot of fluids (Febrile illness, burns, diarrhea, diuretics)

Hypernatremia

- **Clinical features**

- Symptoms of hypernatremia are non-specific
 1. Irritability, confusion, stupor, and coma
 2. Convulsions occur in severe hypernatremia.

- **Causes of hypernatremia**

- Most important ones are:
 1. Central diabetes insipidus
 2. Nephrogenic diabetes insipidus
 3. Impaired thirst in elderly

? Box 9.16 Causes of hypernatraemia

Antidiuretic hormone (ADH) deficiency

- Pituitary diabetes insipidus (see p. 641)

Iatrogenic

- Administration of hypertonic sodium solutions
- Administration of drugs with a high sodium content (e.g. piperacillin)
- Use of 8.4% sodium bicarbonate after cardiac arrest

Insensitivity to ADH (nephrogenic diabetes insipidus)

- Lithium
- Tetracyclines

- Amphotericin B
- Acute tubular necrosis
- Osmotic diuresis
- Total parenteral nutrition
- Hyperosmolar hyperglycaemic state (see p. 725)

Plus

- Deficient water intake: impaired thirst or consciousness
- Excessive water loss through skin or lungs

Hypernatremia

- Diabetes insipidus means loss of ADH effects causing:
 1. Polyuria
 2. Polydipsia
- Central Diabetes insipidus:
 1. There is little or Low ADH
 2. It's usually due to trauma, Surgery or tumor in the brain but there are many other causes
- Nephrogenic Diabetes insipidus:
 1. ADH is normal but the kidneys don't response to ADH
 2. Resistance to ADH
 3. Causes:
 - Electrolyte disturbances: Hypercalcemia, Hypokalemia (severe: < 3.0 meq/L)
 - Drugs: Lithium, Amphotericin B, and demeclocycline
 - Hereditary: ADH Receptor mutation

Hypernatremia

- **Diagnosis**

- Serum Osmolarity: Hypernatremia or normal sodium
- Urine Osmolarity is low (50-200 mOsm/L)
- Diluted urine: Specific gravity < 1.006

- **Water deprivation test**

1. After 8 hours of no fluid, urine should be concentrated
2. No increase in urine osmolarity (Urine remains diluted) which excludes primary polydipsia

- **Desmopressin test**

1. Desmopressin is ADH analogue which stimulates ADH V2 receptor triggering water reabsorption and urine concentration
2. Urine osmolarity increase significantly (Concentrated urine) : Central DI
3. Diluted urine : Nephrogenic DI

Hypernatremia

- **Treatment**

- Water (ideally PO)
- IV Fluids (D5W)

- **Caution: cerebral edema**

1. Occurs with overly-rapid correct of hypernatremia
 2. Maximum correction: 12 mEq/L/day
- From high to low, your brain will blow (Cerebral edema)

- **Central diabetes insipidus treatment: Desmopressin (DDAVP)**

- Desmopressin (DDAVP) doesn't have any vasoconstrictive effects

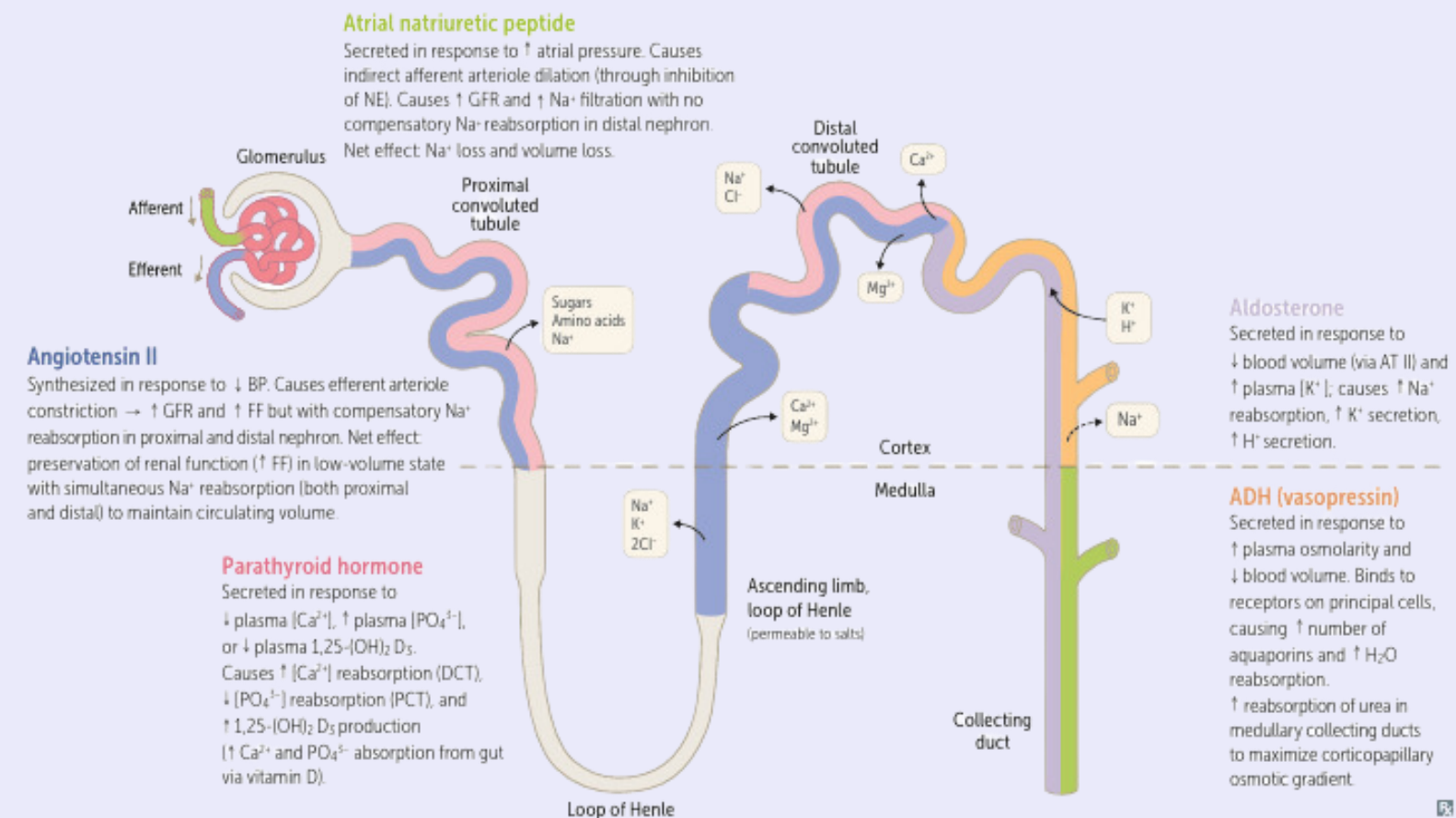
Hypernatremia

- **Nephrogenic diabetes insipidus treatment:**

- Low sodium diet
- **Thiazide diuretics: HCTZ**
 1. Inhibit NaCl cotransporter
 2. Loss of a high amount of NaCl
 3. Activation of RAAS
 4. Reabsorption of Na and water
 5. Paradoxical antidiuretic effect

- **NSAIDs: Indomethacin**
- prostaglandins antagonize the effect of ADH
- NSAIDS inhibit renal synthesis of prostaglandins which improves ADH activity

Hormones acting on kidney



Hypernatremia

- Central vs Nephrogenic diabetes insipidus

	Central DI	Nephrogenic DI
Definition	Low ADH	ADH resistance
ADH Level	LOW	NORMAL
Water deprivation test	Urine remains diluted	Urine remains diluted
Desmopressin test	Concentrated urine	Diluted urine
Treatment	Desmopressin (DDAVP)	HCTZ Indomethacin Low sodium diet

Hypokalemia

Pathophysiology

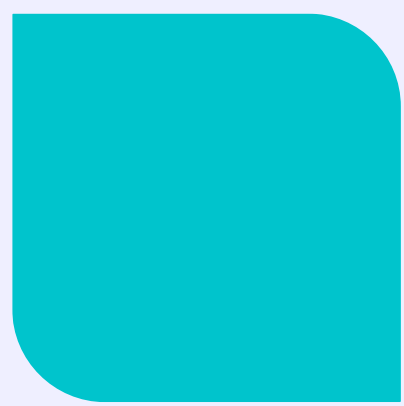
Causes

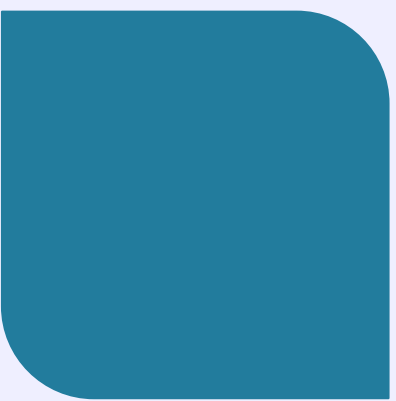
Clinical Features

Management

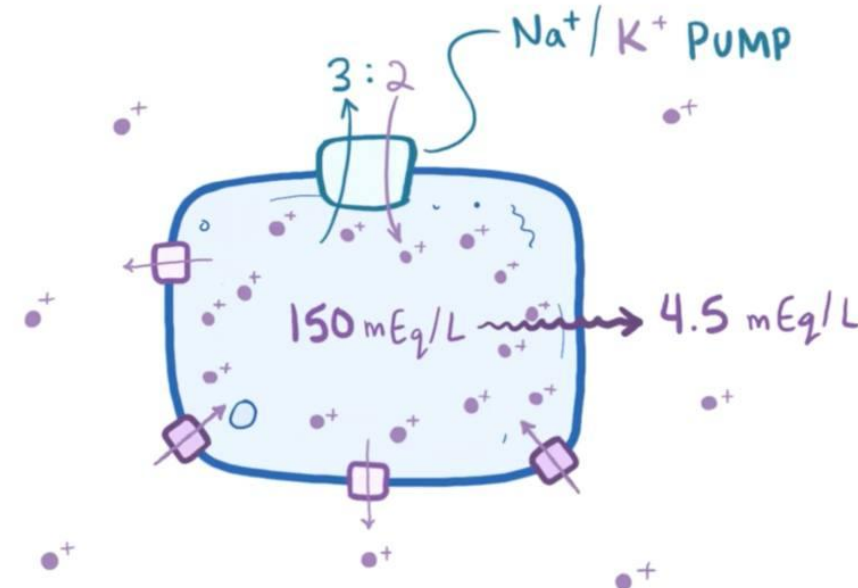


Introduction to Potassium and hypokalemia



- Normal serum potassium levels: **3.5–5.0 mEq/L**.
 - Potassium distribution: 98% intracellular, 2% extracellular.
 - Importance of potassium: nerve conduction, muscle contraction, cardiac rhythm.
 - Hypokalemia: serum potassium **<3.5 mEq/L**.
 - Effects of pH:
 - **Alkalosis**: Potassium shifts into cells → **Hypokalemia**.
 - **Acidosis**: Potassium shifts out of cells → **Hyperkalemia**.
- 
- 

INTERNAL K⁺ BALANCE

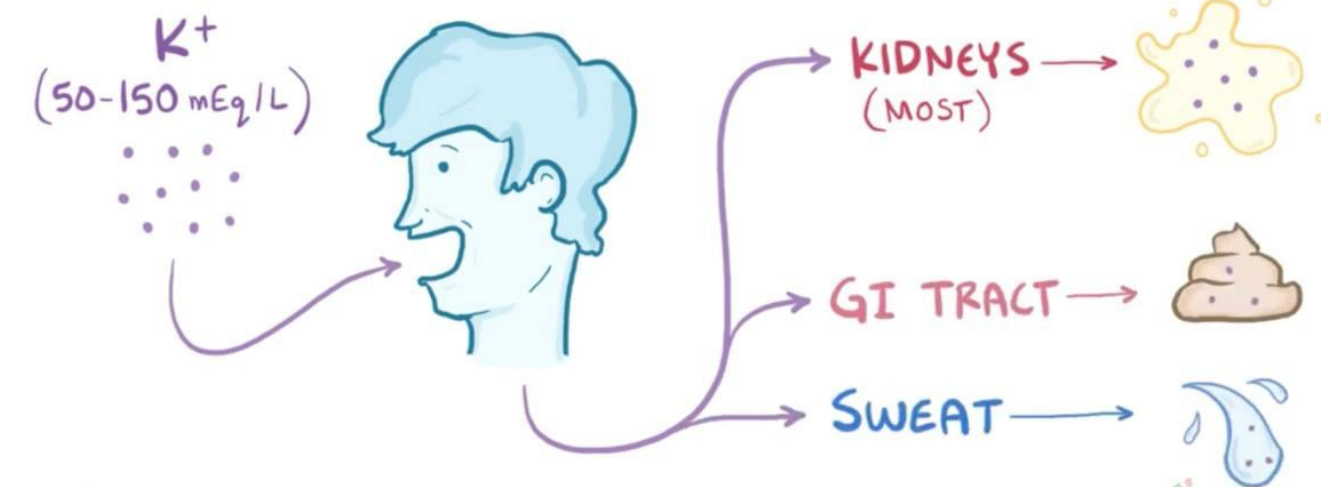


ELECTROCHEMICAL
GRADIENT
↓
SETS **RESTING**
MEMBRANE POTENTIAL
↓
NEEDED for **CONTRACTION**
of **SMOOTH, CARDIAC**
& **SKELETAL MUSCLE**

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2023 Edition

EXTERNAL K⁺ BALANCE

* EXCRETE MOST of WHAT'S TAKEN IN



Causes of hypokalemia

1. Gastrointestinal Losses:

- Diarrhea (causes both hypokalemia and non-AG metabolic acidosis)
- Vomiting, nasogastric suctioning.
- Laxatives, enemas, intestinal fistulas.

2. Renal Losses:

- Diuretics (loop and Thiazide), renal tubular or parenchymal disease.
- Hyperaldosteronism (primary and secondary).
- **Magnesium deficiency.**
- Bartter syndrome: Defect in salt reabsorption → Increased renin and aldosterone.

Causes of hypokalemia

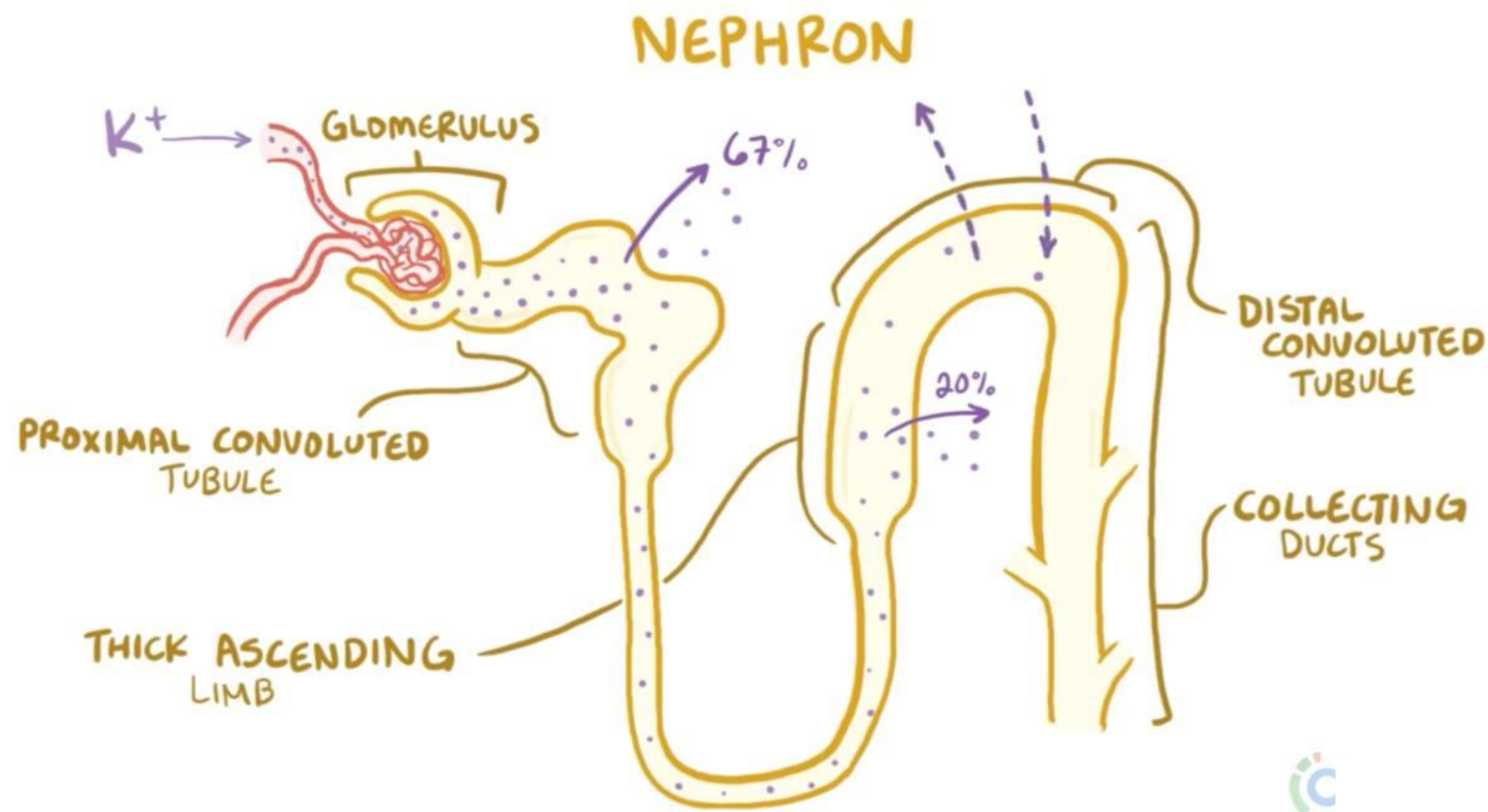
3. Other Causes:

- Poor dietary intake.
- Insulin administration.
- β 2-agonists (e.g., epinephrine).
- Antifungal like amphotericin B.
- Profuse sweating.

**Quick Tip: Presence or absence
of hypertension helps
differentiate causes.**

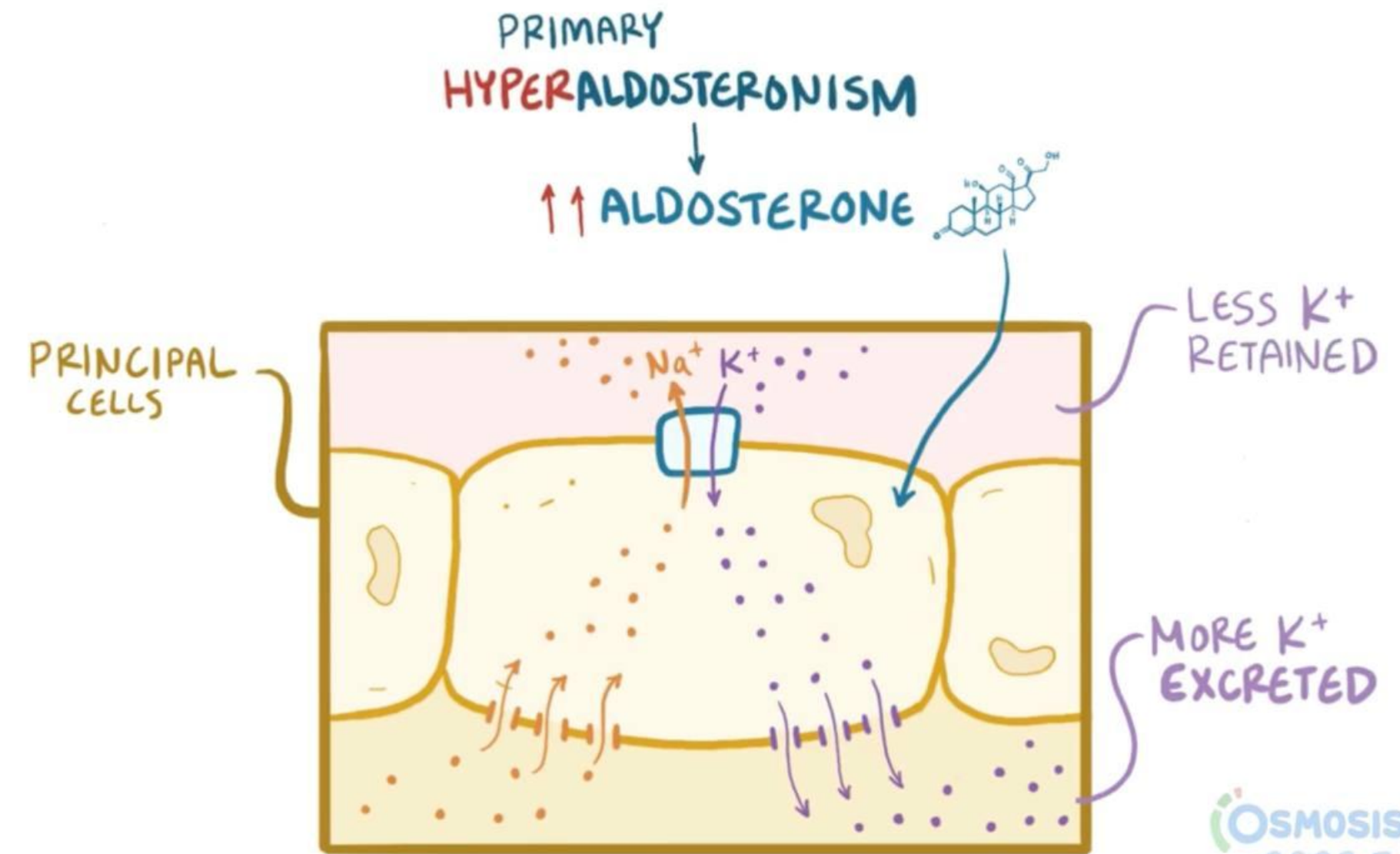
- **Hypertension: Likely
aldosterone-related.**

Normotension: GI or renal loss.

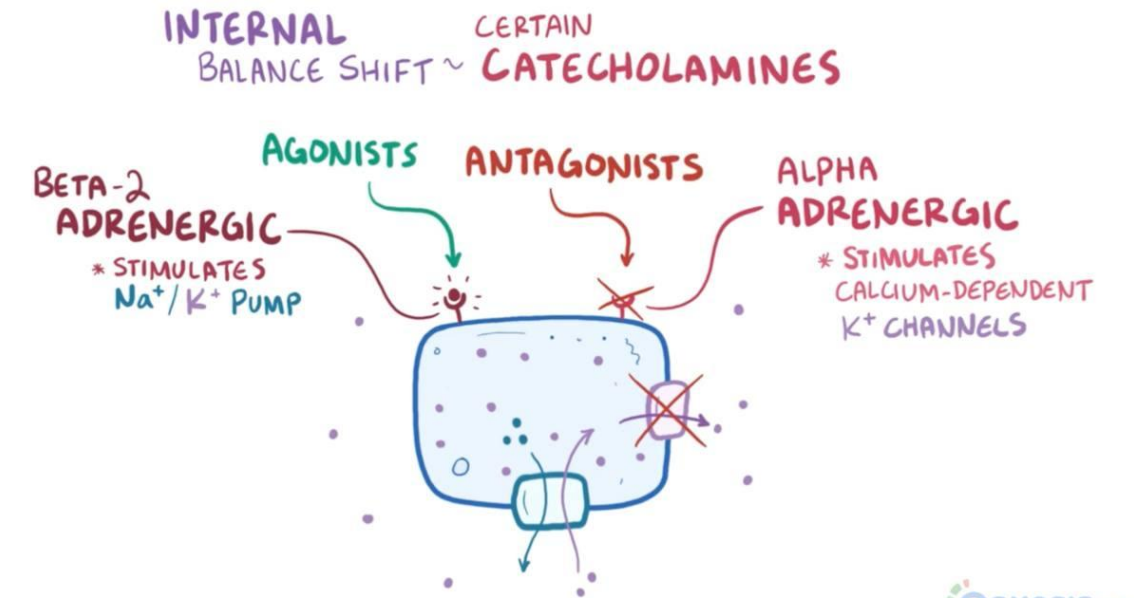
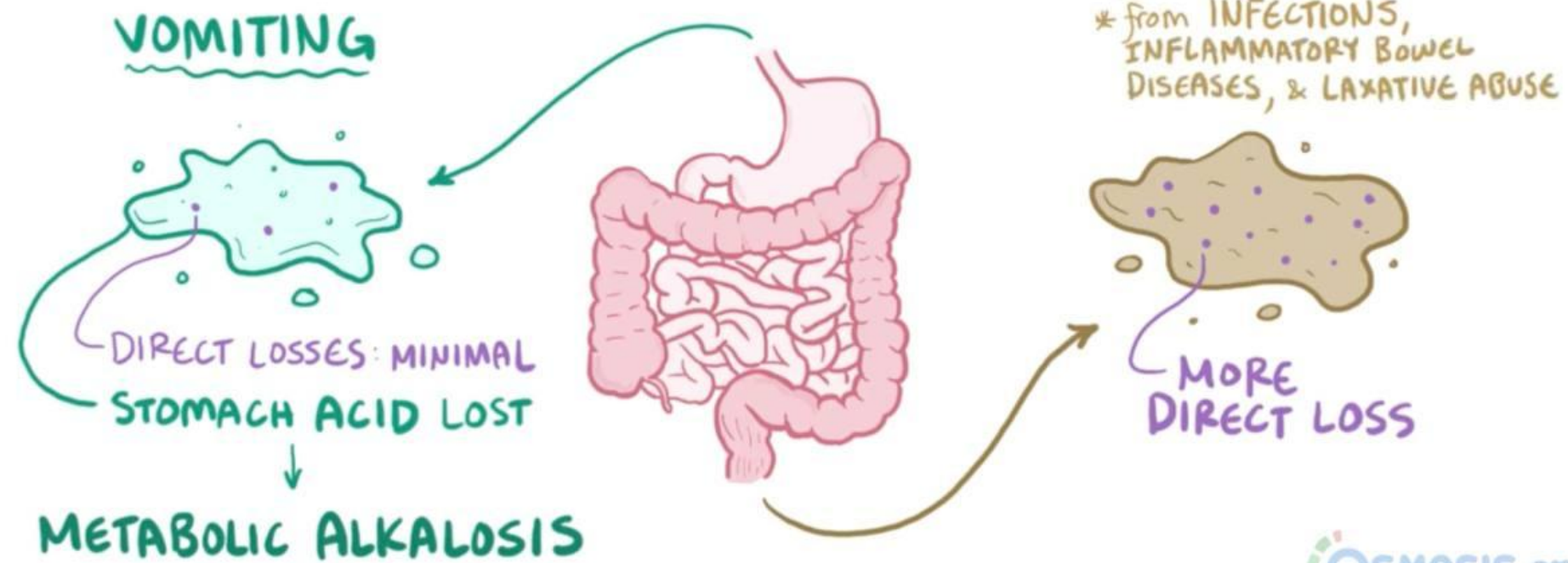


Key sites of absorption and secretion of potassium in nephron

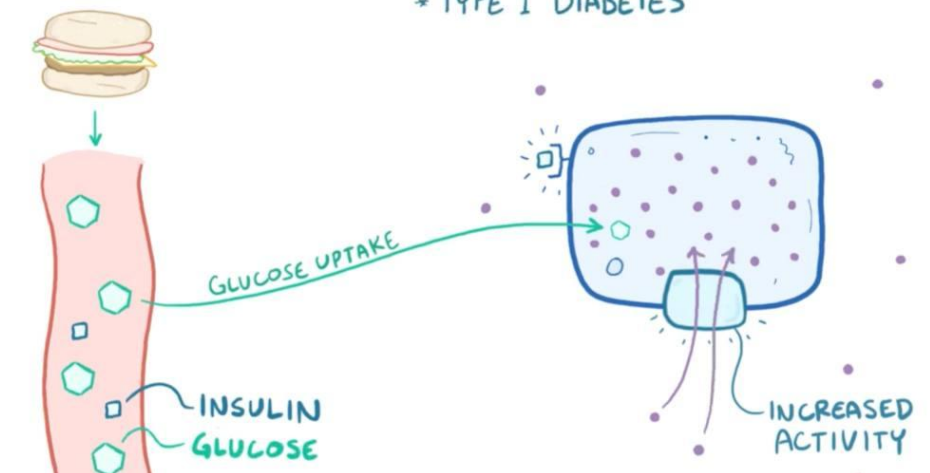
Aldosterone's effect on the secretion of potassium in the distal convoluted tubules



EXTERNAL
BALANCE SHIFT ~ LOSS of GASTROINTESTINAL
SECRETIONS



INTERNAL
BALANCE SHIFT ~ EXCESS INSULIN
* TYPE I DIABETES



Clinical presentation

1. Cardiac Symptoms:

- Arrhythmias (most dangerous).
- ECG changes: Flattened/inverted T waves, U waves if severe.

2. Neuromuscular Symptoms:

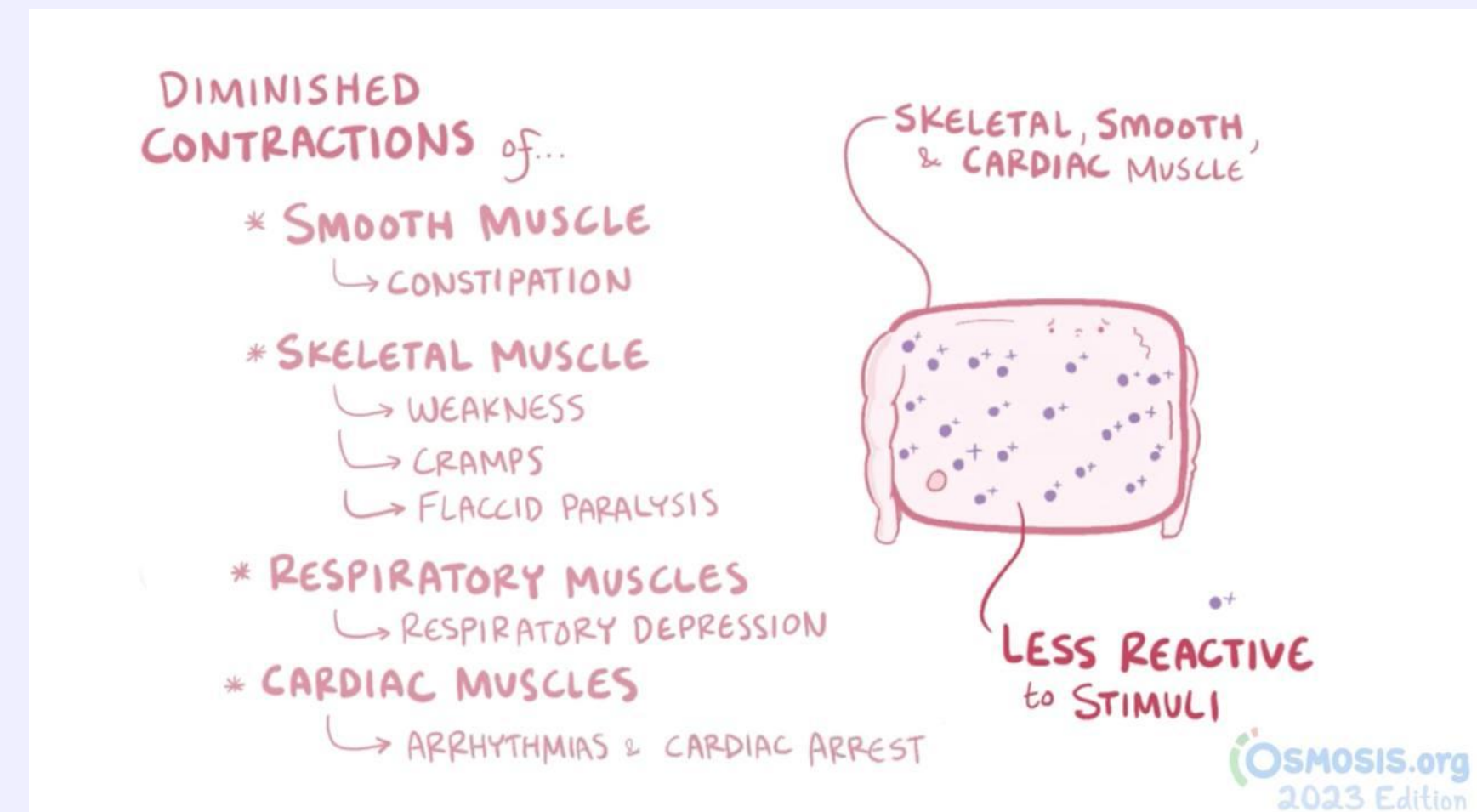
- Weakness, fatigue, muscle cramps.
- Paralysis, decreased deep tendon reflexes.

3. Gastrointestinal Symptoms:

- Paralytic ileus.
- Nausea/vomiting.

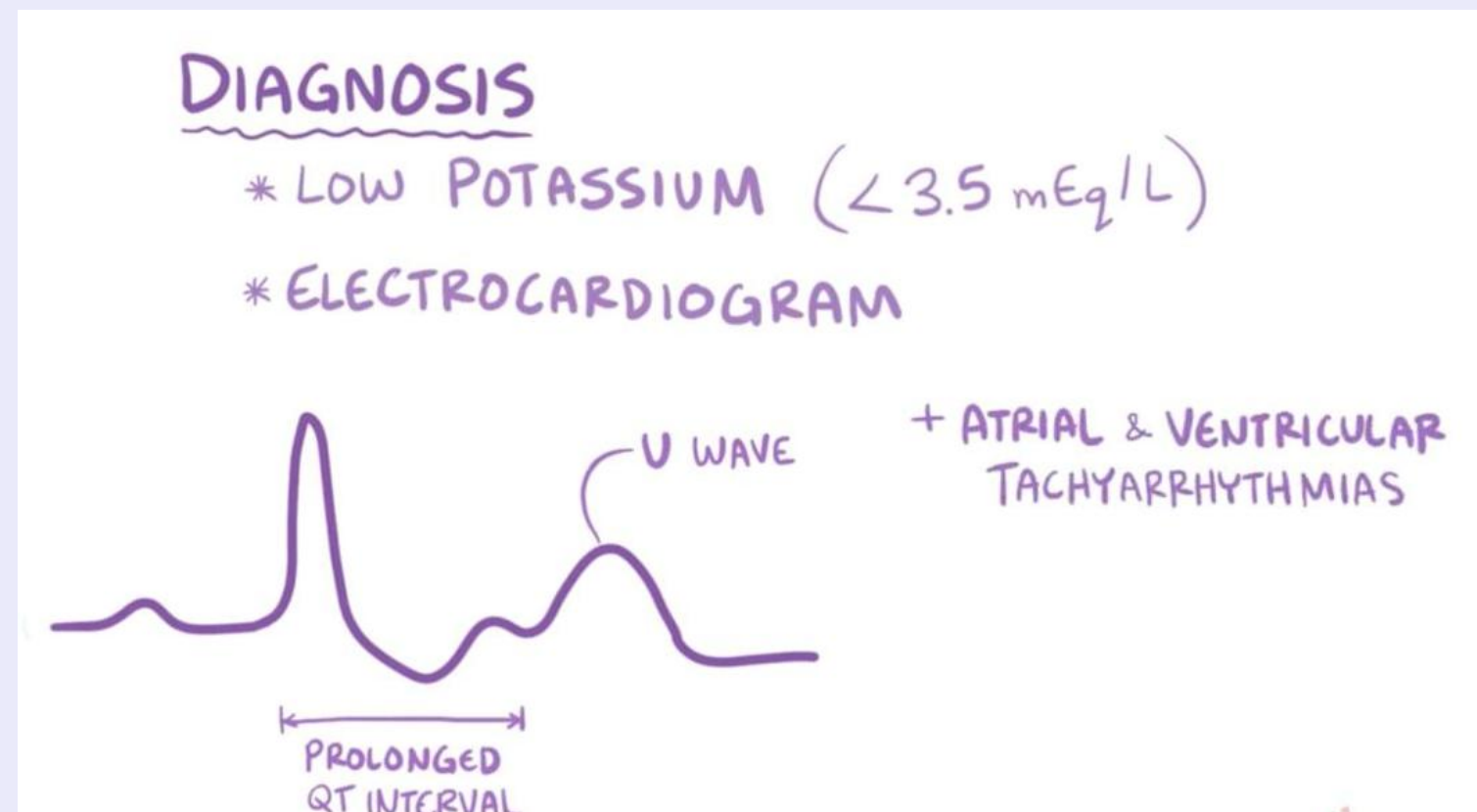
4. Renal Symptoms:

- Polyuria, polydipsia.



Diagnostic evaluation

- Interpretation of Urine Potassium:
 - **GI losses:** Urine K⁺ <20 mEq/L.
 - **Renal losses:** Urine K⁺ >20 mEq/L.
- Other diagnostic tools:
 - Blood tests (serum potassium, magnesium).
 - **ECG** for cardiac changes.



Management

1. Identify and treat the Underlying Cause:

- Discontinue offending medications.

2. Potassium Replacement:

- **Oral KCl:**

- Preferred for mild-moderate hypokalemia.
- Rule: 10 mEq KCl raises serum K⁺ by ~0.1 mEq/L.

- **IV KCl:**

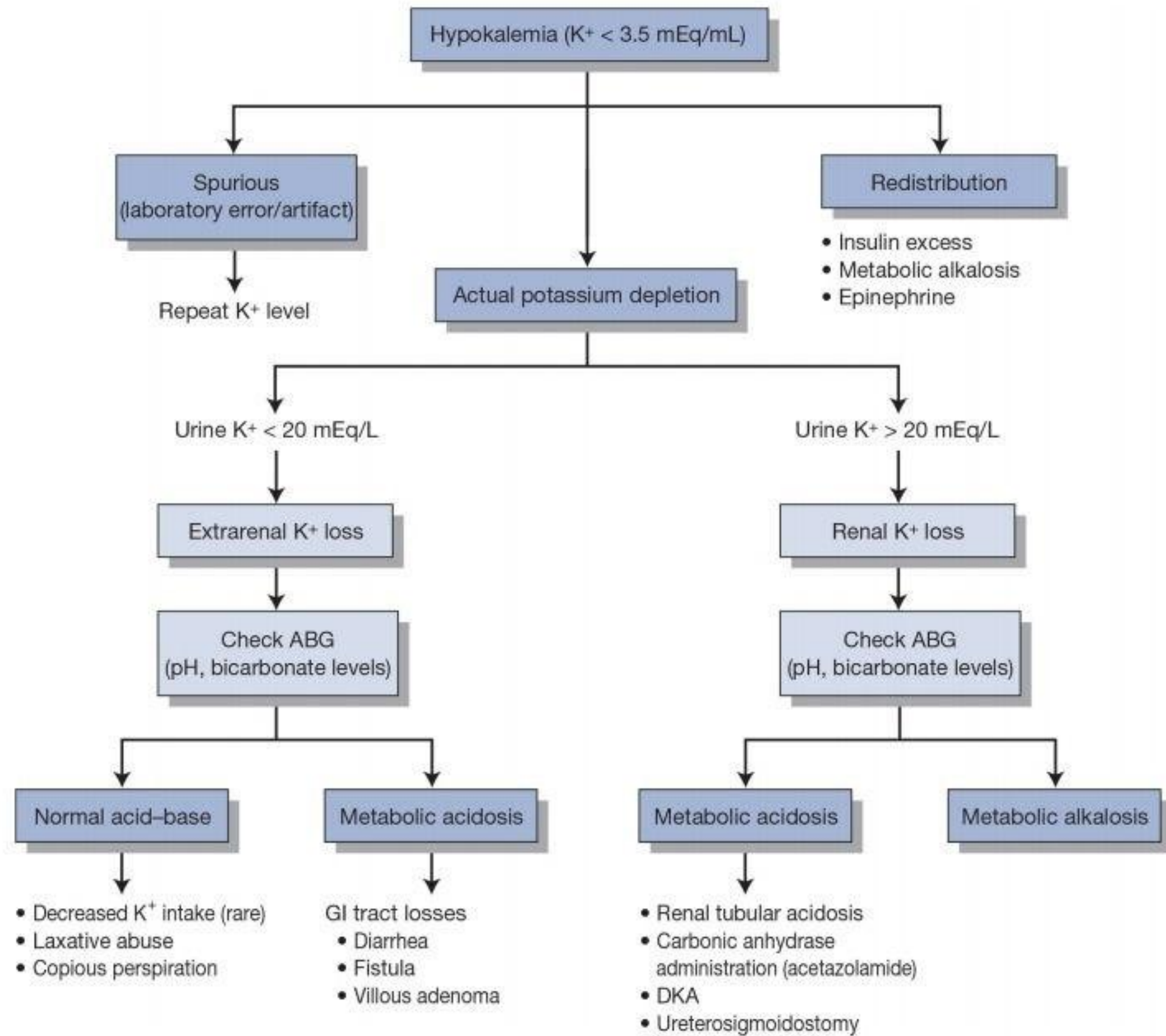
- Indicated for severe hypokalemia (<2.5 mEq/L) or arrhythmias.
- Administer **slowly**:
 - Peripheral line: Maximum infusion rate is 10 mEq/hr.
 - Central line: Maximum infusion rate is 20 mEq/hr.
- May add 1% lidocaine to bag to decrease pain as potassium burns!

3. Monitor and Adjust:

- Recheck serum potassium.
- **Correct hypomagnesemia if present (required for proper potassium correction).**

Complications & special considerations

- Complications:
 - Severe arrhythmias → cardiac arrest.
- **Exacerbation of digoxin toxicity.**
- IV administration risks:
 - Phlebitis (inflammation of veins).
 - Pain during infusion (reduce with lidocaine).



Hyperkalemia

Definition

- Hyperkalemia : Elevated K⁺ level in the blood
- Normal level of [K⁺] is (3.5 - 5mEq/L)
- Hyperkalemia is when [K⁺] > 5mEq/L

Etiology (Causes) of Hyperkalemia

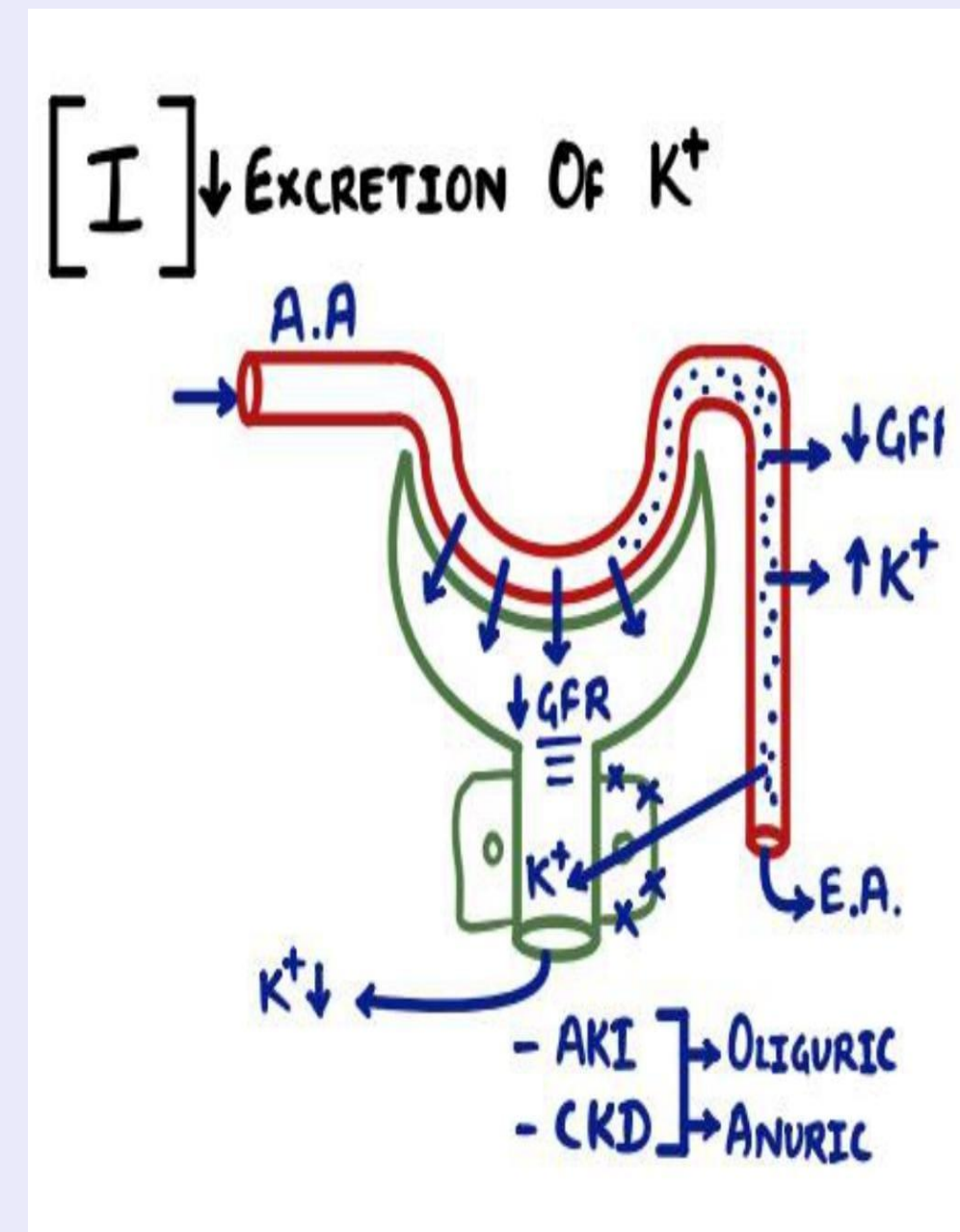
A.Reduced excretion of K⁺

II. K⁺ is primarily excreted into the kidney tubule in the DCT but it depends on a very specific type of hormone (aldosterone).

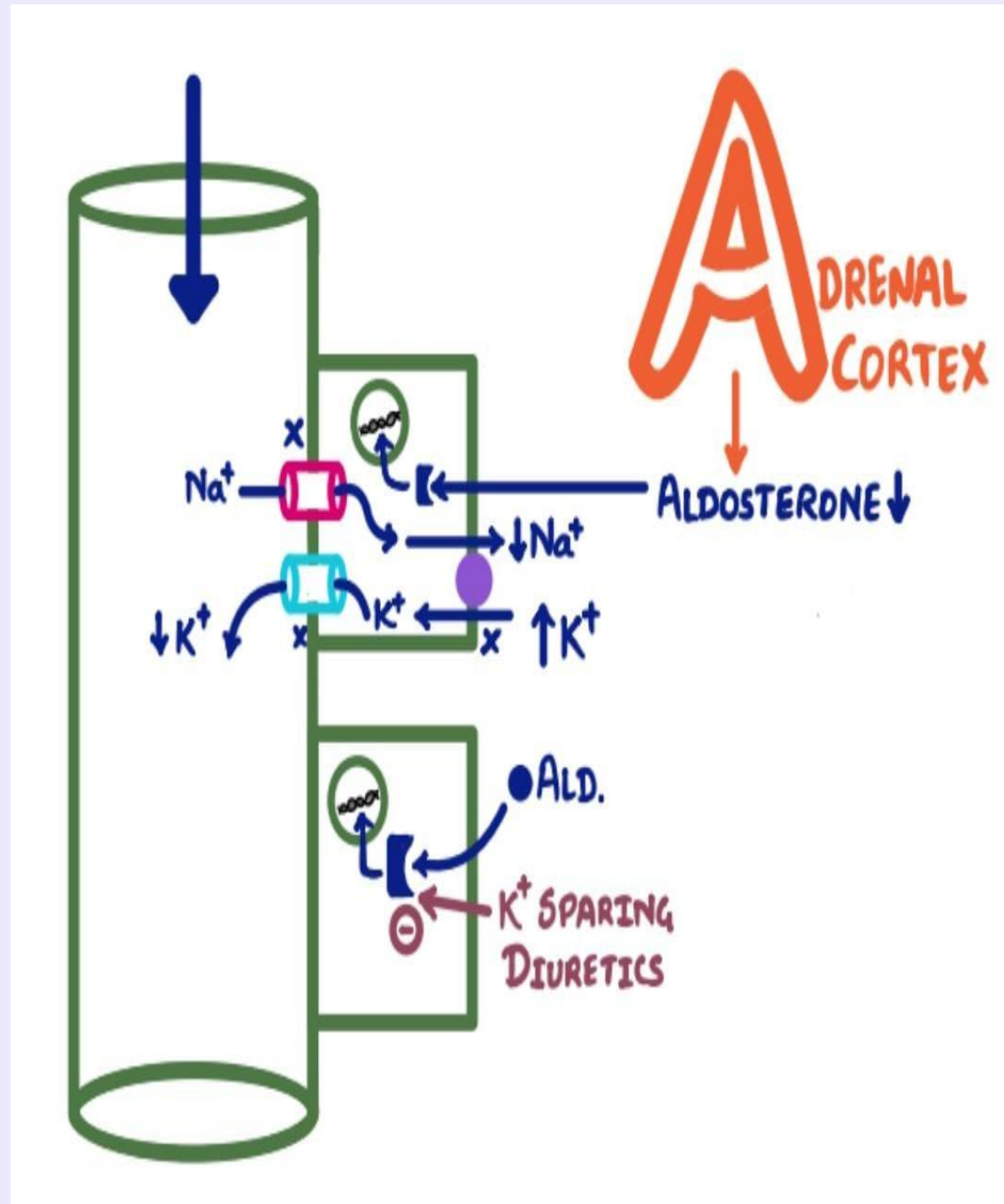
III.AKI or CKD : Cells of DCT are damaged , Their ability to respond to hormone and excrete the K⁺ is decreased.

IV.AKI or CKD: there are reduced GFR, less K⁺ filtered, less K⁺ excreted, K⁺ stays in the bloodstream, increased K⁺ level in the blood.

V.Renin Angiotensin Aldosterone System



Etiology (Causes) of Hyperkalemia



A. Reduced excretion of K⁺:

Aldosterone:

Person with decreased aldosterone activity :

-These processes got affected , Less Na⁺ reabsorbed and Less K⁺ excreted - End results , Increased K⁺ level in blood , hyperkalemia .

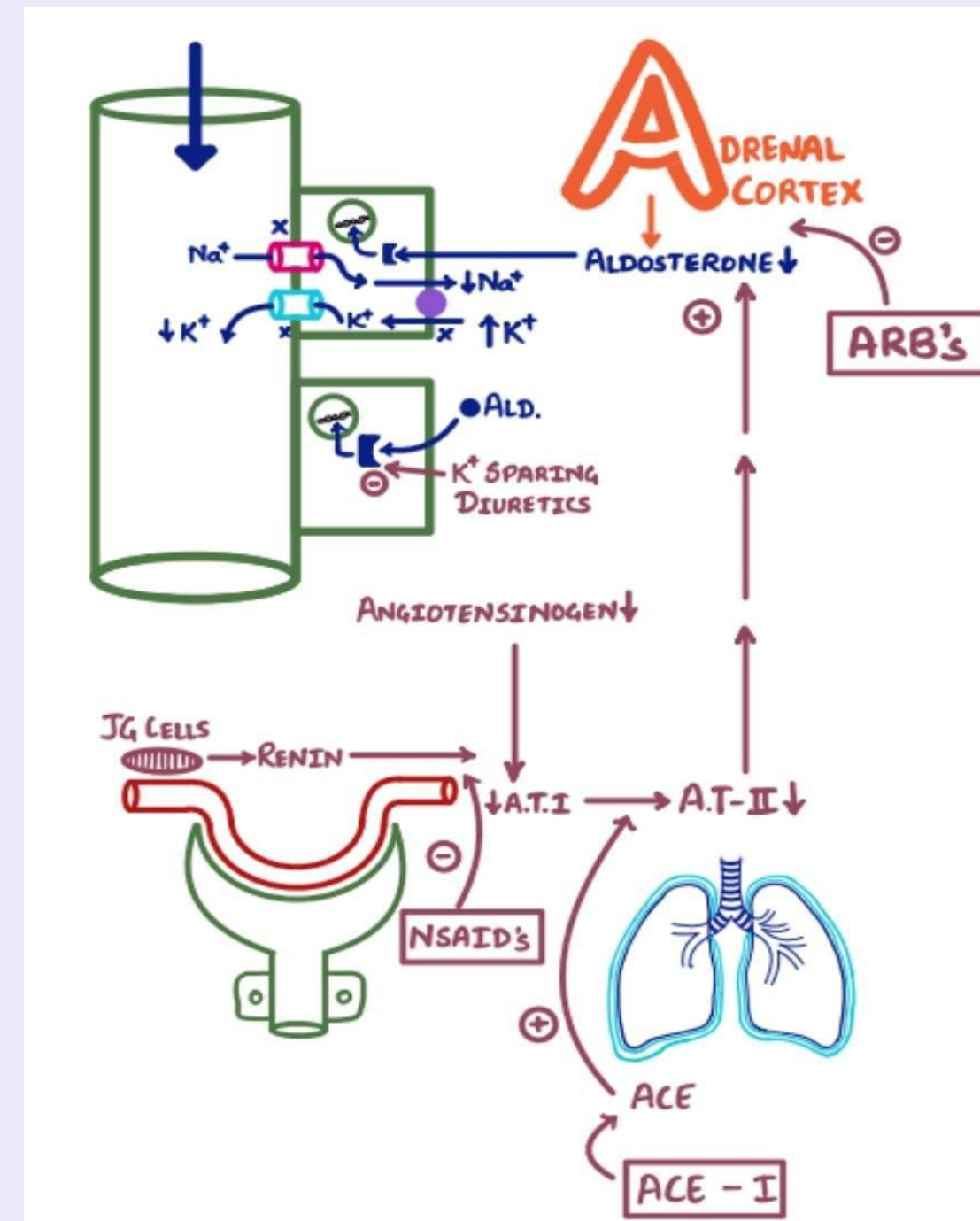
Renal failure:

(that caused by DM , HTN , polycystic kidney disease , SLE & amyloidosis) can contribute to hyperkalemia

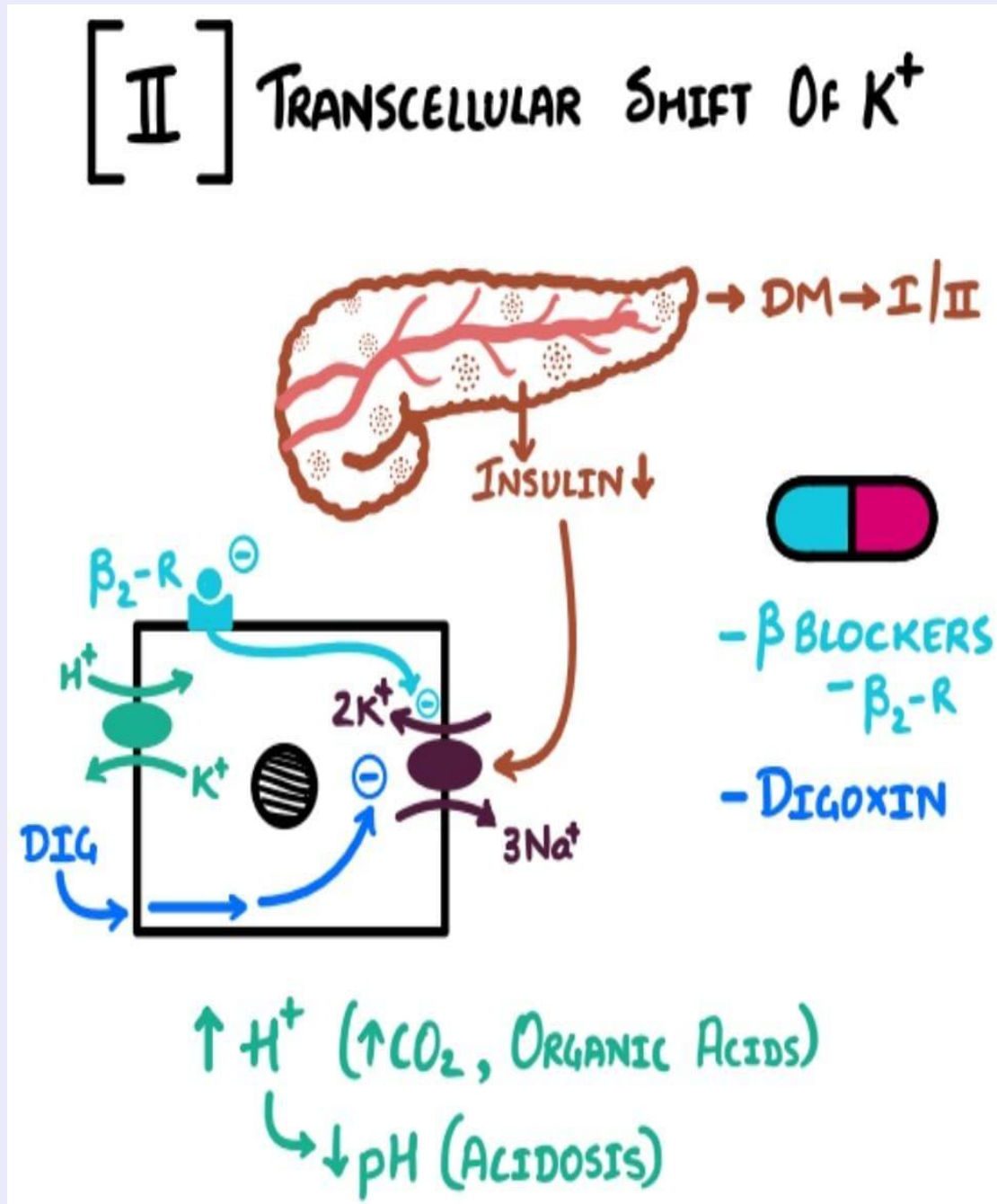
Etiology (Causes) of Hyperkalemia

A. Reduced excretion of K⁺:

- (3) Drugs affecting K⁺ excretion:
 - 1.NSAIDs.
 - 2.ACE-I (Angiotensin Converting Enzyme Inhibitor).
 - 3.ARB (Angiotensin II receptor blocker).
 - 4.Potassium-sparing diuretics. (spironolactone)



Etiology (Causes) of Hyperkalemia



B. Transcellular shift of K^+

(1) Decreased or no insulin (DM type 1):

Decreased insulin , inhibit Na^+ / K^+ ATPase pump , doesn't pump K^+ into the cell that keep it outside the cell in the blood or extracellular fluid , causes hyperkalemia.

Etiology (Causes) of Hyperkalemia

Transcellular shift of K⁺

(2) Drugs :

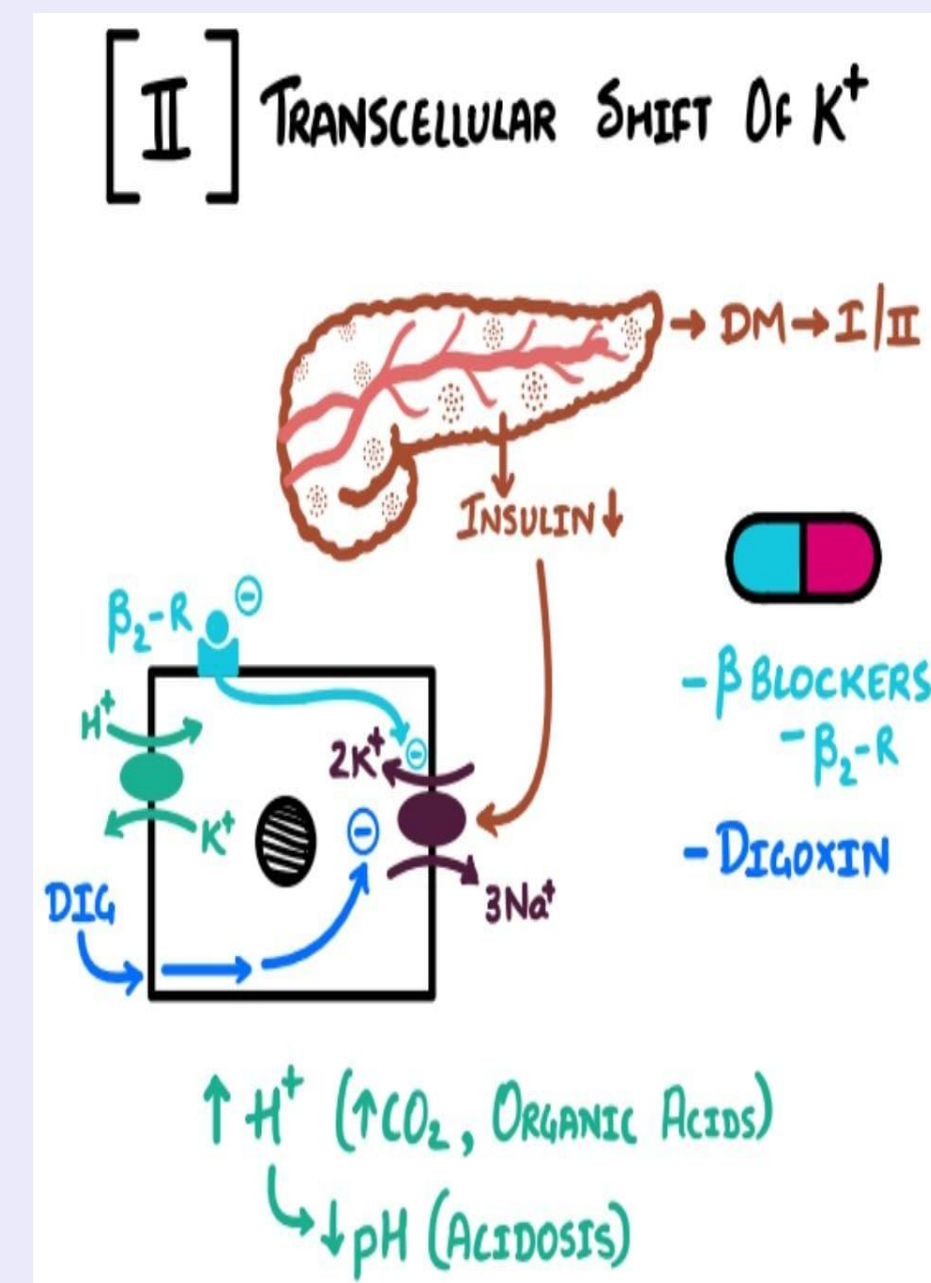
- B-blockers
- Digoxin (cardiac glycoside)

(2) Acidosis (respiratory or metabolic acidosis)

- In acidic environments [H⁺] increased .
- Push K⁺ out in exchange for that H⁺ , The more H⁺ we push in, the more K⁺ we push out , hyperkalemia.

(4) Tumor lysis syndrome:

Damaged cells from the chemotherapy ,
Releasing K⁺, PO₄³⁻ (phosphate), uric acid.

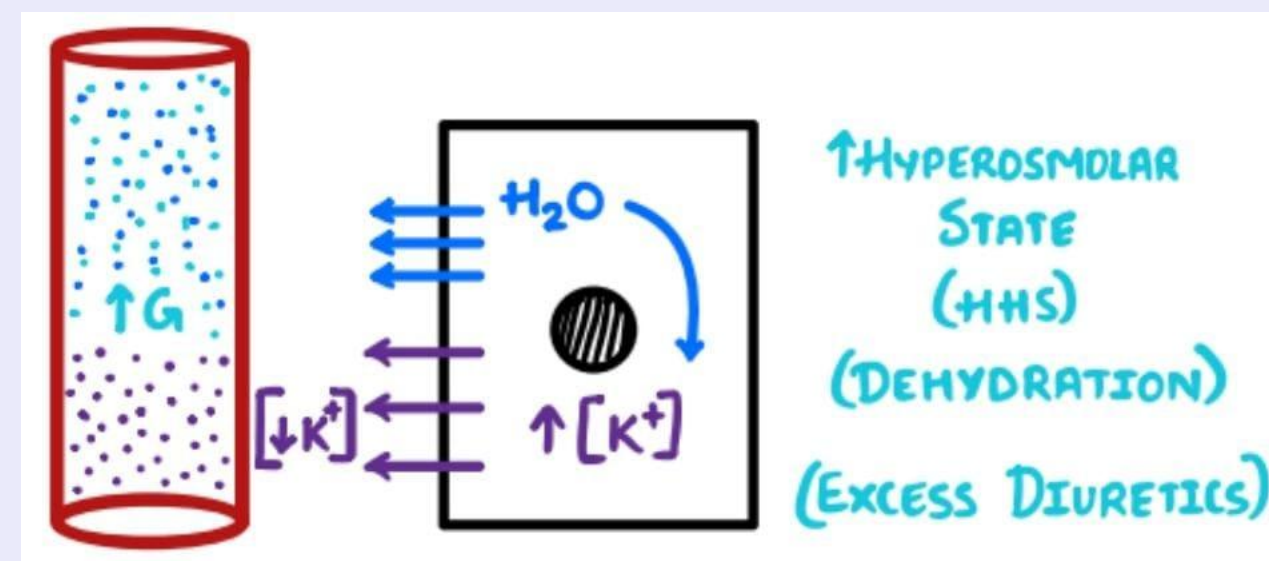


Etiology (Causes) of Hyperkalemia

(5) Hyperosmolar state :

In hyperosmolarity state, water flows out of cells causing K^+ concentration to rise inside the cells.

- K^+ ions move out of cells into interstitial fluid then blood down concentration gradient, resulting on Hyperkalemia.



(6) Damage to the red blood cell :

Cells store 98% of body K^+ ions, cell lysis will cause it to release to interstitial fluid and blood causing Hyperkalemia.



(7) Rhabdomyolysis

(C) Increased K^+ intake (food or iatrogenic error during IV infusion)

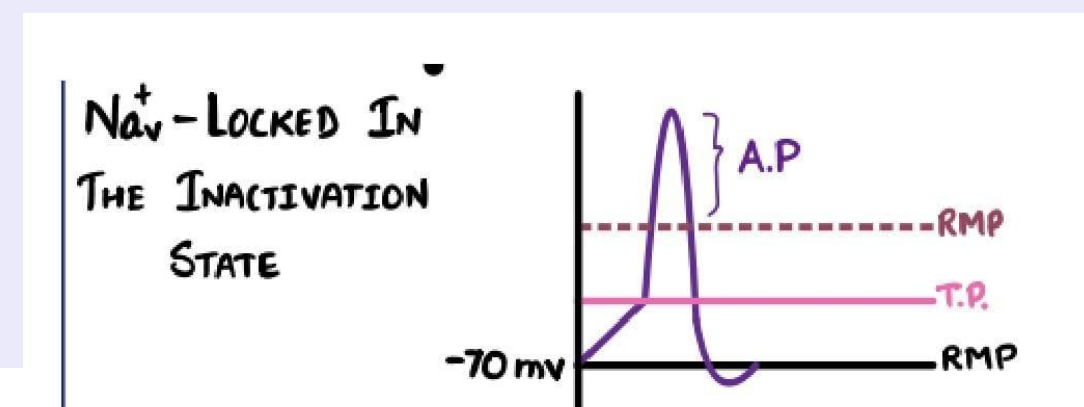
What happens in hyperkalemia ?

K⁺ is high concentration outside the cell (Normally we have K⁺ inside the cell and low K⁺ outside the cell Result)

- Less K⁺ leaves the cell (due to changes in concentration gradient)
- RMP less negative

New more positive RMP

- Because of that when we give the stimulation , It can't go back to rest phase
- Na⁺ voltage gate locked in the inactivation state
- Can't stimulate the cell because the cell stuck in inactivation state
- Net result The excitable cell activity declines



Symptoms of hyperkalemia

1) Skeletal muscles and neurons:

Skeletal muscles Weakness **and** Decreased AP of neurons.
Decreased deep tendon reflexes (DTR).

2) GIT

- Develop constipation
- Sometimes maybe even cause ileus
- nausea and vomiting

3) cardiac muscle:

Decreased contractility of the heart

4) Skeletal muscles for breathing

Affects diaphragm and intercostal muscles

5) AKI/CKD.

Mnemonic

• MURDER

- **M**uscle weakness
- **U**rine: oliguria/anuria
- **R**espiratory distress
- **D**ecreased cardiac contractility
- **E**KG changes
- **R**eflexes

DIAGNOSIS OF HYPERKALEMIA

If potassium level in the blood is > 5 mEq/L :

- 1.Rule out pseudo-hyperkalemia

- 2. Make a true diagnosis of hyperkalemia :
 - (1) History of AKI or CKD.
 - (2) Hypoaldosteronism .
 - (3) Medications: Discontinue and see what happens to the K⁺ level.
 - (4) Hyperosmolar cause due to diabetes
 - (5) Acidosis
 - (6) Transcellular shifting
 - (7) Rhabdomyolysis
 - (8) Tumor lysis syndrome
 - (9) Hemolysis

- 3. Look for EKG changes

PSEUDOHYPERKALEMIA

- Definition: artificially elevated plasma K⁺ concentration due to K⁺ movement out of cells immediately before or after venipuncture. Contributing factors include prolonged use of a tourniquet with or without repeated fist clenching.

- Pseudohyperkalemia can be due to :

- a. Hemolyzed sample Due to trauma or prolonged tourniquet use

- b. Thrombocytosis (platelet count >500,000/ μ L): Potassium is released from activated platelets during clot formation in serum

- c. Leukocytosis Occurs when **fragile white blood cells** (e.g., in leukemia) release potassium in the serum sample due to cell lysis after the blood is drawn.

- Seen in conditions like **chronic lymphocytic leukemia (CLL)** or other leukemias with fragile leukocytes.

- Mechanism: Potassium leaks out of cells in vitro (in the blood sample)

- Serum potassium is falsely high, but plasma potassium is normal.

REVERSE PSEUDOHYPERKALEMIA

- Definition: A falsely elevated potassium in plasma while there is normal potassium levels in serum. due to **hemolysis** or **sample handling issues**
- Causes:
Hemolysis of RBCs during sample collecting (improper handling or excessive force)
- Plasma potassium is falsely high, but serum potassium is or normal .

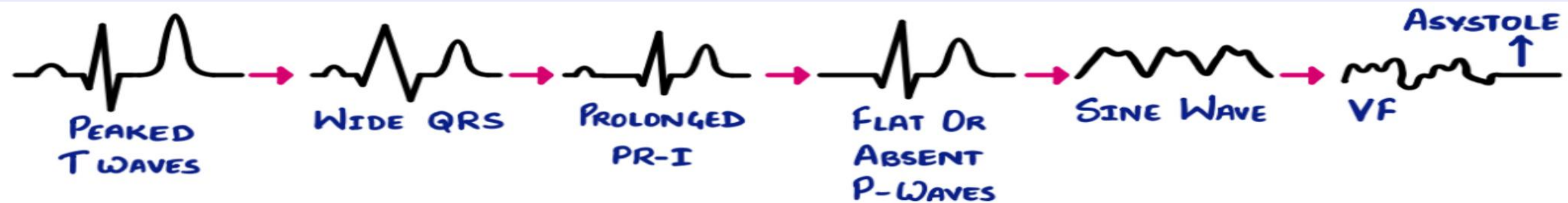
Condition	Serum K	Plasma K	ECG Change
Hyperkalemia	High	High	Usually yes
Pseudohyperkalemia	Falsely high	Normal	No
Reverse pseudohyperkalemia	Normal	Falsely high	No

Abbreviations: ECG, electrocardiogram; K, potassium.

Investigations :

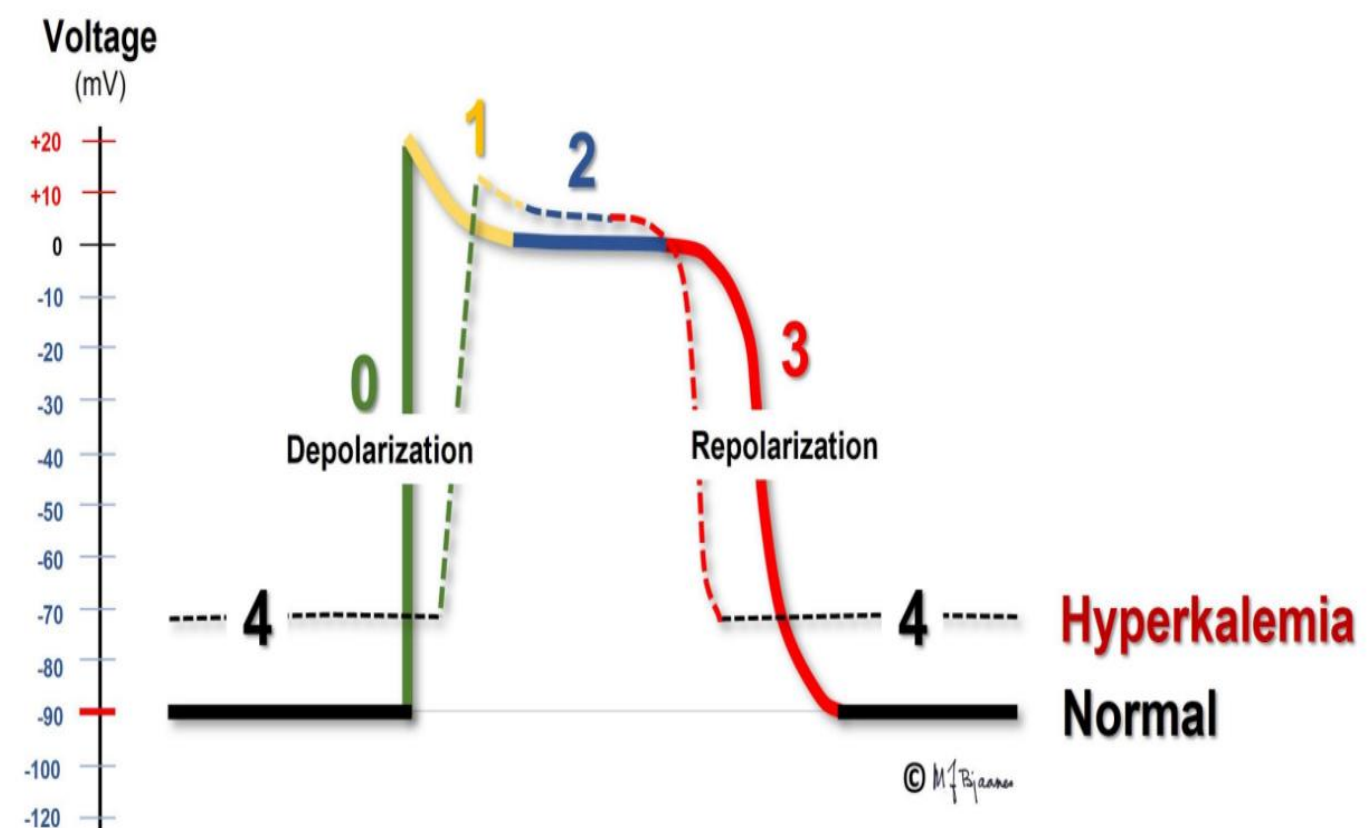
- Confirm elevated serum k
- ECG
- BUN , creatinine , eGFR , Cr clearance
- Glucose level , CBC
- Urine k (<20 mEq/L : decreased renal excretion but osmolality should be < 700 mOsm/kg)
- ABG (acidosis as in DKA or RF)
- Urine analysis (proteinuria , hematuria and casts)

EKG CHANGES:

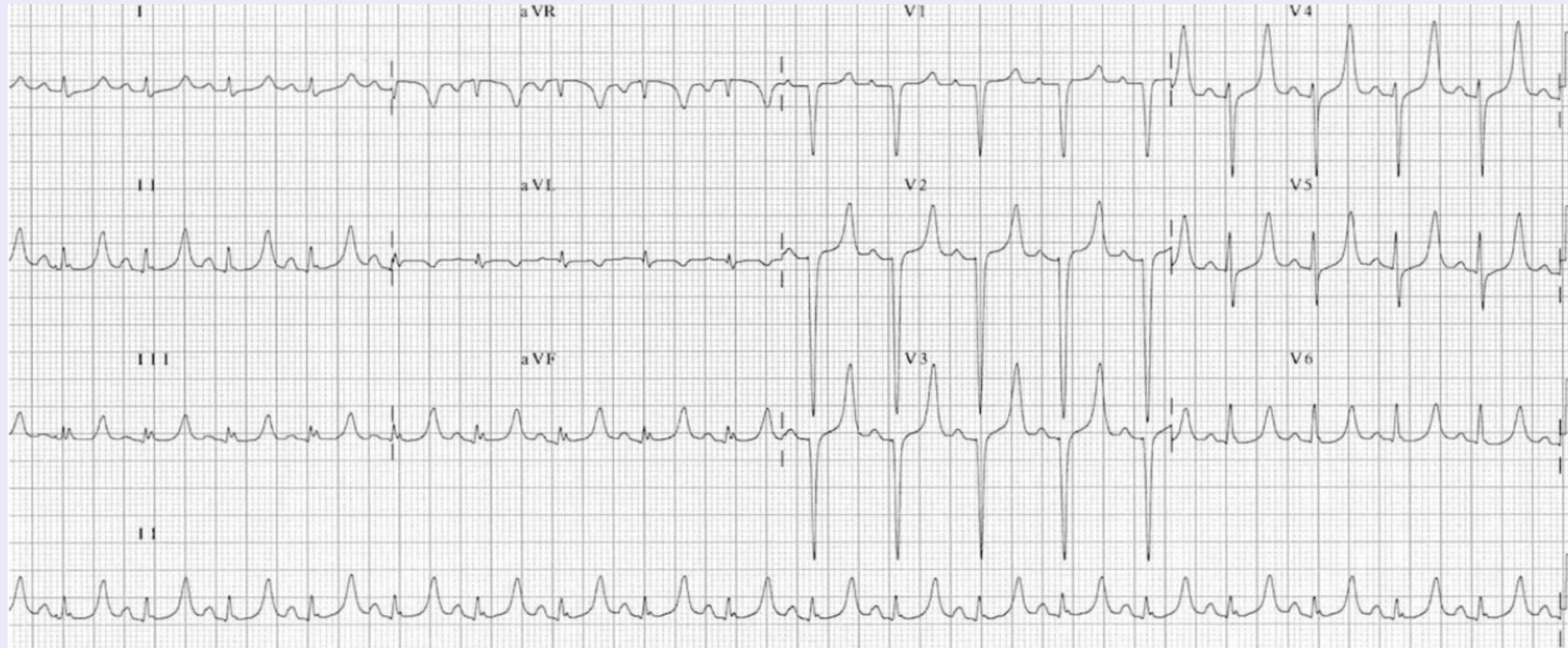


In clinical situation, it's not always K⁺ dependent level.

Hyperkalemia decreases cardiac excitability and contractility by raising the resting membrane potential:



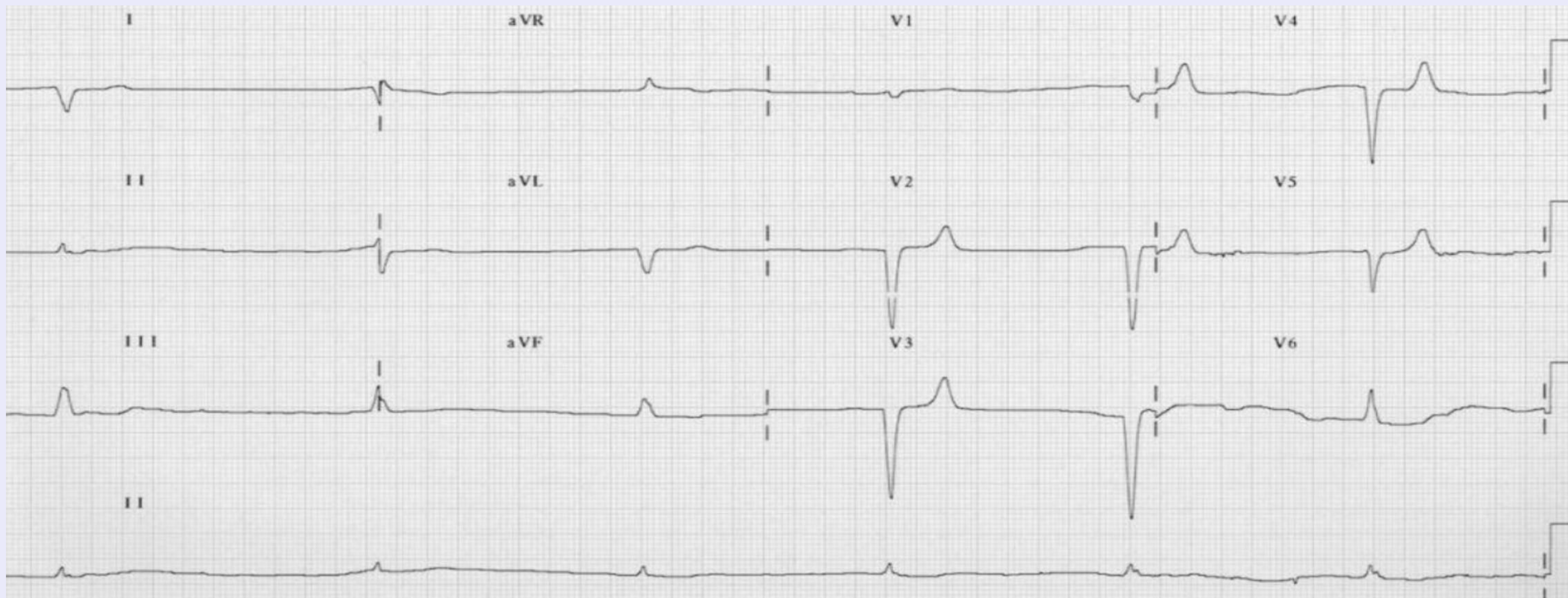
(1) Peaked T-waves



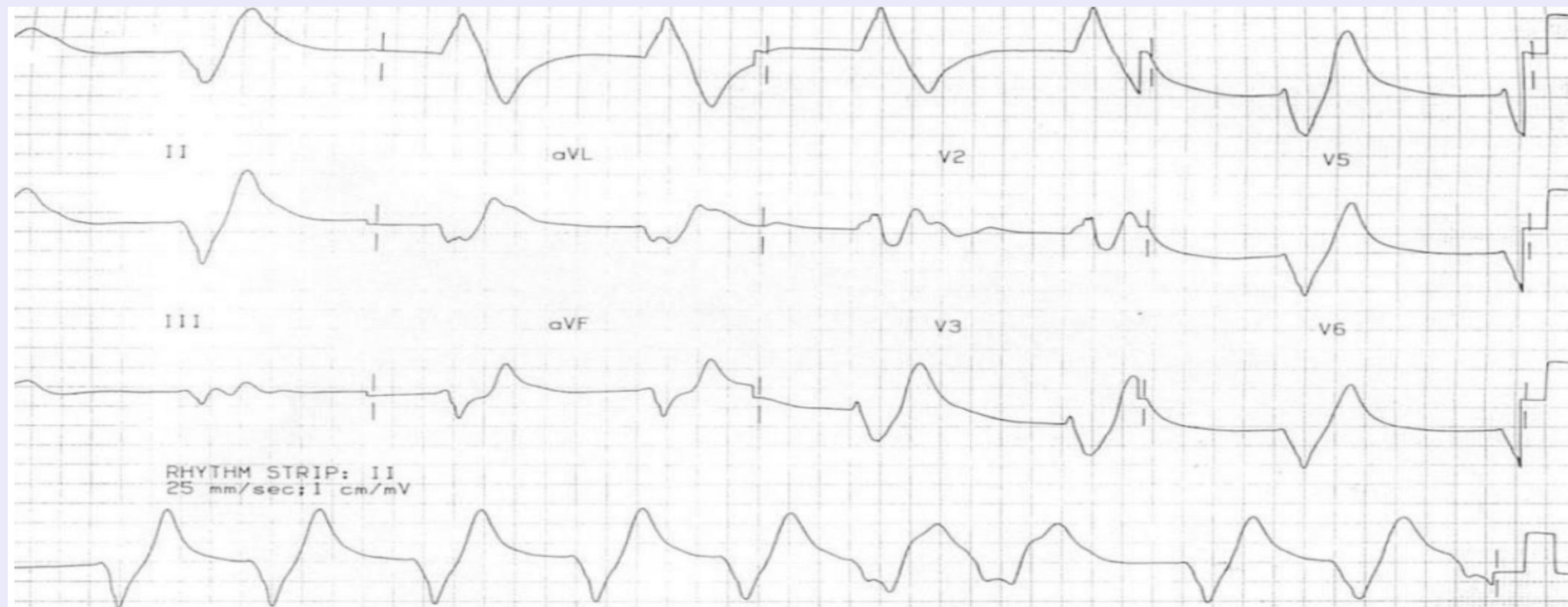
(2) Wide QRS

(3) Prolonged PR interval : Leads to bradycardia and Heart-block

(4) Flat or absence of the P wave : AV node taking over → junctional rhythm



5) Sine wave : The widened QRS complexes and peaked T waves are almost indistinguishable . The unique wave in patients with severe hyperkalemia Usually, this wave precedes VF and cardiac arrest → asystole



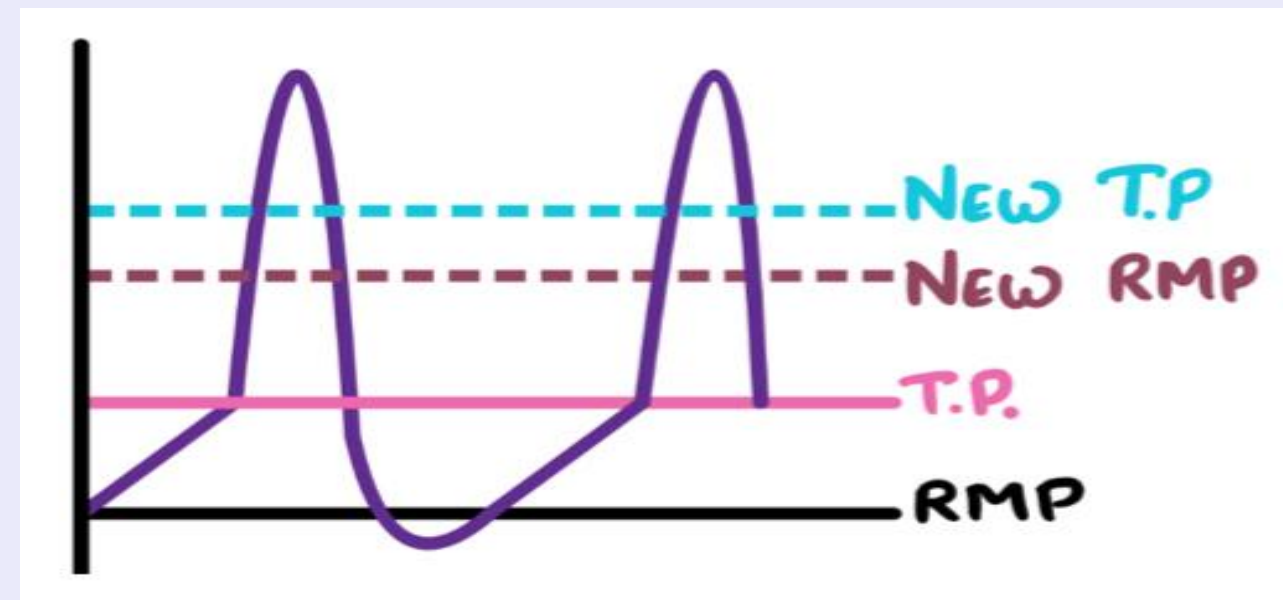
TREATMENT OF HYPERKALEMIA

First Rule out pseudo Hyperkalemia.

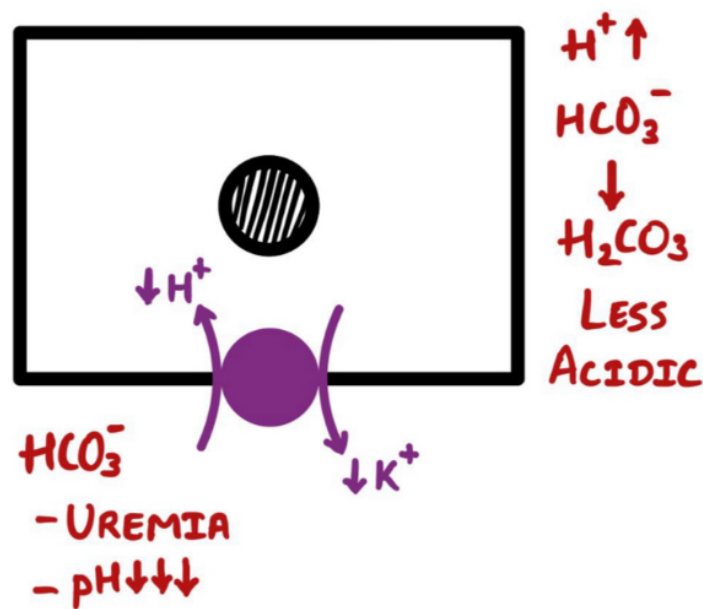
- THEN : Three main approaches to the treatment of hyperkalemia
 - 1) Cardiac membrane stabilization: BY ECG
 - 2) Intracellular potassium shifting : Push K⁺ back into the cells and get it out from the blood
 - 3) Enhanced potassium elimination
- TREAT UNDERLYING CAUSE
- DIALYSIS in resistance cases
- Patient must be on cardiac monitor in ICU

1) Cardiac membrane stabilization:

- Give calcium gluconate 10 ml of 10% IV over 2-3 minutes
- Hyperkalemia makes new RMP that is above the real RMP at which the Na⁺ channels are active
- **Calcium does not lower serum potassium**
- Calcium gluconate and calcium chloride Create a NEW threshold potential- So the cells may actually get to resting state

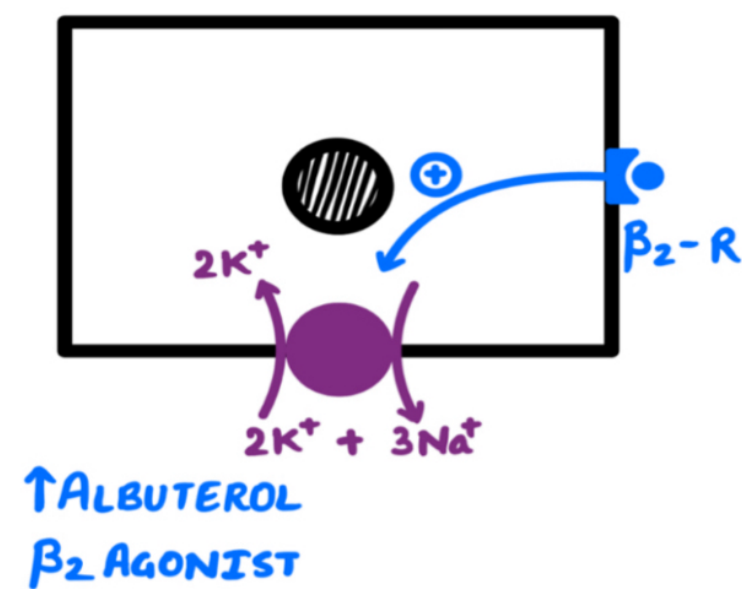


2) Intracellular potassium shifting :



Bicarbonate

Give it when there is severe acidosis
 $pH < 7.2$

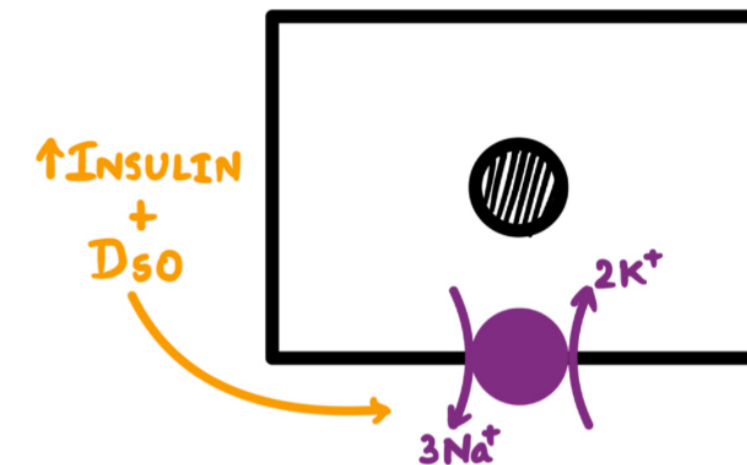


B2 agonist

Salbutamol or albuterol

Side effects :

Tachycardia and tremors



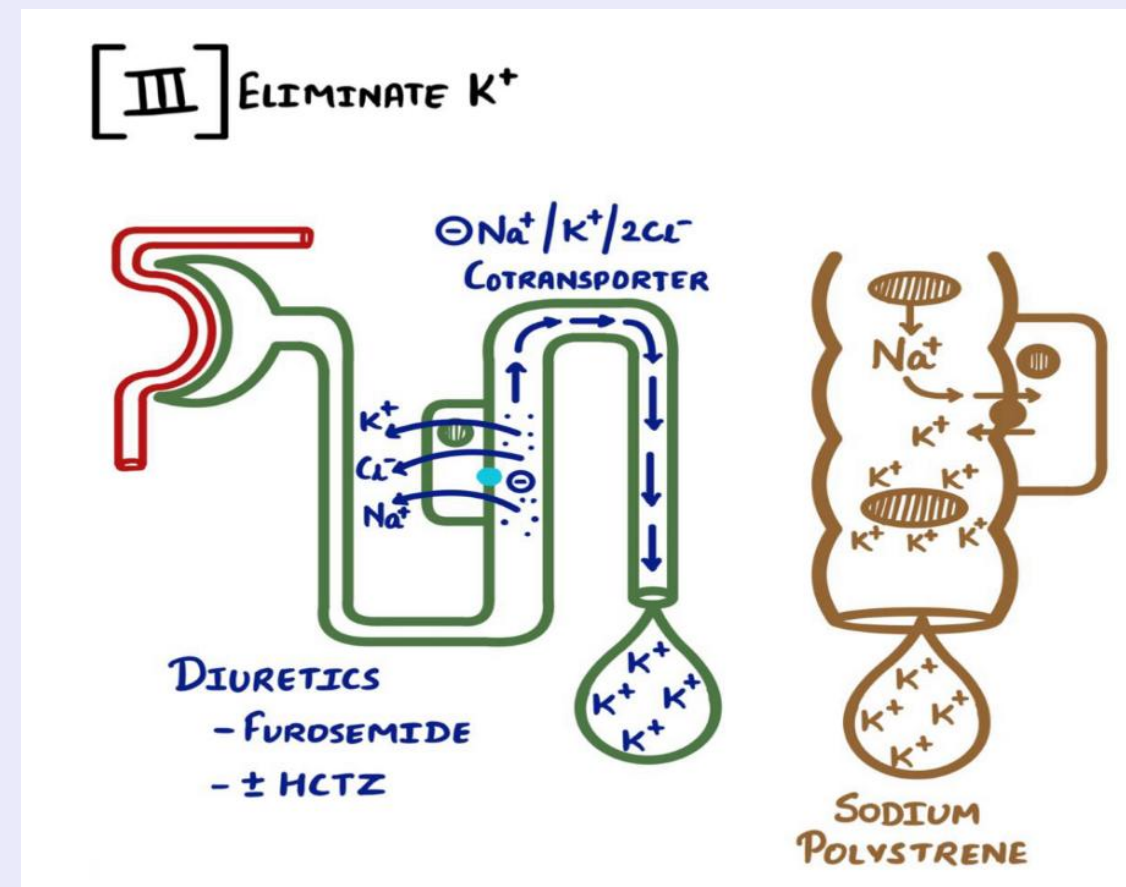
Insulin + dextrose

Give dextrose to avoid hypoglycemia

3) ELIMINATE THE K⁺ :

Only IF THE KIDNEYS AND BELLY HAVE THE ABILITY TO DO THAT !

- 1- Diuretics : GIVE LASIX (with IV saline to avoid hypovolemia)
- 2) Sodium polystyrene (Kayexalate)
- has dangerous side effect : intestinal necrosis





THANK YOU

PRESENTER (C2 GROUP)

- 1 Malek Al Zbedy
 - 2 Ibrahim Alhindy
 - 3 Shehab Al-Ammer
 - 4 Anoud Alsoud
 - 5 Asma'a Al Dawaimeh
- 