Potassium Imbalance

HYPOKALEMIA

HYPERKALEMIA

Done by : Mohammed Abu Qassem

Hypokalemia

- Introduction
- Signs and symptoms
- Causes
- Diagnosis
- Management and Treatment

Introduction

- Normal blood Potassium : (3.5-5) mEq/L
- Total body Potassium
- 1- intracellular (98%) (150 mEq/L)
- 2-extracellular (2%) (3.5-5 mEq/L)
- Daily Potassium intake can reach 100-150 mEq/L
- Internal balance is maintained by Na K Pump, K leaky channels and K rectifier channels.
- External balance (Excretion) is regulated by:
- 1- the Kidneys (80%) (ALDOSTERONE)
- 2-the GI tract (stool)
- 3-the Skin (sweat)

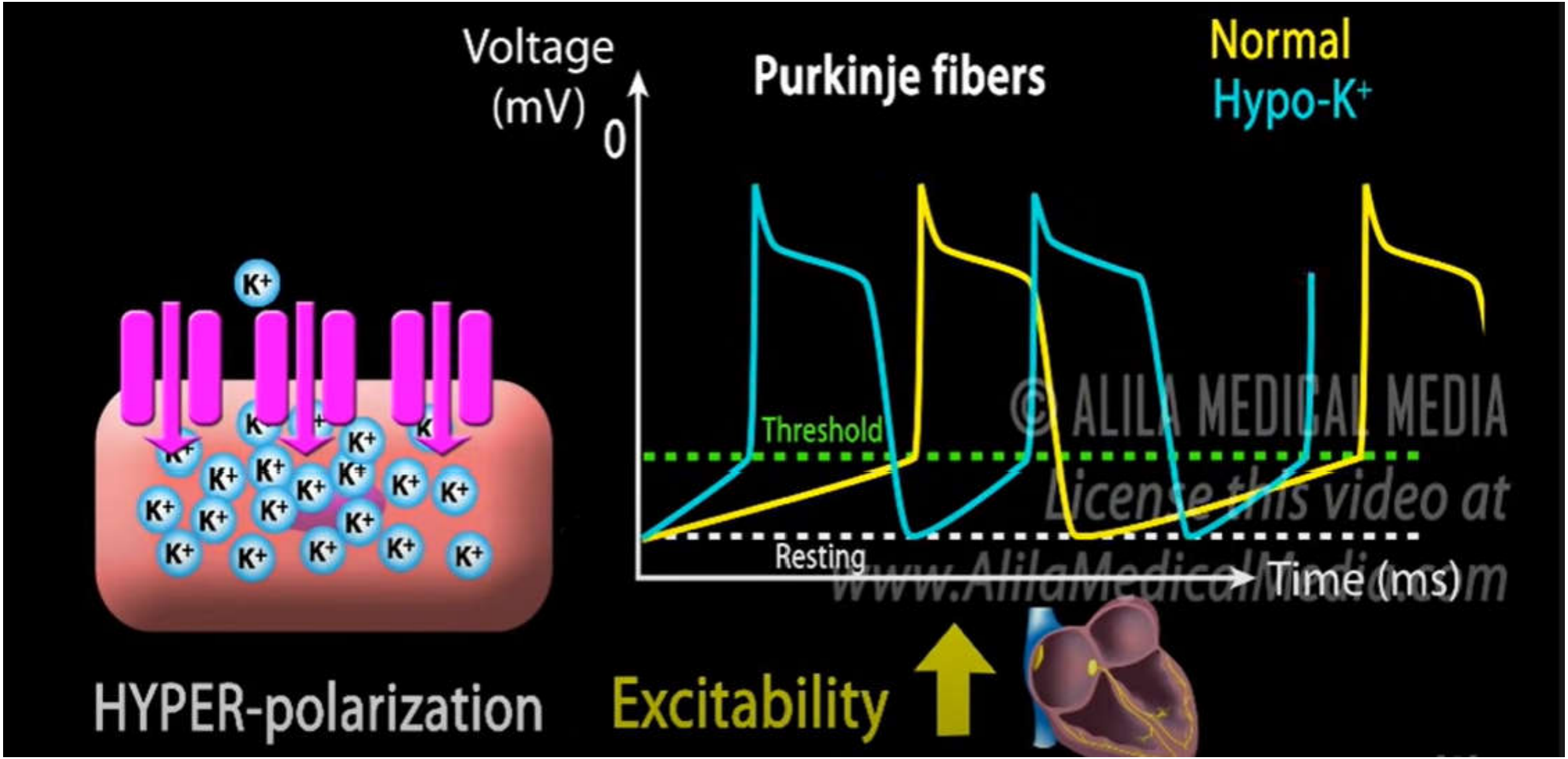
- Potassium has an important role in the action potential of skeletal , cardiac and smooth muscles .
- So a patient with hypokalemia or hyperkalemia will have symptoms related to:
- 1-arrhythmias
- 2-ECG changes
- 3-muscle weakness

Signs and Symptoms of Hypokalemia :

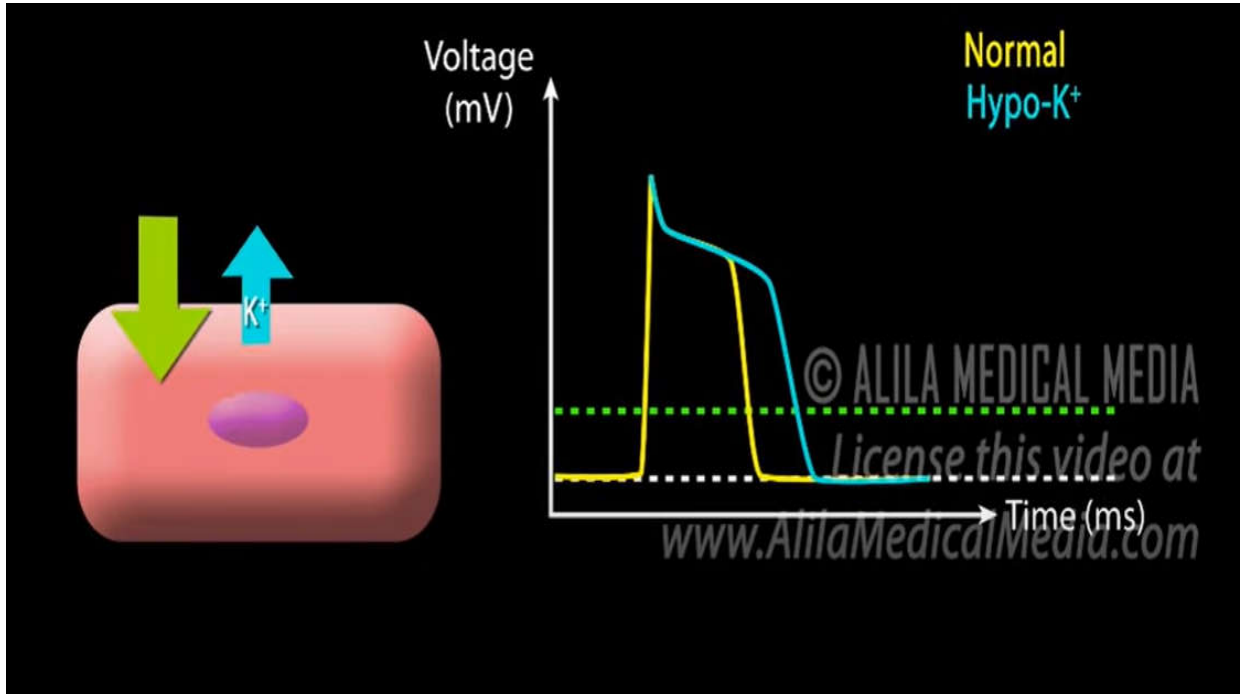
MILD HYPOKAIAEMIA plasma K <3mmol/L Asymptomatic.
SEVER HYPOKALAEMIA plasma K <2.5mmol/L

- 1-skeletal muscles : diminished contraction (weakness/fatigue/ cramps/flaccid paralysis) (lower the upper limbs)
- 2-smooth muscles :(constipation / hypotension / paralytic ileus)
- 3- respiratory muscles :(respiratory depression/hypoventilation)
- 4-cardiac muscle :
- A- arrhythmias (PVC/PAC/fibrillation/tachycardia)
- B-ECG (Flat T wave / U wave / T wave invertion/ST-depression)

Hypokalemia and Arrhythmias:



ECG changes :


Voltage
(mV)
Normal
Hypo-K+
K+
Time (ms)
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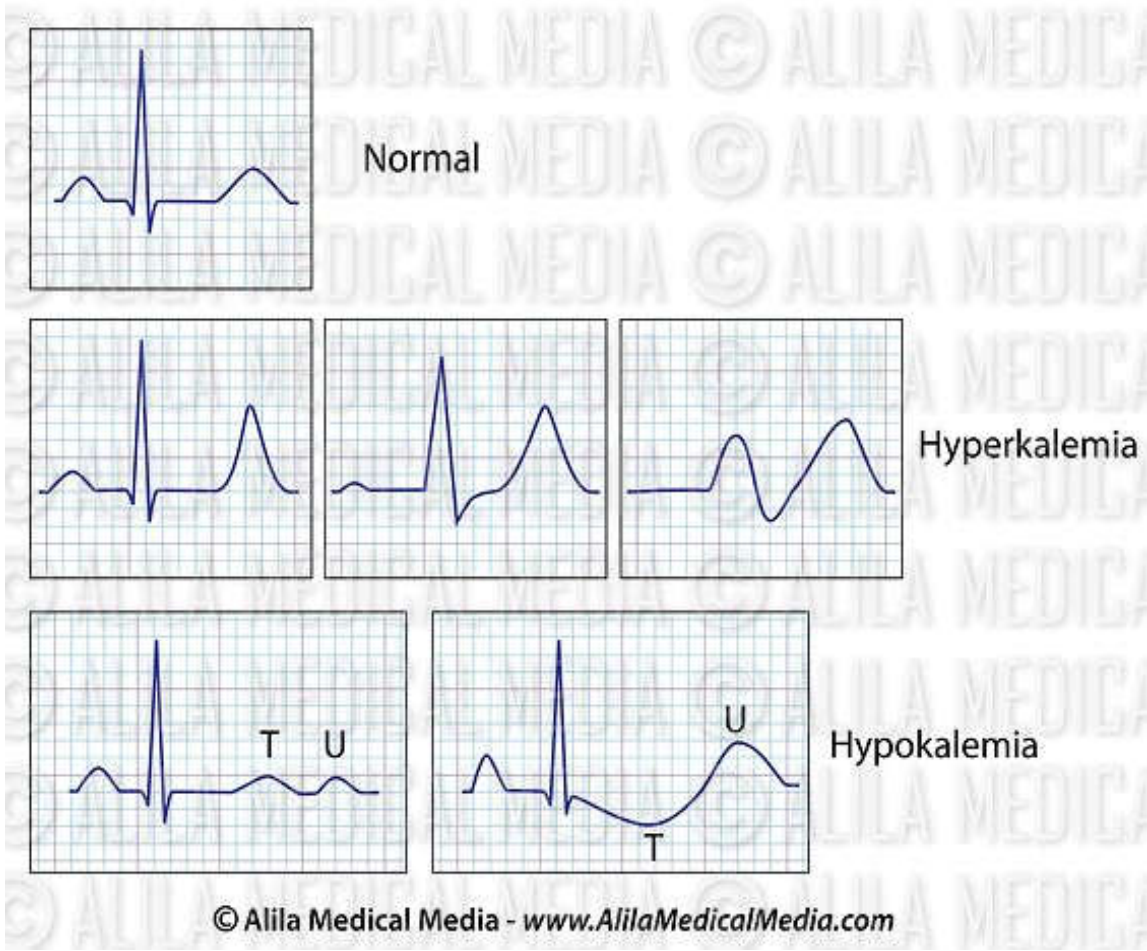
Normal
Hyperkalemia
T U
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Hypokalemia
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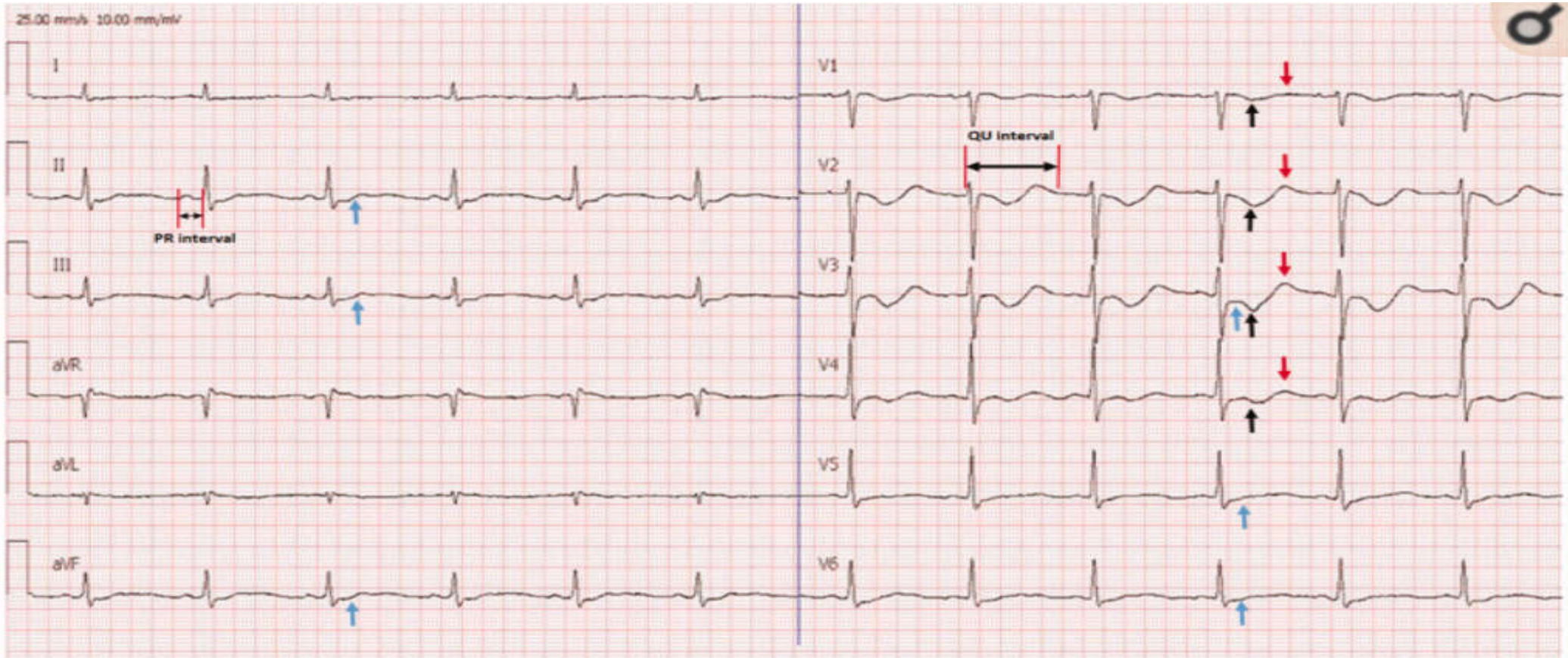

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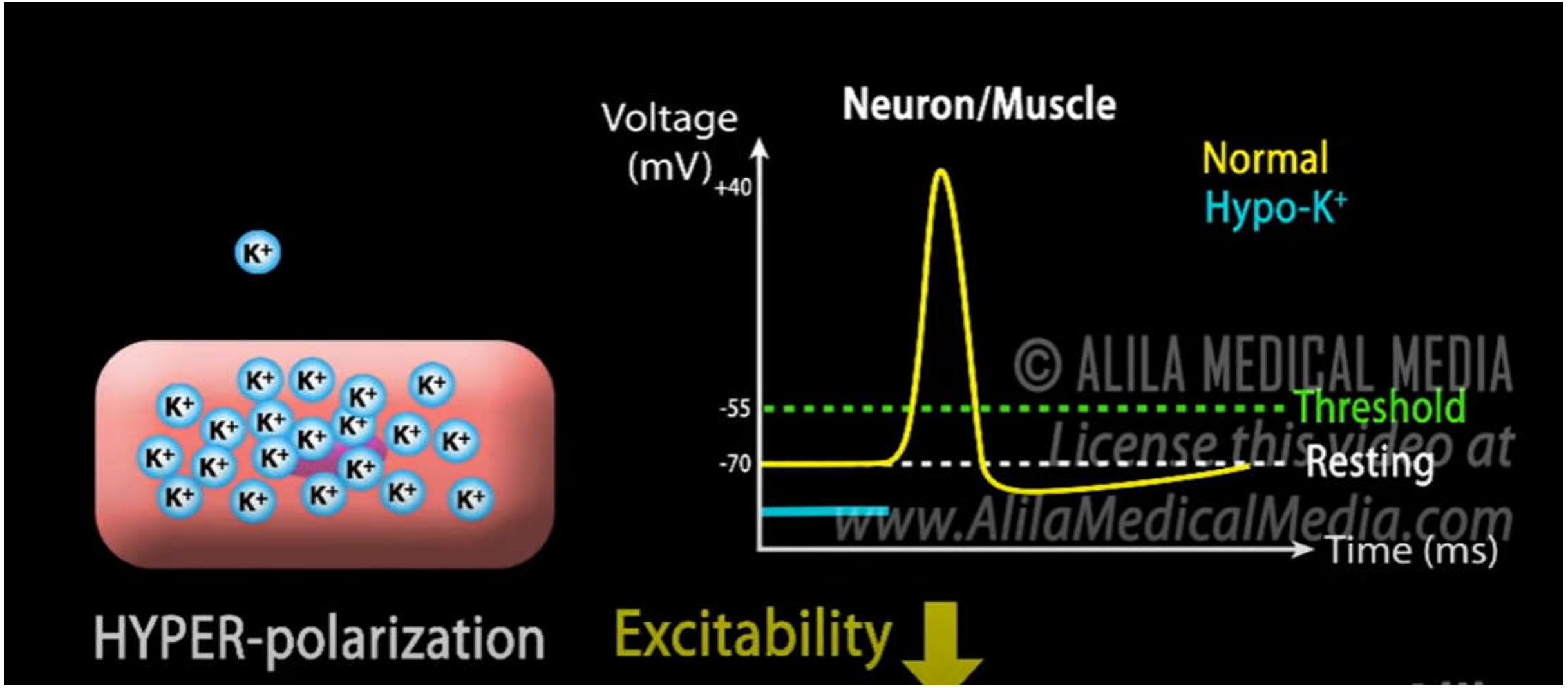
Figure 1.

Twelve-lead electrocardiogram taken on admission from a 57-year-old man with severe diarrhea, paralysis of the lower extremities, weakness, inability to walk, and severe hypokalemia (1.31 mmol/L; normal value: 3.5–5.5 mmol/L). Electrocardiography shows bradycardia, a prolonged PR interval, a prolonged QU interval, ST-segment depression, T wave inversion, U waves best seen in the precordial leads (particularly in leads V2–lead

Hypokalemia and skeletal muscles.



Hypokalemia

Done by:rana alhajri
4th year medical student

HYPOKALEMIA

- Total body K+ can essentially be spread into 2 components:
- Intracellular K+ ➡

- Extracellular K+ ➡ INCLUDE BOTH
- Intravascular space: which it space with blood and lymphatic vessels
- Interstitial space: space between cells where we typically found (fibers – protein –and long chains of carbohydrate which called glycosaminoglycan
- The vast majority around 98% of all the body K+ is intracellular

- In fact the concentration of K+ inside the cells is about 150 meq/L where's outside the cells is only about 4.5 meq/L

- Keep in mind that these K+ ions carry a charge so the different in concentration will lead to a different in charge which establish an electrochemical gradient and this called ➡ INTERNAL K+ BALANCE
- This balance Is maintain by the NA/K+ PUMP

- THIS concentration gradient is extremally important for setting the resting membrane potential which needed for COCNTRACTION of smooth – cardiac-skeletal muscles

External K+ balance

Total dietary potassium intake

- ▶Total dietry intake of K is 50-150 mmol/day or 1mmol/kg/day
- ▶90% of it is absorbed in GI tract
- ▶sudden rise in plasma K+ is prevented by:
- Shift of K+ into the cell by insulin
- Exess K+ EXCRETED IN URINE

Excretion of K+

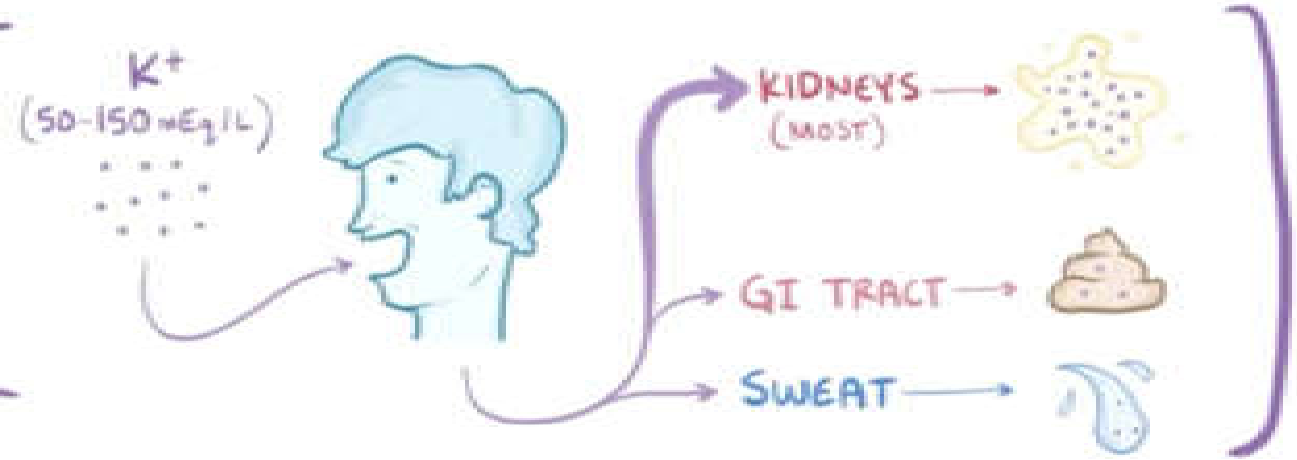
Renal excretion is the major route for elimination of dietary and other sources of K+ excess

Also there a small amount of dietary K+ Loss through the gastrointestinal , and sweat

HYPOKALEMIA ~ TOO LITTLE in BLOOD

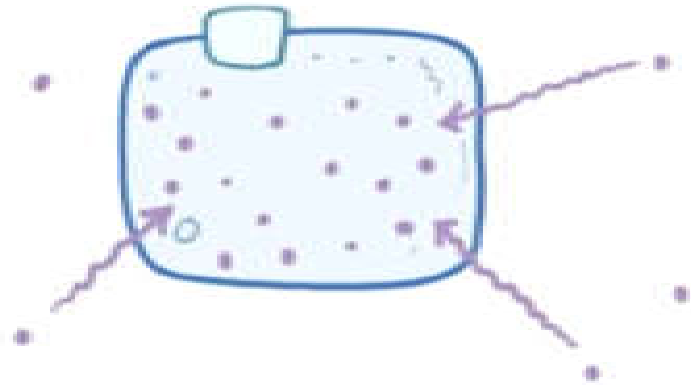
EXTERNAL BALANCE SHIFT

* INCREASED K^+ EXCRETION
  → DECREASE in BLOOD



INTERNAL BALANCE SHIFT

* K^+ MOVES INTO CELLS
  → DECREASE in BLOOD



HYPOKALEMIA

➡ It is potassium concentration less than 3.5 mmol/L

- CAUSES:
- GI losses:
- ▶Vomiting and nasogastric drainage(volume depletion and metabolic alkalosis also result)
- ▶Diarrhea
- ▶Intestinal fistulas- particularly after inflammatory bowel disorders such as Chrons
- ▶Decreased potassium absorption in intestinal disorders

HYPOKALEMIA

- CAUSES:
- RENAL LOSSES:
- ➡Diuretics
- ➡Renal tubular disease
- ➡Excessive glucocorticoids-due to mineralocorticoid action at high serum levels
- ➡Bartter syndrome-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 leas to increased renin levels and secondary aldosterone elevations

HYPOKALEMIA

CAUSES:

other causes:

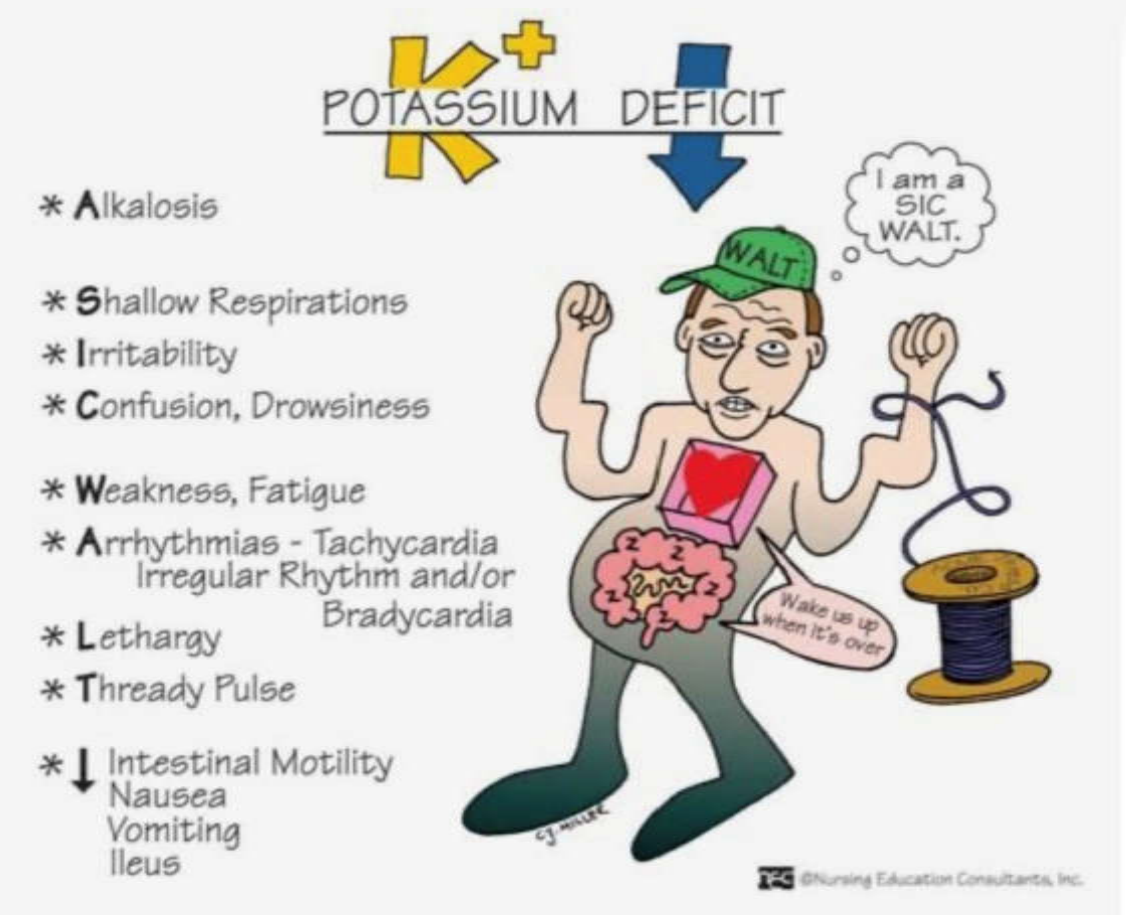
- insufficient dietary intake
- Insulin administration
- sweating
- certain antibiotics espcially(amphotericin B)

What is renal tubular acidosis!

- Renal tubular acidosis (RTA)occurs when the kidneys do not remove acids from the blood into the urine as they should . the acid level in the blood then become too high , a condition called acidosis. Some acid in the blood is normal , but too much acid can disturb many bodily functions
- There are three main types of RTA
- We will discus them in the next slide

	Type 1	Type 2	Type 4
Location	Distal nephron	Proximal tubule	Distal nephron
Defect	Failure of H+ secretion in distal nephron	Failure of bicarbonate resorption in proximal tubule	Reduced secretion of H+ and K+ in distal tubule from aldosterone deficiency or resistance
Urine pH	High (>5.5)	Variable	Variable, usually <5.5
Plasma K	Low	Low	High
Plasma HCO3	<10	12-20	>17
Causes	Sjogren's, SLE, lithium, amphotericin, sarcoidosis, hyperparathyroidism, Wilson's, etc	Fanconi syndrome, MGUS, multiple myeloma, acetazolamide, congenital carbonic anhydrase deficiency	Obstructive uropathy, NSAIDs, adrenal insufficiency, spironolactone, (pseudo)hypo-aldosteronism
Associated conditions	Calcium phosphate stONEs (from hypocitraturia + hypercalciuria), osteomalacia.	Growth retardation, osteomalacia (Tiny Tim, Type Two)	Hypertension
Treatment	Oral sodium bicarbonate, citrate	Oral sodium bicarbonate plus potassium	Control hyperkalemia, remove offending agent

THIS is a quick summary of potassium defect!



Thank you for your listening

Diagnosis & Treatment of Hypokalemia

By Mohammad Elayyan – 1831159

Group C44

Hypomagnesemia

- 50% of hypokalemia has **concomitant magnesium deficiency**.
  - Loop diuretics or thiazide therapy
  - Diarrhea
  - Alcoholism
  - Bartter & Gitelman syndromes
  - Tubular injuries from nephrotoxic drugs
- The mechanism of hypokalemia in magnesium deficiency **remains unexplained**, some theories suggest:
  - Magnesium deficiency **impairs the Na^+-K^+ ATPase** reducing cellular uptake, and with increased renal and GI excretion there will be resulting hypokalemia
  - Magnesium also **decreases K+ secretion from the distal nephron**

Diagnosis

$[K+_{Serum}] < 3.5$ mEq/mL

Redistribution

- Hx of **Insulin/Epinephrine** administration
  - Administer KCl and monitor carefully to prevent Rebound Hyperkalemia.
- Metabolic Alkalosis

Actual K^+ Depletion

- $[K^+_{Urine}]$ **< 20 mEq/L**
  - Extrarenal K^+ Loss
- $[K^+_{Urine}]$ **> 20 mEq/L**
  - Renal K^+ Loss

Extrarenal K^+ Loss

$[K^+_{Urine}] < 20$ mEq/L

- Next step, check ABGs
- **Normal** acid-base
  - Decreased K^+ intake
  - **Laxative** abuse
  - Copious perspiration (Excessive **Sweating**)
- Metabolic **Acidosis**
  - GI losses; **Diarrhea**/Fistula/Villous adenoma
  - Anorexia/**bulimia** nervosa

Renal K+ Loss

$[K^+_{Urine}] > 20$ mEq/L

- Next step, check ABGs
- Metabolic **Acidosis**
  - Renal tubular acidosis (types I & II)
  - Carbonic anhydrase inhibitors administration (acetazolamide)
  - DKA
  - Ureterosigmoidostomy
- Metabolic **Alkalosis**
  - Severe potassium depletion leads to **redistribution of H+ from the ECF to ICF**.
  - In the process, ECF HCO3– is gained.

Urinary Na^+/Cl^- Ratio

- In **tubular disorders** → ratio ≈ **1** (both $[Na^+_{Urine}]$ & $[Cl^-_{Urine}]$ are high)
- In **laxatives** abuse → ratio ≈ **0.4**
- In **anorexia/bulimia nervosa** patients → ratio ≈ **1.6**

Treatment

- Identify and treat the **underlying cause**
- Discontinue any **medications** that can aggravate hypokalemia

Potassium Chloride (KCl)

- Oral or IV
  - Oral is preferred
- Oral KCl is the **safest and most appropriate**
  - 10 mEq → increase K^+ levels by 0.1 mEq/L (more if patient has renal insufficiency)
  - Comes in slow-acting and long-acting forms.
- However, if patient has **severe hypokalemia** ([K+ Serum] < 2.5 mEq/mL) or is symptomatic → **IV KCl**

IV KCl

- In cases of **severe** hypokalemia (< 2.5 mEq/L), **symptomatic**, pr patient **cannot take oral** KCl
- **Immediate** effect
- Give **slowly**
  - To avoid **hyperkalemia**
  - Can cause **phlebitis** and/or **arrythmias**
- Monitor K+ concentration and cardiac rhythm
- Infusion rates
  - Peripheral line → 10 mEq/hr
  - Central line → 20 mEq/hr
- Potassium **BURNS!** Add 1% lidocaine to decrease pain

Potassium Sparing Diuritics

- Spironolactone, Eplerenone, Amiloride … etc.
- Inhibit **aldosterone** → Inhibit K^+ secretion → HYPERKALEMIA
- But in patient with hypokalemia (especially chronically low K^+) it is approved

Hypomagnesemia & Hypokalemia

- Hypomagnesemia **promotes K^+ loss**
- Cannot correct hypokalemia **until hypomagnesemia is corrected**
- **ALWAYS TREAT HYPOMAGENESIAMIA FIRST IF PRESENT**

Hyperkalemia

Done by Asem AL-Rawashdeh

Introduction to hyperkalemia

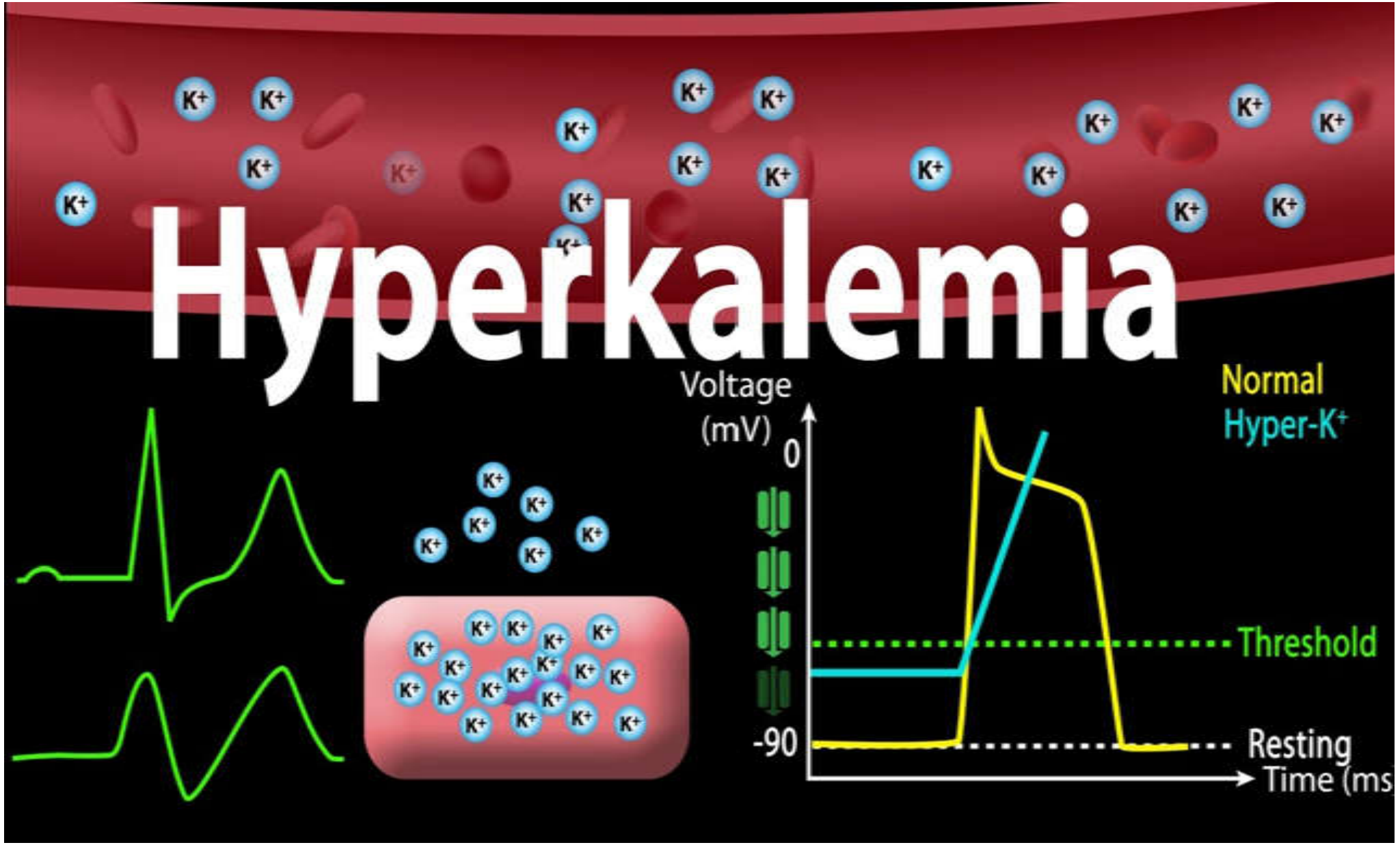
- Hyper means more than, kal stands for potassium and emia means blood so hyperkalemia is:
- the Increased levels of potassium in the blood more than 5 mEq/L
- Happens mainly because of :
- 1- decreased excretion
- 2- excissive intake
- 3- extracelluar shift

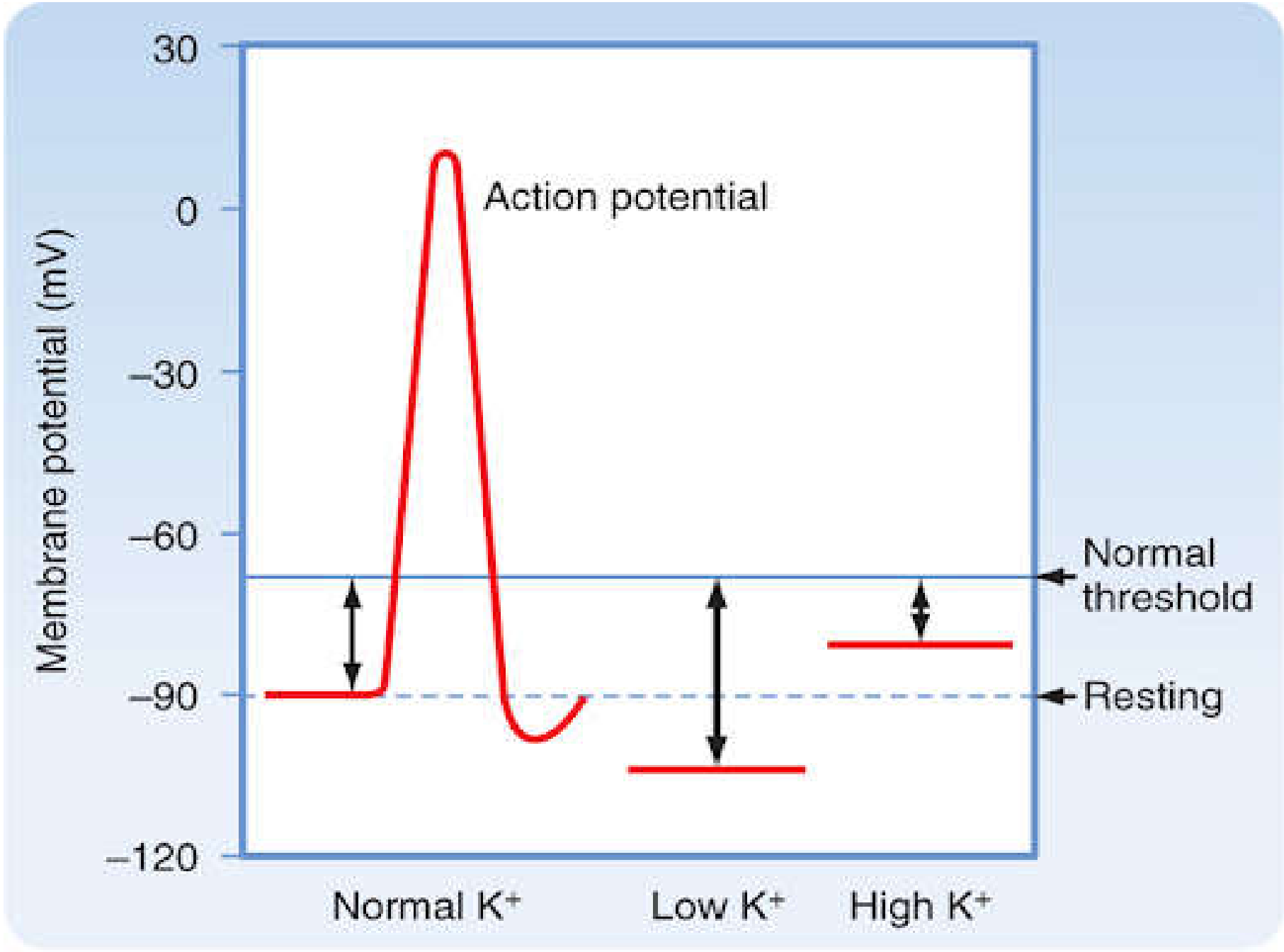
hyperkalemia

- The most scenario is renal insufficiency combined with excessive intake or certain drugs(ACE inhibitors)
- Chronic hyperkalemia is less dangerous because over time the kidneys adapt to excreting more potassium
- Acute hyperkalemia, however, can be fatal because the primary effect is on cardiac functions

Signs and symptoms

- Mostly asymptomatic
- Severe/rapid-onset hyperkalemia cause:
- Muscle weakness, flaccid paralysis (starts in lower extremities, moves upward) → respiratory failure
- Decreased deep tendon reflexes
- Arrhythmias, cardiac arrest
- Nausea, vomiting, intestinal colic, diarrhea


Hyperkalemia
Voltage
(mV)
0
-90
Normal
Hyper-K+
Threshold
Resting
Time (ms



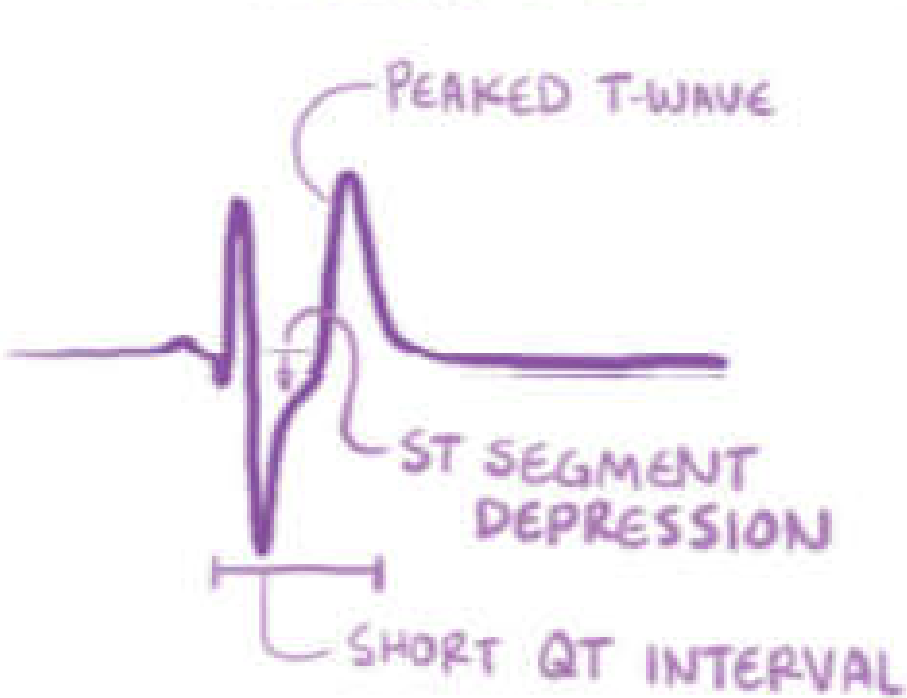
- Ekg changes include:
- Prolonged PR interval, tall, peaked T-waves with narrow base, shortened QT interval, depressed ST segment
- In severe cases:
- small/indiscernible P wave, widened QRS complex → strip mimics sine wave

- MNEMONIC: MURDER for Signs & symptoms of Hyperkalemia:
- Muscle weakness
- Urine: oliguria, anuria
- Respiratory distress
- Decreased cardiac contractility
- EKG changes: peaked T waves; QRS widening
- Reflexes: hyperreflexia or areflexia (flaccid)

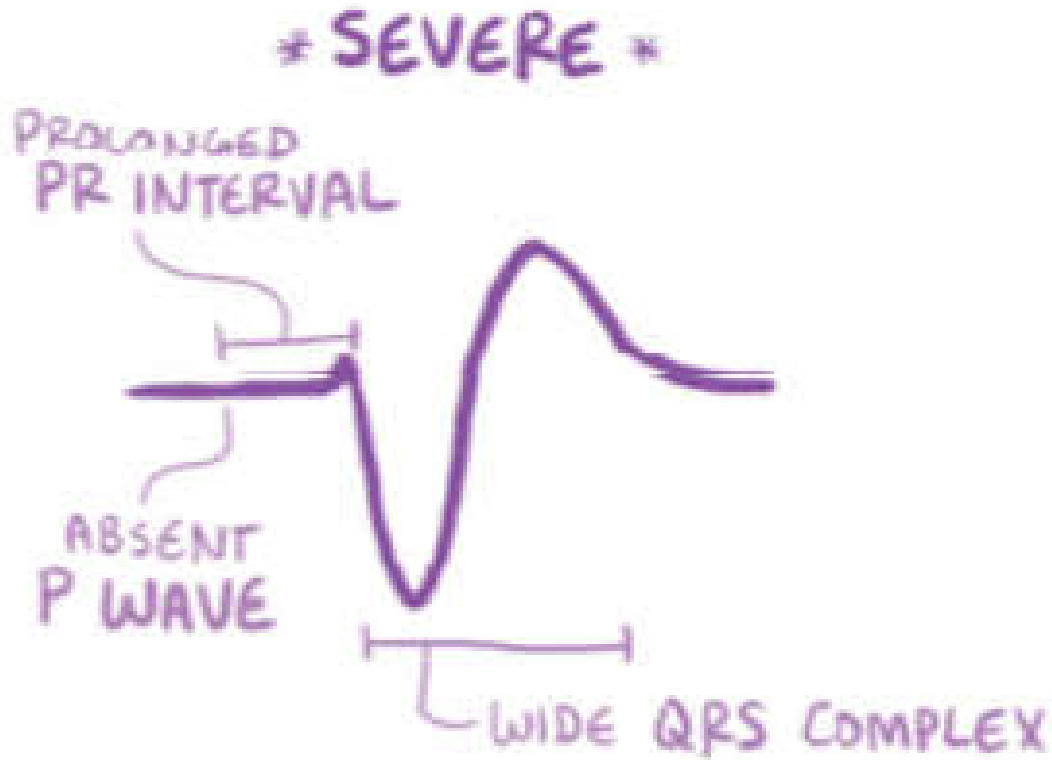
EKG changes in hyperkalemia

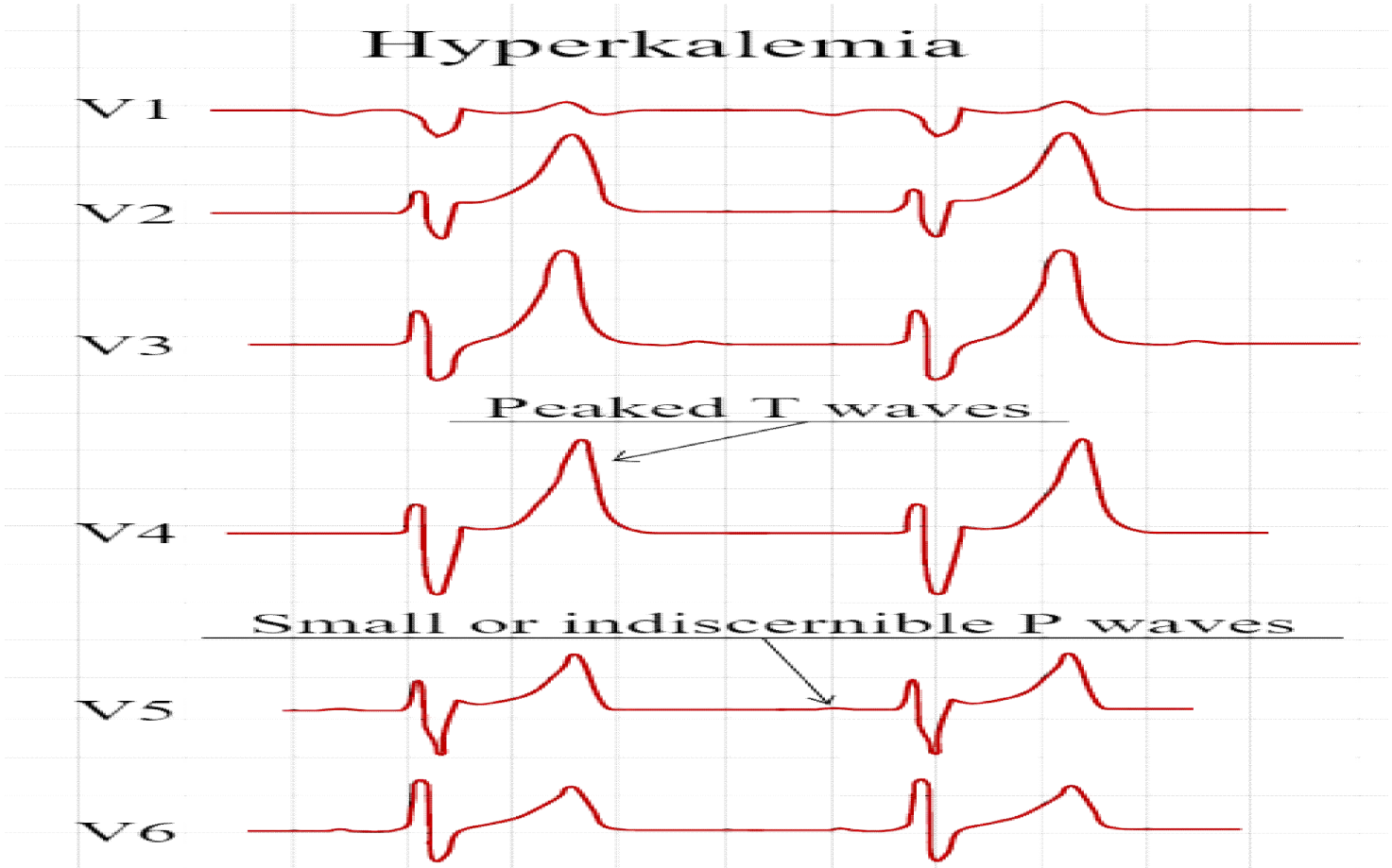
* ↑ $[K^+]$ in BLOOD ~ > 5.5 mEq/L

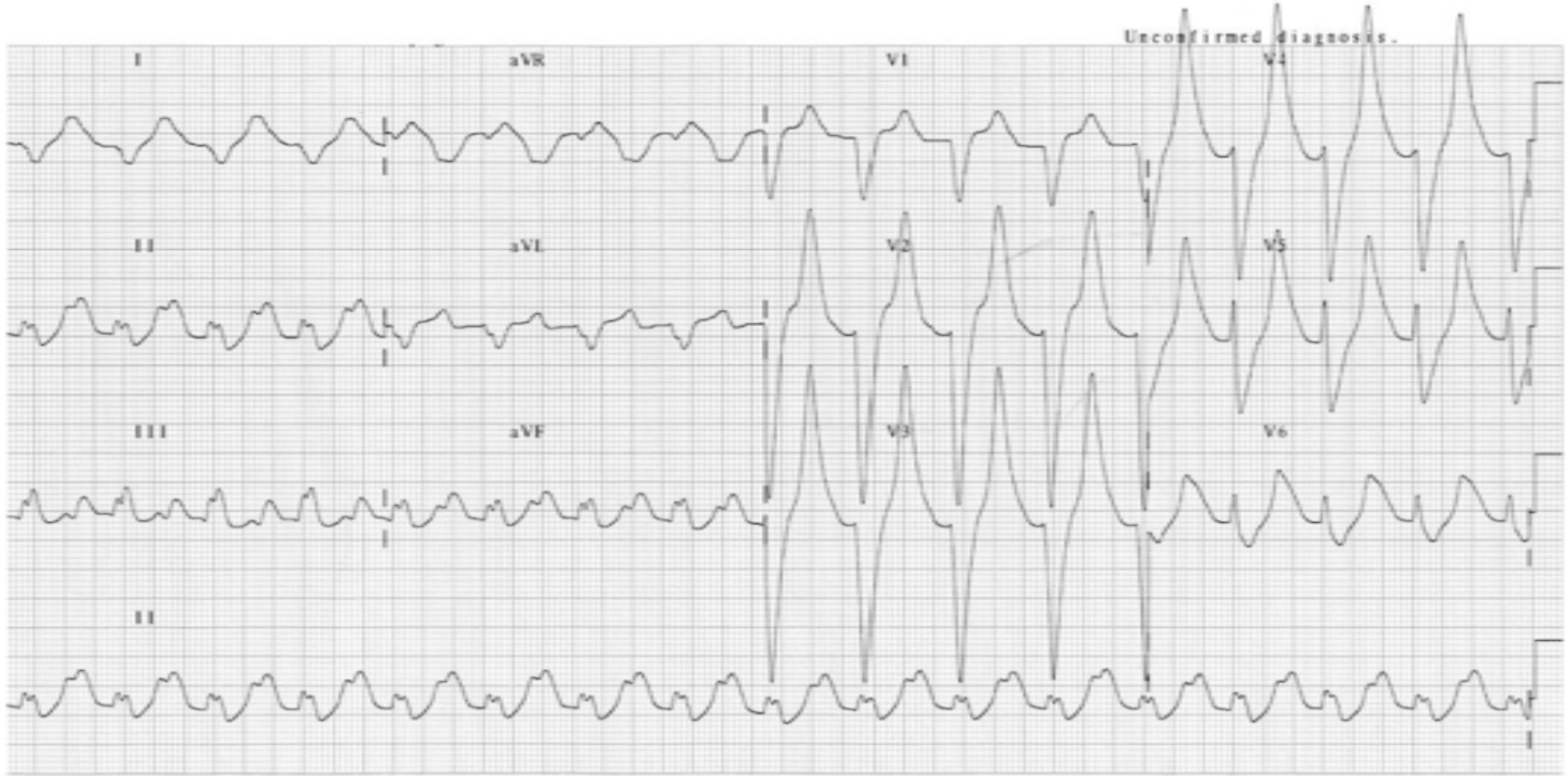
* ELECTROCARDIOGRAM



* SEVERE *




Hyperkalemia
V1
V2
V3
Peaked T waves
V4
Small or indiscernible P waves
V5
V6



This ECG displays many of the features of hyperkalaemia:

- Prolonged PR interval.
- Broad, bizarre QRS complexes — these merge with both the preceding P wave and subsequent T wave.
- Peaked T waves.

This patient had a serum K+ of 9.3

Causes of Hyperkalemia

by Ahmad Khalid Ibrahim

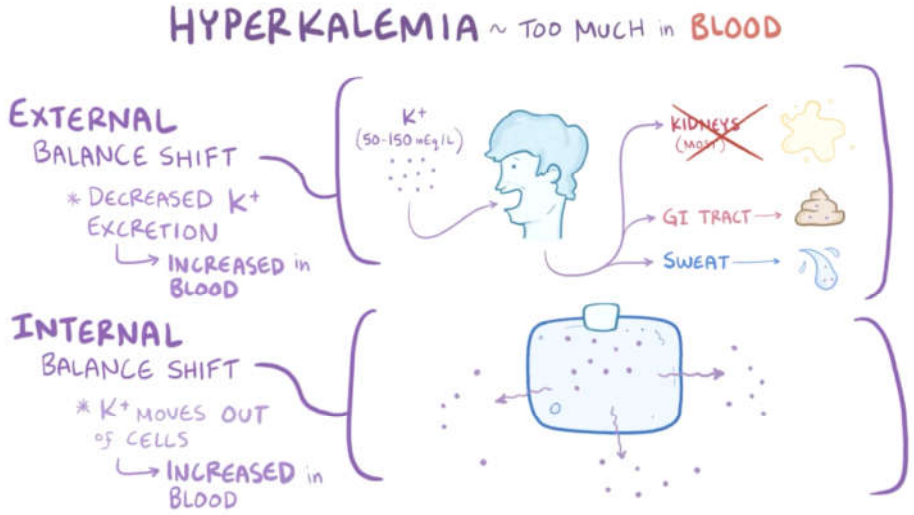
7th NOV 2021

Mechanisms of Hyperkalemia

A. Internal potassium [k+] shift (due to leak of intracellular k+ out of cells)

B. Reduced [K+] excretion from body (most common cause)

C. Rapid excessive [K+] intake

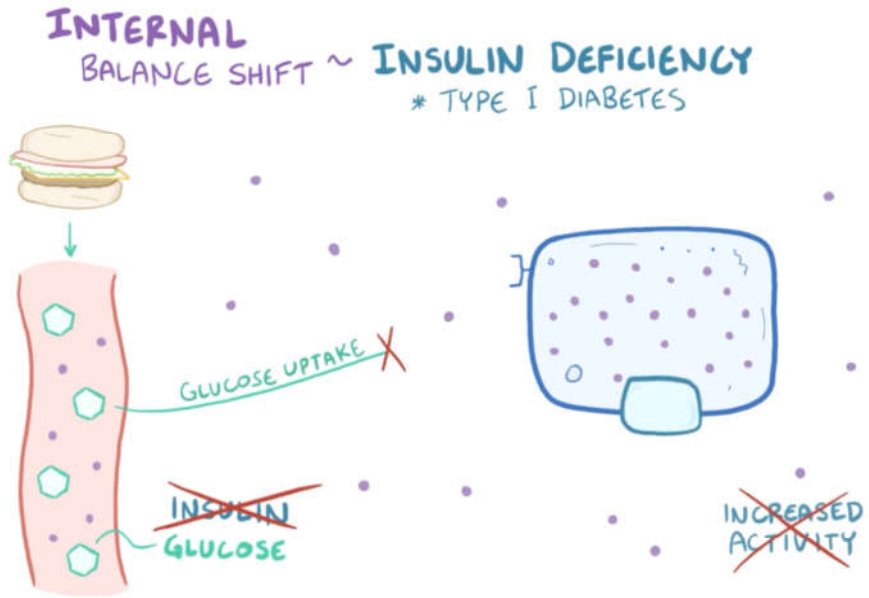


A. Internal potassium shift

Causes of Hyperkalemia

A.1 Insulin deficiency

- Insulin binding to cells increases the uptake of glucose and activity of Na/K pump.
- Deficiency in insulin (like in DM type1) results in less activity of Na/K pump, thus reduced cellular uptake of K+.
- The K+ ions from a meal will thus accumulate in blood stream, causing Hyperkalemia



Causes of Hyperkalemia

A.2 Acidosis

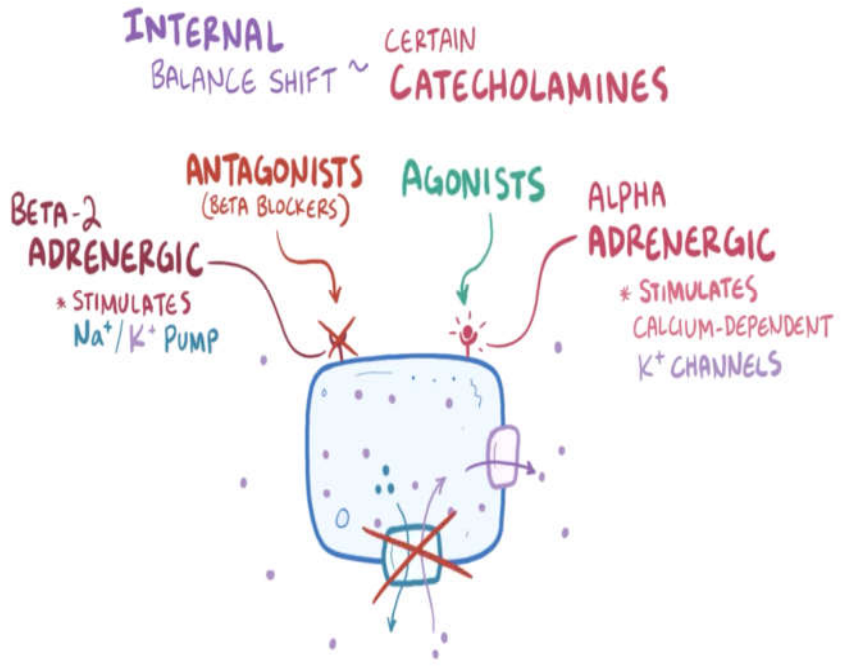
- In state of acidosis, body will combat it by replacing H+ ions from blood with intracellular K+ ion.
- Acidosis is reduced and results in Hyperkalemia.
- An exceptions for this are:

1. respiratory acidosis where [CO2-] can move into cells freely due to its lipid solubility.
2. metabolic acidosis from excess organic acids (lactic & ketoacid), they can enter cells without the exchange of K+ ions.

Causes of Hyperkalemia

A.3 Some catecholamines

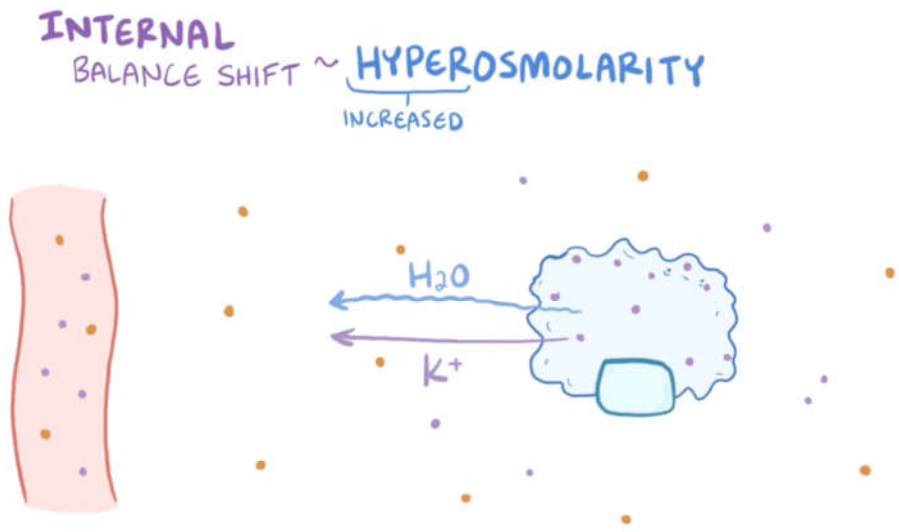
- Alpha & Beta-2 adrenergic receptors both affect K+ concentration.
- Alpha receptors activation causes outward movement of K+ via stimulation of Calcium-dependent K+ channels. Thus Alpha agonists cause Hyperkalemia
- Beta-2 receptors activation stimulates Na/K pump, exchanging Na+ ions for K+ ion. Thus beta antagonist/beta blocker causes Hyperkalemia.



Causes of Hyperkalemia

A.4 Hyperosmolarity

- 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.



Causes of Hyperkalemia

A.5 Cell lysis

- Since cells store 98% of body K+ ions, cells breakdown/lysis will cause it to release to interstitial fluid and blood causing Hyperkalemia.
- It is only significant when on a large scale like in the cases of:
  - Sever burns
  - Rhabdomyolysis (skeletal muscles breakdown)
  - Tumor lysis during chemo therapy

Causes of Hyperkalemia

A.6 Exercise in specific people

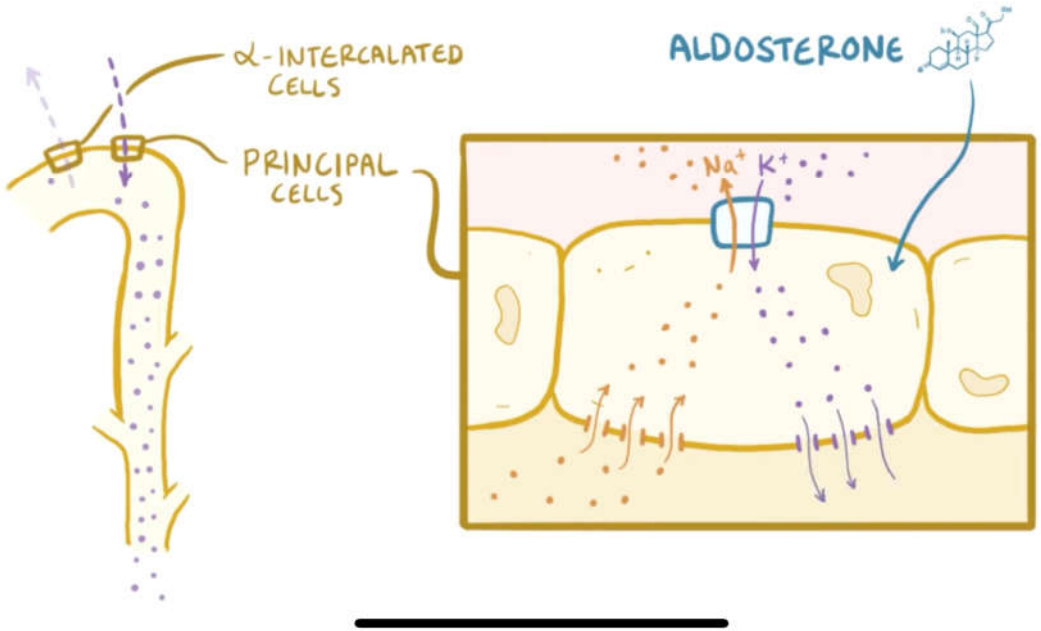
- Depletion of cellular ATP causes K+ channels to open and K+ moves out of cells into blood.
- this shift is insignificant normally, however in a person with kidney problems or on a beta blockers medication it can result in a Hyperkalemia.

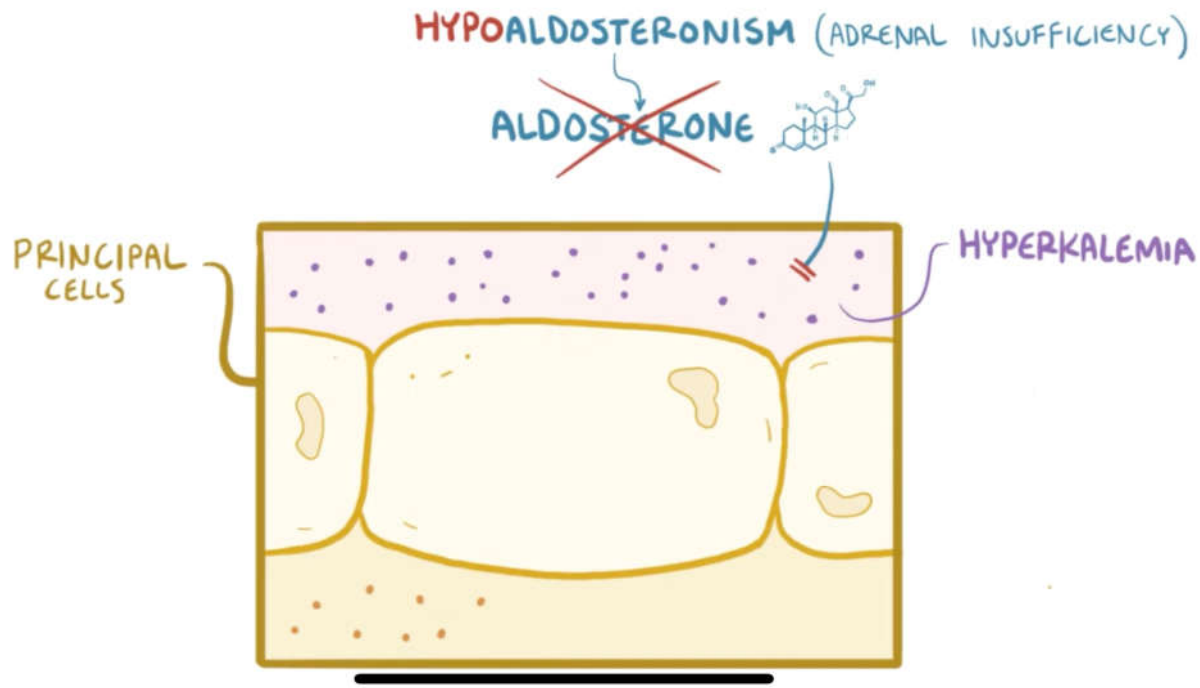
B- Reduced potassium excretion

Causes of Hyperkalemia

B.1 Adrenal insufficiency/Hypoaldosteronism

- Aldosteron increases Na reabsorption and K secretion in the distal convoluted tubule part of nephrons throw its effect on principal cells.
- In case of reduced aldosterone or its absence the patient will develop Hyperkalemia and hyponatrimia due to the inability of excreting K and reabsorbing Na.
- In addition to pathological causes there are drugs that reduce the effect of aldosterone thus causing Hyperkalemia in similar mechanism.


α-INTERCALATED CELLS
PRINCIPAL CELLS
ALDOSTERONE
Na^+ K^+


HYPOALDOSTERONISM (ADRENAL INSUFFICIENCY)
ALDOSTERONE
PRINCIPAL CELLS
HYPERKALEMIA

Causes of Hyperkalemia

B.1 Adrenal insufficiency/Hypoaldosteronism cont.

- Drugs:
    1. renin inhibitors
    2. ACE inhibitors
    3. Angiotensin II receptor antagonists
    4. selective aldosterone inhibitors
    5. potassium sparing diuretics

Causes of Hyperkalemia

B.2 Acute kidney injury [AKI]

- In AKI GFR [glomerular filtration rate] is reduced causing majority of Na and water to be reabsorbed before reaching the distal tubule.

- due to the low volume of water remained when reaching the distal tubule the k concentration will be higher the lumen of distal tubule potassium will not be secreted, also the lower Na concentration means less Na/K exchange thus K will not be secreted to the urine and will develop Hyperkalemia.

Causes of Hyperkalemia

C.1Rapid excessive intake

- Like in an iatrogenic error during iv infusion.

Thank you

Diagnosis of Hyperkalemia

Done by : Ali Shawagfeh

- All patients require an ECG and Laboratory studies to confirm the diagnosis and assess the need for urgent treatment .

1) Laboratory Studies

A) **Basic metabolic panel (BMP) :** laboratory test that include the serum consentration of several compunds .

- **Glucose :** if very high consider hyperkalemia secondary to hyperglycemic crisi .

along with water , K+ shift to the extracellular space as a result of hyperglycemic hyperosmolality , resulting in high serum K+ level despite total body K+ deficit . These patients do not require treatment and may in fact require K+ replacement during treatment of hyperglycemia .

Na+ : can be decreased **(** Hyponatremia **)** in adrenal insfficiency , because of decreased aldosterone hormon which increase the reabsorption of Na+ and secreation of K+

Laboratory Studies count

K+ : Repeat to confirm the diagnosis and rule out Pseudohyperkalemia .

Pseudohyperkalemia is a falsely elevated K+ consentration in a laboratory report . Frequently caused by lysis of erthrocytes either during or after the coolection of the blood sample . Due to prolonged use of tourniquet or delayed analysis of the sample . Should be suspected in asymptomatic patient without risk factor .

- **Renal function test :** often shaow a renal impairment .

Hyperkalemia prevalence is up to 50% in patient with CKD .

- **CBC :** Can show hemolytic anemia or Thrombocytosis .

- **Blood gases :** Often show metabolic acidosis .

- **Liver function test :** may be abnormal in hemolysis or tumor lysis syndrome

2) Investigation of underlying causes

A) **Creatine kinase :** Increased in rhabdomyolysis

A) **LDH :** Increased in tumor lysis syndrome or hemolysis

A) **Cortisol :** Can be decreased in addisnons diease

A) **Renin angiotensin aldosterone system :**

Increased aldosterone : pseudohypoaldosteronism , nephropathy due to sickle cell disease , RTA

- Decreased aldosterone with normal or increased renin : hypoaldosteronism due to Addison disease for example

Investigation of underlying causes count

E) Drugs : NSAIDs , ACEI , K+ sparing diuretics , heparin

Treatment Of Hyperkalemia

Done by : Ali Shawagfeh

❖ Three main approaches to the treatment of hyperkalemia .

1) Cardaic membrane stabilization .

1) Intracellular potassium shifting .

1) Enhanced potassium elimination .

1) Cardiac membrane stabilization

. ECG manifestations of hyperkalemia(signs of cardiotoxicity) is a medical emergancy and treated urgently .

. Patients with significant hyperkalemia (K > 6.5 mEq/L) in the absence of ECG changes shold be aggressively managed .

- **first give IV Calcium Salt** (Calcium gluconate , Calcium chloride) .

A) Calcium antagonizes the effect of hyperkalemia at the cellular level through 3 major mechanisms **:**

1: in the setting of hyperkalemia , the resting membrane potential is shifted to less negative value (form -90mV to -80mV) , which in turn move the RMP closer to the normal threshold potential of -75mV , resulting in increased myocyte excitability .

When calcium is given , the threshold potential shifts to a less negative value (from -75mV to -65mV) so that the initial difference between the RMP and threshold potentials of 15mV can be restored .

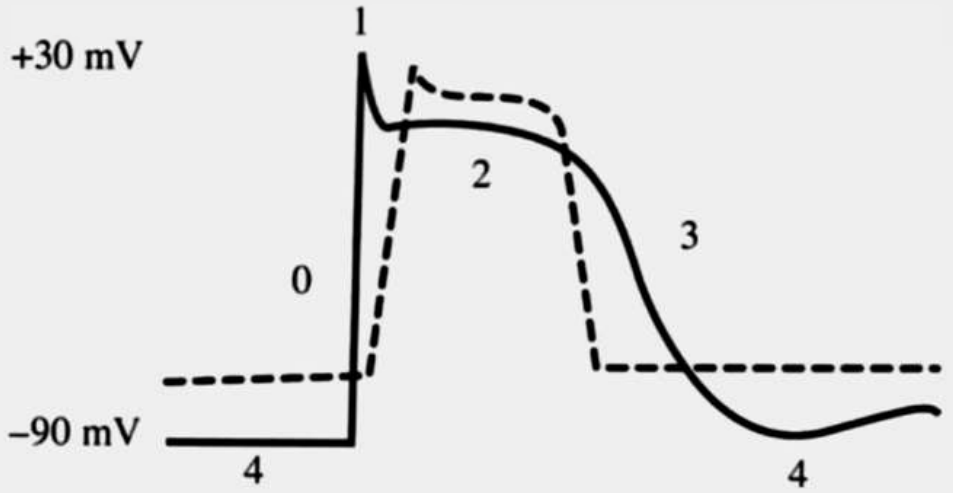


Fig. 3 Illustration of a normal action potential (solid line) and the action potential as seen in the setting of hyperkalemia (interrupted line). The phases of the action potential are labeled on the normal action potential. Note the decrease in both the resting membrane potential and the rate of phase 0 of the action potential (V_{max}) seen in hyperkalemia. Phase 2 and 3 of the action potential have a greater slope in the setting of hyperkalemia compared with the normal action potential.

2 : there is an inverse relationship between RMP and V_{max} (The maximum rate of rise of th action potential , maximum sodium conductance) , at RMP = -90mV we have the maximum amount of Na^+ channels activated during the action potential ; therefore , we will have a steep rise in the phase 0 (normal V_{max})

as the RMP gets higher the number of Na+ channels opened during the action potential decreased slower V_{max}

Now , Calcium goes to specific channels called (NAV 1.5 Channels) and activat it , so in phase 0 the calcium increases the slope back to normal .

3 : in cells with calcium dependent action potentials, such as SA and AV nodal cells , an increase in extracellular calcium concentration will increase the magnitude of the calcium inward current and the Vmax by increasing the electrochemical gradient across the myocyte

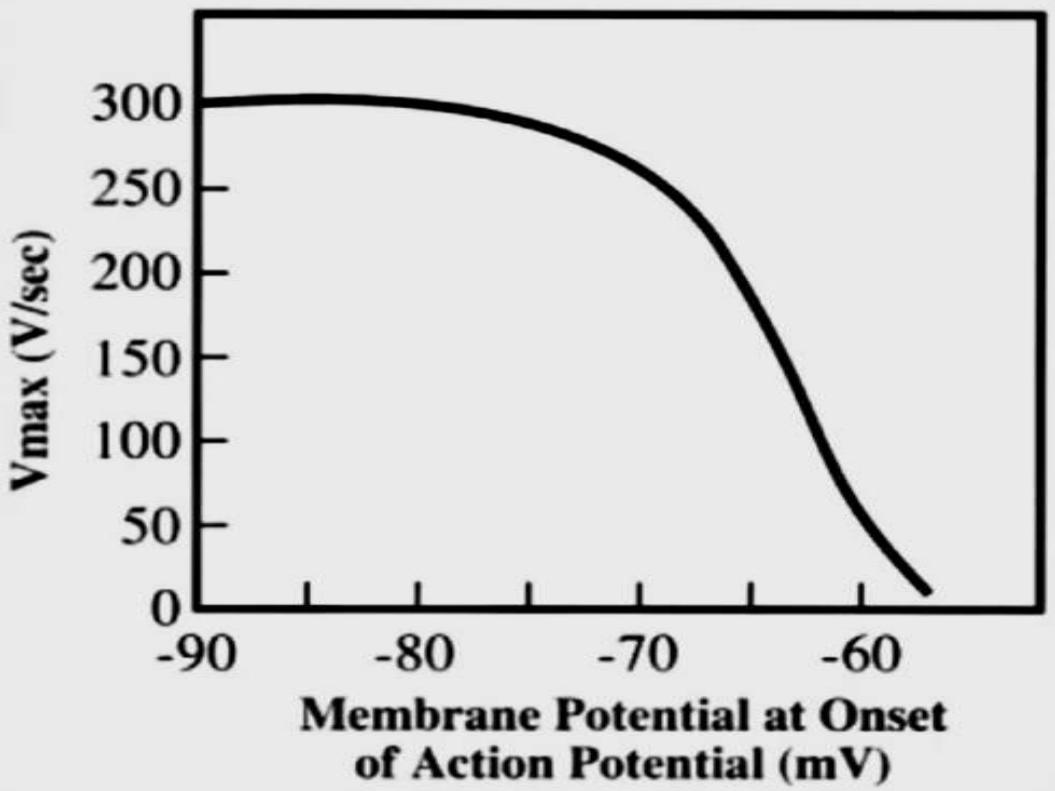


Fig. 4 Curve relating V_{max} to the resting membrane potential at the onset of action potential. As the membrane potential becomes less negative, as in the setting of hyperkalemia, the V_{max} decreases, leading to a depression of conduction through the myocardium.

B) Calcium salt have no influence on serum K^+ level and therefore shold be given with K^+ lowering agent .

C) Use caution in giving calcium to patient on Digoxin , hypercalcemia predisposes the patient to Digoxin toxicity

D) Recommended dose is 10mL of 10% calcium gluconate , infused IV over 2--3 min with cardiac monitoring , the effect of infusion start after 1--3 min and lastes for 30 – 60 min .

E) Dose shold be repeted if there is no change in ECG finding and recur after intial improvement .


NDC 63323-311-19
31119
CALCIUM GLUCONATE
INJECTION, USP
10%
(100 mg/mL)
For Direct Slow IV or IV Additive Use Only
Rx only
10 mL Single Dose Vial
Preservative Free


NDC 0517-6710-01
10% Calcium Chloride
Injection, USP
1 Gram/10mL
(100 mg/mL)
For Intravenous Use Only
Sterile, nonpyrogenic
10 mL Single-dose vial
Rx only
AMERICAN REGENT, INC.
SHIRLEY, NY 11967

2) Intracellular potasium shifting

Inssulin and glucose

A) Insulin shifts K+ into cells by stimulating the activity of Na+/H+ antiporter on cell membrane , promoting the entry of NA+ into cells which leads to activation of the NA+/K+ PUMP causing an electrogenic influx of K+

B) Glucose alone will stimulate insulin secretion from B-cells in the pancreas , but exogenus insulin is more rapid . Give both to prevent hypoglycemia .

C) In hyperkalemic patients with glucose consentration > 200 mg/dL , insulin shold be given without glucose with blood glucose monitering .

D) Combined treatment with B2 agonist provid a synergistic effect with insulin lowering plasma K+ , the B2 agonist used is albuterol . B2 AGONIS NOT EVECTIVE AS MNONTHERAPY .

Sodium bicarbonate

A) Causes acute metabolic alkalosis , which can lead to increased intracellular K+ uptake in excahnge for H+ , thereby lowering serum K+ .

B) Consider in patients with metabolic acidosis .

3) Enhanced Potasium elimination

Cation exchange resin

A) These drugs release Na+ or Ca+ ion in the gut which are exchanged for K+ , therby enhancing enteral K+elimination .

B) Used to treat hyperkalemia by accelerating K+ loss through the gut , especially in the context of poor urin output.

C) Onset of action after 2 hours and the peak effect after 6 hours . So, these drugs used in nonurgent lowering of K+

. Sodium polystyrene sulfonat (SPS) : falling out of favor due to its addverse effect

. Sodium zirconium cyclosilicate

Loop diuretics and Thiazide

A) Can be used to reduced plasma K+ consentration in hpervolemic patients with sufficient renal function for diuretic response

B) Usually combined with IV saline or isotonic biacarbonate to achieve or maintain euvolemia

Hemodialysis and Pertonial dialysis

A) Is the most effective and reliable method to reduce plasma K+ . It is not a first line option because of its invasive nature and adverse effect .

B) it is the treatment of choice for patients already receiving regular renal replacement therapy .

Therapeutic approach to hyperkalemia [2][9][14][21]		
Treatment strategy	**Acute hyperkalemia** [24]	**Chronic hyperkalemia**
Cardiac membrane stabilization	• If signs of cardiotoxicity: IV calcium chloride or calcium gluconate	• Not routinely required
Intracellular K^+ shifting	• **Short-acting insulin with glucose** • Consider inhaled SABAs.	

Enhanced K^+ elimination	• In refractory hyperkalemia:
	  ◦ Oliguria/ESRD: hemodialysis
	  ◦ Hypervolemia: Consider diuretics.
	  ◦ Metabolic acidosis: Consider IV 8.4% sodium bicarbonate.
	• Consider cation exchange medications
		• Consider one of the following:
		  ◦ Cation exchange medications
		  ◦ Diuretics

Reduced K^+ intake	• Low-potassium diet [10][25]	
Treatment of underlying cause	• Medication review: Adjust medications that affect K^+ metabolism. • Identify and treat other reversible conditions: e.g., primary adrenal insufficiency, AKI, tumor lysis syndrome.	

- Thank you