: Hyperkalemia :∴ decreas excitability and inhibit contractions
: Hypokalemia :∴ increase excitability and increase contractions

POTASSIUM

- Potassium is the major intracellular cation in the body with a concentration 20 times greater inside the cells than outside
- Many cellular functions requires that the body maintains a low ECF concentration of K. As a result, only 2% of the body's total potassium circulates in the plasma
- Function of potassium in the body include
  1. Neuromuscular excitability
  2. Contraction of the heart
  3. ICF volume
  4. Hydrogen ion concentration

= لما يقول الشخص عنده hypokalemia وأعطه IV potassium لازم أركبله ECG عشان ما يصير عنده hyperkalemia وارجع لنفس المشكلة .

Hypokalemia
⇓
alkalosis (↓ H^+ in blood)

POTASSIUM

Hyperkalemia
⇓
acidosis (↑ H^+ in blood)

- The potassium ion concentration has a major effect on skeletal and cardiac muscles. A lower than normal difference increases cell excitability leading to muscle weakness.
- Severe hypokalemia can cause muscle excitability which may lead to paralysis or fatal cardiac arrhythmia
- Hypokalemia decreases cell excitability resulting in an arrhythmia or paralysis
- the heart may cease to contract in extreme case of hypokalemia or hyperkalemia
- Potassium concentration affects hydrogen ion concentration in the blood. In hypokalemia, when potassium ion is lost from the blood, sodium and hydrogen ions move to into the cells. The hydrogen ion concentration decreases in ECF resulting to alkalosis

= acidity of blood relate with potassium level due to H^+/K^+ pump.

1. when K^+ increase in blood, the pump enters K^+ into the cell and exits H^+, Na^+ out of the cell. → ↑ H^+ في الدم → acidosis.
2. when K^+ decrease in blood, the pump enters H^+, Na^+ into the cell and exits K^+ out of the cell. → ↓ H^+ في الدم ← alkalosis

FACTORS AFFECT K LEVEL IN ECF

- Three factors that influence the distribution of potassium between cells and ECF are:
- (1) **Potassium loss** frequently occurs whenever the NaK ATPase pump is inhibited by conditions such as:
  - (1) hypoxia (hypokalemia)
  - (2) hypomagnesemia (hypo)
  - (3) or digoxin overdose
- (2) **Insulin** promotes acute entry of K ions into skeletal muscle and liver by increasing NaK ATPase activity
- (3) **Catecholeamines** such as epinephrine (β2-stimulator), promotes cellular entry of K whereas propranolol (β-blocker) impairs cellular entry of potassium .
- With preexisting condition such as **dietary deficiency** (or excess) can enhance the degree of hypokalemia (or hyperkalemia)but rarely the primary cause.

- digoxin antgonise the K^+ entering to cell.
- dietary deficiency or excess are not the primary cause of hypokalemia or hyperkalemia but affect the degree of the condition

Factors affect K level in ECF

- **Exercise:** potassium is released from cells leading to increase by 0.3-1.2 mmol/L with mild to moderate exercise and 2-3 mmol/l with exhaustive exercise (reversed after several minutes of rest).
- **Hyperosmolality:** like in uncontrolled diabetes mellitus, causes water to diffuse from the cells carrying potassium ions leading to gradual depletion of potassium if kidney function is normal.
- **Cellular breakdown:** cellular breakdown releases K into the ECF like in severe trauma, tumor lysis syndrome and massive blood transfusion.

- K^+ normal range = (3.4 - 5.0)

HYPOKALEMIA → $K^+ < 3.0$ mmol/L

- ❑ Hypokalemia is a plasma potassium concentration below the lower limit of the reference range
- ❑ Hypokalemia can occur with GI or urinary loss of potassium or with increased cellular uptake of K
- ❑ Common causes of hypokalemia like:
- ❑ GI loss occurs when GI fluid is lost through vomiting, diarrhea, gastric suction or discharge from intestinal fistula
- ❑ Increased potassium loss in the stool also occurs in certain tumors, malabsorption, cancer therapy and large doses of laxatives
- ❑ Renal loss of K can result from kidney disorders such as potassium losing nephritis and renal tubular acidosis (RTA). In RTA, as tubular excretion of H+ decreases, K excretion increases

= Cancer therapy → cell damage in GI.

RTA ⇒ ↑ H^+ in blood ↓ K^+ in blood.

- aldosteron → reabsorb Na^+ and secreat K^+.

HYPOKALEMIA, COMMON CAUSES

- **Hyperaldosteronism:** lead to hypokalemia and alkalosis
- **Magnesium deficiency:** inhibits NaK ATPase and enhances secretion of aldosterone (treated by Mg and K supplement) ⇒ hypokalemia in blood.
- **Alkalemia and insulin**: increase the cellular uptake of K → ↓ K^+ in blood.
- **Drug induced:** thiazide diuretics and corticosteroids are the most important, carbanoxolone has mineralocorticoid activity
- **Alkalosis:** may cause a shift of potassium from the ECF to the ICF (0.1 increase in pH leads to 0.4 mmol/l decrease in potassium)

SYMPTOMS OF HYPOKALEMIA

- Mild hypokalemia (3-3.4 mmol/L) is asymptomatic
- Weakness, fatigue and constipation at K< 3 mmol/l
- Muscle weakness and paralysis that interfere with breathing
- Dangerous for patients with cardiovascular disease as it may cause arrhythmia leading to sudden death in some patients

= ECG changes

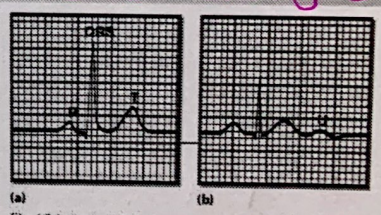

Fig 12.1 **Typical ECG changes associated with hypokalaemia. (a)** Patient with hypokalaemia; note flattened T-wave. U-waves are prominent

TREATMENT

- Potassium salt are unpleasant to take orally and are usually given prophylactically in an enteric coating
- Severe potassium depletion often has to be treated by intravenous potassium
- Intravenous potassium should not be given faster than 20 mmol/hour except in extreme cases and under ECG monitoring
- Mild chronic hypokalemia can be treated by diet rich K (dried fruits, nuts, banana and orange juice)

HYPERKALEMIA $K^+ > 6.0$ mmol/L

- Hyperkalemia is the commonest and most serious electrolyte emergency encountered in clinical practice.

- Hyperkalemia causes muscle weakness that may be preceded by paraesthesiae. However, the first manifestation may be cardiac arrest.

- Above 7.0 mmol/l there is a serious risk of cardiac arrest. However, the ECG changes in hyperkalaemia may mimic other conditions such as myocardial infarction, thus, it is important to check the serum potassium concentration in patients after cardiac arrest

CAUSES OF HYPERKALEMIA

1. ❑ **Renal failure.** The kidneys may not be able to excrete when the glomerular filtration rate is very low. The acidosis associated with renal failure contributes to the problem.

2. ❑ **Mineralocorticoid:** this the most frequently seen in Addison's disease (↓ aldosterone level) or in patient receiving aldosterone antagonists. In these patients, there is an increase in total body potassium

3. ❑ **Acidosis:** Hyperkalemia results from the redistribution of potassium from the intracellular to the extracellular fluid space

CAUSES OF HYPERKALEMIA

(4)

- **Potassium release from damaged cells:** because of the very high potassium concentration inside cells, cell damage can give rise to a very high serum potassium as occurs in trauma and malignancy
  ← continous cell damage.

(5)

- **Diabetes mellitus:** fast shift of potassium from cells to the blood due to insulin deficiency in addition to hyperosmolality that pulls water to outside the cells

(6)

- **Various drugs:** specially in patients with renal insufficiency or diabetes mellitus as captopril (ACEI), NSAID, digoxin, spironolactone, cyclosporine and heparin therapy

(7)

- **Warming after surgery** leads to release of K from cells, hypothermia may cause hypokalemia

لما انخفض درجة حرارة الجسم

PSEUDOHYPERKALEMIA

له يصير cell lysis بعد سحب العينة من الجسم .

- Refers to elevation in the measured potassium concentration potassium movement out of cells during or after the drawing of the blood specimen.
- The commonest cause is hemolysis. This can occur due to mechanical trauma during venopuncture. Hemolysis is characterized by release of potassium from red blood cells.
- A small amount of potassium is released from white blood cells and platelets during normal clotting
- In patient with grossly elevated white cells and platelets due to hematological malignancies, the amount of potassium released is much greater
- Pseudohyperkalemia should be suspected when there is no apparent cause for hyperkalemia and there are no ECG changes reflecting altered cardiac muscle contractility

له بعد نقل وصدات الدم .

له الناس اللي عندهم سرطانات قديه عندهم نسبة k بعد سحب الدم بسبب العدد الكبير لخلايا الدم البيض

* مهم ← تميز بين pseudohyperkalemia عن real hyperkalemia من خلال ECG .

SYMPTOMS OF HYPERKALEMIA

- Muscle weakness at K conc of 8 mmol/l
- Tingling, numbness and mental confusion
- Cardiac arrhythmia and cardiac arrest at conc of 6-7 mmol/l which alter ECG
- Fatal cardiac arrest at conc > 10 mmol/l

incompatable with life.

TREATMENT OF HYPERKALEMIA

- Treatment should be started if K > 6-6.5 or if ECG changes occur
- An infusion of calcium gluconate may be given to potential of myocardial cells reduce threshold
- the commonest form of treatment of acute hyperkalemia is the infusion of sodium carbonate, insulin and glucose to move potassium ions into cells
- K can be removed by loop diuretics in good renal function
- Na polystyrene sulphonate enema which binds K secreted in the colon
- Dialysis is frequently necessary to treat severe hyperkalemia

COLLECTION OF THE SAMPLE

- Simultaneous collection and processing of serum and plasma specimens may help, the anticoagulant in plasma specimens prevents clotting from occurring.
- Care must be taken during drawing of blood as high platelet counts or when tourniquet is left for long time on the arm may increase the conc of K
- Whole blood samples should be stored at room temperature (not iced) or rapid centrifugation of the sample to remove cells. عشان ما يصير cell lysis
- Specimen used may include serum, plasma, whole blood or 24-hr-urine sample
- Reference ranges of potassium are:
  - Serum and plasma 3.4-5.0 mmol/l
  - Urine (24-hr) 25-125 mmol/day