ELECTROLYTE -IV

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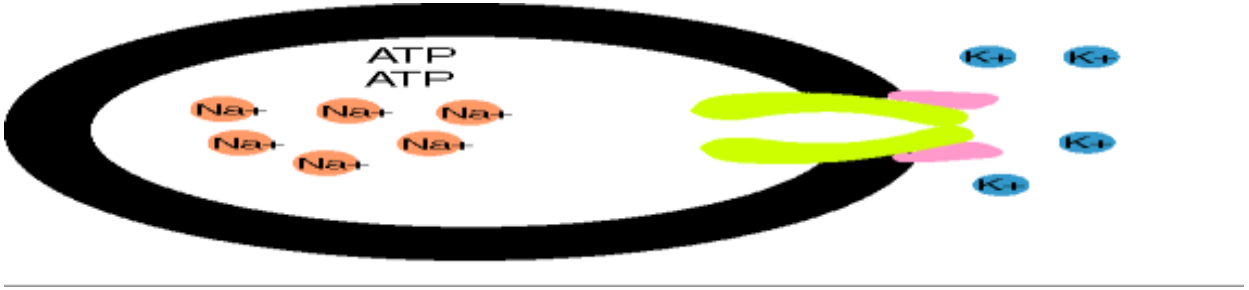
Sodium

- Sodium is the most abundant cation in the ECF, representing 90% of all extracellular cations, and largely determines the osmolality of the plasma.
- The Na-K ATPase ion pump moves three sodium ions out of the cell in exchange for two potassium ions moving into the cell as ATP is converted to ADP.



- Because water follows electrolytes across cell membranes, the continual removal of sodium from the cell prevents osmotic rupture of the cell by also drawing water from the cell.

- **Normal serum range: 135-150 mmol/L**
- **Panic levels in serum are 110 mmol/L**

Regulation of body sodium

- The plasma sodium concentration depends greatly on the intake and excretion of water and, to lesser degree, the renal regulation of Na.

Three processes are of primary importance:

(1) Intake of water in response to thirst, stimulated or suppressed by plasma osmolality.

(2) Excretion of water, largely affected by ADH release in response to changes in either blood volume or osmolality.

(3) Blood volume status, which affects sodium excretion through aldosterone, angiotensin II, and ANP (atrial natriuretic peptide).

Hyponatremia

Defined as a serum/plasma level <135 mmol/L.

Levels below 130 mmol/L are clinically significant.

Causes of hyponatremia:

I- Depletional hyponatremia (increased sodium Loss):

-Renal lose: Diuretic, hypoaldosteronism & addisons dis.

-Non renal cause:

- GIT: Prolonged vomiting, diarrhea
- Skin: burns & trauma

II- Dilutional hyponatremia:

Inappropriate ADH secretion (SIADA), generalized edema (CHF, cirrhosis & nephrotic syndrome), hyperglecemia & administration of hypotonic fluids

Symptoms *of hyponatremia.*

- Symptoms depend on the serum level.
- Between 125 and 130 mmol/L, symptoms are primarily gastrointestinal including nausea and vomiting.
- Below 125 mmol/L more severe symptoms are seen, neurological dysfunction due to brain edema muscular weakness, headache, seizures, coma, and respiratory depression.
- This is a result of the shift of water into brain cells, with concurrent expansion of these cells.

Hypernatremia

- Hypernatremia is increased serum sodium concentration above 150 mmol/L.

- Hypernatremia is less commonly seen in hospitalized patients than hyponatremia.

Causes of hypernatremia

- Excess Water Loss

  Prolonged diarrhea

  Profuse sweating

  Diabetes insipidus

- Increased Intake

  Hyperaldosteronism

  Administration of hypertonic fluids containing sodium

- Decreased Water Intake

  Older persons

  Infants

***Symptoms of hypernatremia*.**

- This is a result of the shift of water out of brain cells, with concurrent shrinkage of these cells.
- These symptoms due to neurological dysfunction
- Serum sodium of more than 160 mmol/L is associated with a mortality rate of 60-75%.

Potassium

Potassium is the major intracellular cation in the body, with a concentration 20 times greater inside the cells than outside. Only 2% of the body's total potassium circulates in the plasma.

Normal range: 3.4-5.0 mmol/L

Panic levels in serum are <2.5 & >6.5 mmol/L

Functions of potassium

1- Regulation of neuromuscular excitability,.

2- A major effect on the contraction of skeletal and cardiac muscles.

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Factors influence the distribution of potassium between cells and ECF :

(1) Potassium loss occurs when the Na K ATPase pump is inhibited by conditions such as hypoxia.

(2) Insulin promotes acute entry of K ions into skeletal muscle and liver by increasing Na K ATPase activity.

(3) Cathecholamines, as epinephrine, promote cellular entry of K, whereas propranolol impairs cellular entry of K.

Hypokalemia.

Causes of hypokalemia

1-GIT Loss

Vomiting

Diarrhea

2- Shift (increase cellular uptake)

Insulin overdose

3- Decreased Intake

4- Renal Loss

Diuretics—thiazides, mineralocorticoids

Nephritis

Symptoms of hypokalemia

- Mild hypokalemia (3.0-3.4 mmol/L) is usually asymptomatic.
- When plasma potassium decreases below 3 mmol/L (weakness, fatigue, and constipation) often become apparent .
- Muscle weakness or paralysis, which can interfere with breathing.
- Increased risk of arrhythmia, which may cause sudden death in certain patients, especially those with cardiovascular disorders.

Hyperkalemia

- **In diabetes mellitus**, insulin deficiency promotes cellular loss of K. Hyperglycemia also contributes by producing a hyperosmolar plasma that pulls water and K from cells, promoting further loss of K into the plasma.

- **In metabolic acidosis,** as excess H^+ moves intracellularly to be buffered, K leaves the cell to maintain electroneutrality. Plasma K increases by 0.2-1.7 mmol/L for each 0.1 unit reduction of pH.

Causes of hyperkalemia

Decreased Renal Excretion

Acute or chronic renal failure (GFR, <20 mL/minute)

Diuretics (potassium sparing duiretics)

Cellular Shift

Acidosis

Muscle/tissue damage potassium is released from cells during exercise

Hemolysis causing release from red cells

Exchange transfusion using stored blood

Increased Intake

Oral or IV potassium replacement therapy

Symptoms of hyperkalemia

- Plasma potassium concentrations of 6-7 mmol/L may produce weakness, parathesia, arrhythmias and concentrations more than 8 mmol/L may cause fatal cardiac arrest.

- First, the coagulation process releases K from platelets, so that serum K may be 0.1-0.5 mmol/L higher than plasma K concentrations. This is avoided by using a heparinized tube to prevent clotting of the specimen.

- If the patient has (thrombocytosis), serum potassium may be further elevated.
- Avoid hemolysis

Chloride

- Chloride is the major extracellular anion.
- Its metabolism is closely linked to that of sodium
- Its precise function in the body is not well understood; however, it is involved in maintaining osmolality, blood volume, and electric neutrality.
- Chloride ions shift secondarily to a movement of sodium or bicarbonate ions.

Normal range: 98-107 mmol/L

Chloride disorders are due to the same causes that disturb sodium level because it passively follow Na

Hyperchloremia

may occur when there is an excess loss of bicarbonate ion as

1- Dehydration
2- Metabolic acidosis.
3- Renal tubular acidosis

Hypochloremia

occur with excessive loss of chloride from:

1- GIT losses: prolonged vomiting,
2- Renal losses: diuretics, metabolic alkalosis
3- Burns

Determination of Chloride, Na, K

Specimen.

-Serum or plasma may be used, with lithium heparin being the anticoagulant of choice.

-The specimen of choice in urine analyses is 24-hour collection because of the large diurnal variation.

Methods.

- Ion selective electrodes (ISE). It is the most routinely used method in clinical laboratories.

Bicarbonate

- Bicarbonate is the second most abundant anion in the ECF.
- Because $HC0_3$ composes the largest fraction of total $C0_2$, total $C0_2$ measurement is indicative of $HC0_3$ measurement.
- Bicarbonate is the major component of the buffering system in the blood.
- It functions as transport form of $C0_2$ produced from metabolic processes in tissues and delivered to lungs for exhalation. Carbonic anhydrase in red blood cells converts $C0_2$ & H_2O to carbonic acid which dissociates to H & HCO_3
- Bicarbonate diffuses out of the cell in exchange for chloride to maintain ionic charge neutrality within the cell (chloride shift). This process converts potentially toxic $C0_2$ in the plasma to an effective buffer bicarbonate.

Anion gap

- It is the difference in the measured cations (positively charged ions) and the measured anions (negatively charged ions).
- **The calculation:**

AG = $(Na^+ + K^+) - (Cl^- + HC0_3^-)$ It has a reference range of 10-20 mmol/L.

- There is never a "gap" between total cationic charges and anionic charges, plasma is electro-neutral (uncharged).

Anion gap

- It represents the concentration of all the unmeasured anions in the plasma.
- Normal AG which represent the negatively charged proteins (major part) ,phosphate, sulphate & organic acids in normal serum.
- If the anion gap increased the likely source is one of unmeasured anions The acid anions (eg lactic, acetoacetic, B hydroxybutyric, sulphate & phosphate,) produced during a metabolic acidosis.

- In metabolic acidosis the net effect is a decrease in the concentration of measured anions (HCO_3) and an increase in the concentration of unmeasured anions (the acid anions) so the anion gap increases.

- Rarely AG less than normal because of increase unmeasured cations as IgG in parapoteinemia, hypercalcemia & hypermagnesemia

***An elevated anion gap* may be caused by:**

1. Renal failure, which leads to PO_4^- and $S0_4^{2-}$ retention;
2. ketoacidosis, as seen in cases of starvation or diabetes; methanol, ethanol, ethylene glycol poisoning, or salicylate; lactic acidosis;
3. hypernatremia
4. Instrument error (abnormal anion gaps in serum from healthy persons may indicate an instrument problem).

Low *anion gap* values are rare but may be seen with hypoalbuminemia (decrease in unmeasured anions) or severe hypercalcemia (increase in unmeasured cations).