1.4.3 ELECTROLYTE

1.4.3.1 Sodium (natrium) Na⁺:
Sodium is the major positive ion (cation) in fluid outside of cells. The chemical notation for sodium is Na⁺. Excess sodium (such as that obtained from dietary sources) is excreted in the urine. Sodium regulates the total amount of water in the body and the transmission of sodium into and out of individual cells also plays a role in critical body functions\[34\]. Many processes in the body, especially in the brain, nervous system, and muscles, require electrical signals for communication. The movement of sodium is critical in generation of these electrical signals. Too much or too little sodium therefore can cause cells to malfunction, and extremes in the blood sodium levels.\[34][35]\n
1.4.3.2 Sodium reabsorption:
There is active reabsorption of sodium (Na⁺) in the proximal tubules up to about 75% of the Na⁺ in the filtrate, further reabsorption occurs in the loops of Henle ONLY ABOUT 10% reaches the distal tubules, in the distal tubules and collecting duct, fine regulation of the sodium reabsorption is occur under influence of aldosteron. According to the needs of homeostasis in the absence of the hormone most of the remaining sodium is lost in the urine when aldosteron is secreted in large amounts, almost all of sodium is reabsorbed \[34]\n
The kidney reabsorbs quite a large amount of sodium each day, the amount which passes in the filtrate is about 6259mmol/day the transport of sodium in the distal tubules in an active process, which occur against concentration gradient from the tubular cell to the tubular fluid. This net movement of sodium creates negative electrical potential inside the tubular lumen. The active sodium transport also result in the pumping of potassium into the cell.\[36]\n
1.4.3.3 Three major hormones are involved in regulating sodium and water balance in the body at the level of the kidney:-

- ADH (antidiuretic hormone) from the posterior pituitary acts on the kidney to promote water reabsorption, thus preventing its loss in the urine.
- Aldosterone from the adrenal gland acts on the kidney to promote sodium reabsorption, thus preventing its loss in the urine.

- ANH (a trial natriuretic hormone) from the atrium of the heart acts on the kidney to promote sodium excretion so that it is excreted in the urine.

1.4.3.4 Clinical significant:

1.4.3.4.1 Hypernatremia is caused by renal and non renal disorders. A common nonrenal cause is hypotonic dehydration from severe diarrhea, extensive burns, or excessive sweating without proper fluid replacement. Infants, the elderly and other patients not able to ingest sufficient amounts of water, and who are not properly hydrated, will also experience hypernatremia. Renal loss of water, such as in nephrogenic diabetes insipidus, can also cause hypernatremia. Serum osmolality and urinary sodium levels can help to differentiate renal loss of water versus nonrenal causes. [37]

1.4.3.4.2 Hyponatremia is caused by renal and nonrenal causes also. Salt-losing renal nephritis, renal tubular acidosis, or syndrome of inappropriate antidiuretic hormone secretion (SIADH) are common causes of renal loss of sodium and may be evaluated by testing for the presence of excess urinary sodium and hyperosmolar urine. Certain diuretics, such as thiazine, can cause renal loss of sodium. Increased urine sodium levels usually indicate sodium loss.[34] Chronic renal failure can cause water overload due to inability to regulate water and results in hyponatremia, while nephritic syndrome can cause fluid imbalances and edema with resulting hyponatremia. Urine sodium levels are usually normal or decreased in hyponatremia due to edema. Nonrenal causes of hyponatremia include psychogenic water overload, cellular shift changes from acidosis, and edema secondary to cirrhosis or congestive heart failu.[37]
1.4.4.1 Potassium:

Potassium is the major positive ion (cation) found inside of cells. The chemical notation for potassium is K+. The proper level of potassium is essential for normal cell function. Among the many functions of potassium in the body are regulation of the heartbeat and the function of the muscles. A seriously abnormal increase in potassium (hyperkalemia) or decrease in potassium (hypokalemia) can profoundly affect the nervous system and increases the chance of irregular heartbeats (arrhythmias), which, when extreme, can be fatal[38]

1.4.4.2 Potassium reabsorption

Potassium reabsorption occurs by two mechanisms:

1. Active reabsorption in the proximal tubule almost completely conserves potassium.
2. Exchange with sodium is stimulated by aldosteron, hydrogen competes with potassium for this exchange, the amount of potassium secreted is direct related to aldosteron secretion and sodium reabsorption.

Small increase of serum potassium directly stimulated the adrenal cortex to release aldosteron and thus (K+) secretion enhance in the absence of aldosteron (K+) secretion stops and reabsorption occur. This is show that (K+) reabsorption taking place all the time but it is usually masked by the normally greater amount secreted . [38]

1.4.4.3 Clinical significant:

1.4.4.3.1 Hyperkalemia

May be caused by decreased renal excretion in acute or chronic renal failure, certain diuretics, or hypoaldosteronism or hypocortisolism. Hyperkalemia may also be caused by ion shift, such as the ion shift that is seen in cases of diabetic ketoacidosis or other metabolic acidosis, leukemia, excessive muscle activity, or hemolysis. Finally, hyperkalemia is associated with iatrogenic causes of excessive intravenous or oral therapy.[38]

1.4.4.3.2 Hypokalemia

is caused by renal loss such as renal tubular acidosis, hyperaldosteronism,
Hypercortisolism and certain diuretics. Potassium can also be decreased due to gastrointestinal dietary deficit or loss from severe vomiting, diarrhea, nasogastric suctioning, laxatives, and malabsorption. A cellular shift in cases of insulin overdose and alkalosis can also cause hypokalemia\textsuperscript{[39][40]}