Summary

Potassium is the major intracellular cation. It is an important electrolyte that affects the body homeostasis. It is a main player in endless biological processes that are responsible for optimal integrity of CNS, GIT, urinary tract and cardiovascular system.

As regarding the beneficial effects of potassium, it was reported that potassium intake participates in regulating the blood pressure in healthy and hypertensive population. Potassium intake also associated with reduction in mortality rates in stroke patients.

Internal (extra- renal) potassium homeostasis is affected by active and passive transport mechanisms. Na + - K+ ATPase, insulin, catecholamines and mineralocorticoids are the main limbs of the active mechanisms. Passive transport mechanisms such as acid - base balance, body fluid tonicity, and active exercise share in the regulation of internal potassium balance. Renal regulation is a crucial in potassium homeostasis. This is due to the extreme capability of the kidney to alter the potassium excretion over a wide range in cases of potassium depletion or potassium overloading. Many peritubular factors could affect the renal handling of potassium such as dietary potassium intake, plasma potassium concentration, acid - base balance, different hormones (e.g. aldosterone and ADH). Flow rate, transepithelial voltage and chloride concentration are all luminal factors that share in controlling the renal handling of potassium.

Over all, potassium excretion is strictly obedient to a rhythmic or circadian pattern that could be attributed to an oscillation in the hypothalamic efferent impulses. This circadian rhythm is of defending value against impairment of potassium homeostasis.

Hypokalaemia (serum potassium < 3.5 mEq/L) is reported in more than 20% of hospitalized patients. Transcellular shift or potassium depletion are the common causes of hypokalaemia.Potassium depletion is caused by inadequate intake and increase potassium

losses. Hypokalaemia has neuromuscular, renal, gastrointestinal and cardiovascular manifestations which are of special clinical importance. Neuromuscular manifestations associating hypokalaemia should be put in mind especially in ICU patients, as a definite cause of bad weaning index in ventilated patient (serum potassium < 2 mEq/L is accompanied with an ascending pattern of paralysis).

Correction of hypokalaemia should be managed wisely over days, unless there is cardiovascular or neuromuscular life- threatening indications. Oral route of correction should be preferred for its safety and reliability. Many regimens for intravenous correction are suggested. The guide for selection is the need for a rapid and rational get rid of life – threatening arrhymias or muscle paralysis.

Hyperkalaemia (serum potassium > 5.5 mEq/L) is reported in 1.4-10% of hospitalized patients. Exclusion of pseudohyperkalaemia is a must for futher diagnosis of hyperkalaemia. Increased potassium load, transcellular shift, and decreased potassium excretion are the main causes of true hyperkalaemia. Cardiovascular manifestations should be managed effectively for the serious hazards of hyperkalaemia. Hyperkalaemia with ECG –changes is a true medical emergency.

Acute hyperkalaemia may be associated with generalized muscle weakness that is may proceed to paralysis. Management of hyperkalaemia aims to deal with the actute increase of serum potassium level and then control the chronic hyperkalaemic state. The principals of management of acute hyperkalaemia depend on: direct antagonism of effects of hyperkalaemia, redistribution of potassium from the extracellular to the intracellular compartments and removal of potassium from the body.

There was a debate about whether hyperkalaemia or hypokalaemia is more common in severe trauma patient. Some authors reported that hypokalaemia is more common in this category of patients and noted in their study that the most acceptable

cause of hypokalaemia is transcellular shift of potassium. Others theorized that epinephrine might play an important role in shock and trauma patient who is suffering from hypokalaemia. Posttraumatic stress response, as a corner stone element, could explain the posttraumatic hypokalaemia. After stimulation, hormonal and inflammatory (cytokines and acute phase proteins) mediators affect the internal metabolism universally all over the body. The net result, indeed, is the transcellular shift of potassium and hypokalaemia.

Despite a reduction in the total body potassium level, diabetic ketoacidosis is marked by a normal or even increased serum potassium concentration. Hyperglycemia increases the serum tonicity, causes a depletion of the intracellular fluid space; this, in turn, increases the intracellular potassium concentration, potassium ion then moves out of the cell.

In acute renal failure, the oliguric phase is associated with cellular dehydration resulting from a shift of water out of cells and increase in extracellular fluid volume. That leads to a shift of potassium out of cells causing hyperkalaemia with a decrease in total body potassium.

In chronic renal failure patient, there is a persistent state of potassium retention secondary to the reduction of functioning nephron units and faliure of certain adaptive renal and extrarenal mechanisms that aim hardly to control the increase in serum potassium and protect the body system from the hazards of hyperkalaemia.

Rhabdomyolysis is a disorder that involves rapid breakdown of skeletal muscle, due to an injury. As a result the components of the skeletal muscle cells (myoglobin, potassium and phosphate) are released into the blood stream, with increased myoglobin levels in the urine which can damage the kidneys.

Hyperkalaemia found in burned patients is seemed to be due to extra cellular water loss and hemoconcentration and the shock state leads to transport of K ions from the cells to tha extra cellular space.

The chronic increased work of breathing in COPD results in anaerobic metabolism of the ventilatory muscles and overproduction of lactic acid; the eventually coexisting profound tissue hypoxia; and the decreased lactate clearance by the liver because of hypoperfusion. Metabolic acidosis results in hyperkalaemia.

Respiratory alkalosis plays a minor role in the hypokalemic state found in severe asthma. The major causes are diuretics and β2- agonists which are taken by patients with cardiovascular diseases which accompany severe asthma.

Excessive use of diuretics and β2-agonists decreases serum levels of potassium, magnesium, and phosphate.

Preoperative hypokalaemia predisposes to potentially life-threatening arrhythmias during general anesthesia. Arrhythmias are related to severity of heart disease, chronic digoxin therapy, diuretic therapy and preoperative potassium levels, especially when potassium levels < 3.0mEq/L. All these data recommend postponing of the operations to correct plasma potassium levels.

Following open heart surgery, a decreased potassium concentration is usually found and this may be caused by receiving preoperative therapy with digitalis and diruretics. In addition to the diluted perfusate, however, other factors such as respiratory alkalosis, and steady postoperative urinary excretion are contributing factors to hypokalemia. Cardiac arrhythmias are prone to occur which may terminate in irreversible myocardial dysfunction.

Microvascular endothelial dysfunction and altered vascular reactivity often occur after ischemic arrest and cardiopulmonary bypass, although cardioplegia has been routinely used for the protection of the myocardium against ischemic injury during cardiac surgery. Recent studies found that KCa channel activation has agreat role in cardioplegic arrest (CP) and reperfusion-related microvascular dysfunction in human coronary resistance arterioles and regulatory properties in this vascular bed.

Studies have shown that altering the intracellular concentration of potassium has

a dramatic effect on the apoptotic process. Enhancing the efflux of potassium has been shown to augment apoptosis. In a separate study, apoptotic nuclease and caspase enzymes activity was shown in vitro to be prevented by increasing concentrations of potassium.