Introduction

Disturbances in potassium homeostasis presenting as low or high serum potassium are common, especially among hospitalized patients.Given the fact that untreated hypokalaemia or hyperkalaemia is associated with high morbidity and mortality, it is critical to recognize and treat these disorders promptly. **(Brown et al., 2005).**

Potassium is the most abundant cation in the body. It is predominantly restricted to the intracellular space, such that only 2% is located extracellulary and the remaining 98% is in the intracellular compartment. The ratio of intracellular to excellular potassium (Ki/Ke) is the major determinant of resting membrane potential, and is regulated primarily by the sodium-potassium ATPase pump located on the plasma membrane of most cells. Although extracellular potassium accounts for only 2% of total body potassium, it has a major effect on the ratio of Ki/Ke and through that on the resting membrane potential. As a result, serum potassium is normally regulated around the narrow range of 3.5-5.5 mmol/l. **(Defronzo et al., 2001).**

Hypokalaemia is probably the most common electrolyte abnormality in hospitalised patients. It is usually defined as a serum potassium of less than 3.5 mmol/l. Hypokalaemia can result from increased loss, transcellular shift, or decreased intake of potassium. Increased potassium loss through the kidney or gastrointestinal tract is the most common cause of hypokalaemia.

Less frequently, hypokalaemia can occure as a result of shift of potassium from the extracellular into cells. Rarely, hypokalaemia can result from decrease intake of potassium. **(Amlal et al., 2006).**

Hyperkalaemia is defined as serum potassium greater than 5.0 m mol/l. True hyperkalaemia should however be distinguished from pseudohyperkalamia
a rise in serum potassium secondary to release of intracellular potassium during phlebotomy or storage of blood sample. Hyperkalaemia could be due to transcellular shift, increase in intake or decrease in output. Transcellular shift is often due to metabolic acidosis; however, a sudden rise in osmolality, especially in association with insulin deficiency, could result in significant hyperkalaemia. B-blockers alone are rarely associated with significant hyperkalaemia, however, they could play a contributory part. In the presence of ECG ghanges, hyperkalaemia should be considered as an emergency and treatment should begain immediately. **(Ethier JH et al 2005).**