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DETERMINATION OF THE SERUM POTASIUM

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Abstract

The main electrolyte is the serum Potassium (cation) and the constituent of the tampon system of the intracellular liquid. 90% of the Potassium is concentrated inside the cell, only small quantities being present in the bones and blood. The injured cells issue Potassium in the blood.

The quantity of Potassium included in the food is absorbed on the level of the small bowel. The body is adapted for the efficient excretion of Potassium. Normally 80-90% of the Potassium is excreted by urine, and the rest by transpiration and stool. The Potassium from food is eliminated through the kidneys in a period of 24 hours.Even when there is no infusion of Potassium (in conditions of feast), 40-50 mEq are excreted in the urine daily. The kidneys don't preserve Potassium and when there is an inadequate food share is produced a severe deficiency. The Potassium balance is maintained for the adult in the conditions of a food share of 80-200 mEq/day.

Keywords: Potassium, electrolyte, Potassium balance

INTRODUCTION

The greatest quantity of Potassium (90%) is under the ionic form, the rest being connected to proteins. The Potassium is indispensable to a normal development of the electric phenomena of membrane. Also it plays an important role in the nervous drive, muscular contraction, acid-base balance, osmotic pressure, protein anabolism and glycogen formaion[1.] The anabolic processes are accompanied by the fixing of K^+ in the cell, and the catabolic by its discharge. The Potassium is concentrated especially in the striate muscles, myocardia and liver. Together with the Calcium and Magnesium, K^+ controls the contraction and debit of the heart. The deficit of Potassium can be underlined on an electrocardiogram by the presence of U wave.

The ions of Potassium and Sodium are important for the renal regulation of the acid-base balance, the ions of Hydrogen being substituted for the ions of Na^+ and K^+ renal tube. The Potassium bicarbonate is the main intracellular inorganic cushion. In the deficit of Potassium is produced the intracellular acidosis, to which the respiratory centers are responding by hyperventilation that leads to the decrease of CO_2 .

The increase and decrease of the concentration of the plasmatic Potassium are determined by the perturbations of the external and internal balance of Potassium. The external balance of Potassium is regulated by the secretion of Potassium in the distal and collector tubes. The modulators of the renal excretion of Potassium and as a consequence, of the external balance, are: the food share of Potassium, the content of Sodium and the rate of the flux in the distal tubes, the acid-base balance, the activity of the mineralocorticoids, the responsivity of the distal tubes to mineralocorticoids, the type and availability of the anions.

The concentration of Potassium is affected by the corticosuprarenal hormones, that stimulate the urinary excretion of Potasium. The mineralocorticoids determine directly the secretion of Potassium in the distal tubes; the glucocorticoids act indirectly, by the increase of the rate of glomerular filtration rate and of the urinary flux, and the increase of quantity of Sodium in the distal tubes.

In the presence of metabolic or respiratory alkalosis, K^+ enters in the cells with the appearance of the hypopotassemia associated with the increase of urinary excretion of Potassium, due to the hyperaldosteronism and the increase of the availability of the bicarbonate in the distal tubes. In the acute metabolic acidosis determined by inorganic acids (NH_4Cl, HCl), K^+ comes out of the cells in exchange with H^+ with acute hypertassemia; in the acidosis determined by organic acids (lactate, ketone bodies) hypertassemia appears secondary to the volemic depletion and the decrease of the urinary flux rate.

The modifications of the Potassium metabolism are considered together with the disorders of the hydrosaline and acid-base balance and with the characteristic electrocardiographic modifications, that appeared in the conditions of the variations of concentration of Potassium.

MATERIAL AND METHODS

The method by which was determined the Potassium is ISE potentiometric (selective electrode ions).

For the determination of the Potassium were considered:

1. The preparing of the patient - à jeun (on empty stomach) or postprandial; will be avoided:

• the needles of puncture very thin, the collection of blood from the veins with very small caliber (they produce mechanic hemolysis);

• the venous stasis caused by the tourniquet and the clenching of the fist during the collecting (increases K^+ with 10-20% due to the acidosis);

• the collection from the arm by which the patient received recently a transfusion;

• the performing of the intense muscular activities with the arm from which will be collected the blood sample (values falsely increased with 10-20%).

2. Specimen collected – venous blood[3].
3. Container for collection – vacutainer without anticoagulant with/without separator gel.
4. Necessary processing after the collection – is separated the serum by centrifugation in a period of <2h.
5. Volume of sample – minimum 0.5 mL serum.
6. Cause of rejection of sample – hemolysate specimen.
7. Stability of the sample – the serum kept in tubes closed hermetically is stable : 14 says at the temperature of the room, 14 days at 2-8°C and for a long time at -20°C.

RESULTS AND DISCUSSIONS

Table 1

Reference values

Age	Values (mmol/L)
Premature	3.2-4.6
1 day-4 weeks	3.6-6.1
1- 12 months	3.6-5.8
Children > 1year	3.1-5.1
Adults <= 60 years	3.3-5.1
Adults >60 years	3.7-5.4

The asymmetric distribution between the extra and intracellular medium of Potassium is owed to the activity of the membrane pump Na+ /K+ (Na-K-ATP-aza) that transports Na outside and K inside the cell in the report 3:2. The food share of Potassium is of 80-200 mEq/L and covers the daily needs of K^+ that are of 2-4 g. All the Potassium obtained from food is absorbed on the level of the small bowel. Normally 80-90% of the

Potassium is excreted by urine, and the rest by transpiration and stool. The regulation of the metabolism of Potassium is exercised by the kidneys. The potassium reaches in the nephron in great quantity, without being filtered and is reabsorbed in proportion of 90% in the proximal tube and Henle's loop, and then is secreted on the level of the collector tubes. The kidneys don't preserve the Potassium and even when there is an adequate share of Potassium, 40-50 mEq they are secreted daily by urine.

The importance of the Potassium results from the multiple functions that is accomplishes or to which it participates. The serum Potassium plays an important role in the nervous direction, the muscular contraction, the maintaining of the acid-base balance and of the osmotic balance. The Potassium influences the transmitting of the nervous influx.

The Potassium plays a more important role than Sodium, because the bicarbonate of Potassium is the main intracellular inorganic cushion.
In the hypotassemia there is a relative deficiency of the bicarbonate of intracellular Potassium, so that the Ph is relatively acid. The respiratory center is responding to this intracellular acidity by hyperventilation, thus decreasing the level of CO2. The level of serum Potassium is influenced by aldosterone, by the variations of pH, of the serum level of glucose and insulin and of sodium.

The concentration of Potassium is affected by the aldosterone, which stimulates the urinary excretion of Potassium. The reabsorption of Sodium leads to the loss of Potassium. The modifications of extracellular pH produce mutually also the exchanges of K^+ between the extra and intracellular medium. K^+ enters in the cell in alkalosis, and comes out from the cell in acidosis. The evaluation of the serum Potassium is made in report with the variations of pH. In general, the potassemia decreases with 0,3mEq/L for each 0.1U over the normal of the serum pH.

The variation of the potassemia, depending on the value of pH have to be anticipated during the evaluation and treatment of the hypo/hyperkalemia; the correcting of the alkaline pH will lead to the increase of potassemia, without the additional administration of Potassium. On the contrary a "normal" serum Potassium, in the context of an acidosis, imposes the administration of Potassium, due to the decrease of the potassemia together with the correcting of the serum pH.

The hypokalemia is defined as serum level of Potasium under 3.5 mEq/L. The hypokalemia can result by the decrease of the food share, the intracellular migration of Potassium or the increase of the Potassium losses.

The symptoms of hypokalemia include: weakness, fatigue, paralysis, difficult breathing, rhabdomyolysis, muscular cramps, constipation, paralytic ileus. In hypokalemia the modifications of ECG include: U waves, T flattened waves, modified ST segment, arrhythmias (especially if the

patient is under treatment with digoxin), electric activity without pulse or systole. The patients treated with digitalis and with hypotassemia smaller than 3 mEq are predisposed to cardiac arrhythmias.

The hypokalemia exacerbates the digitalis toxicity, for this reason it has to be avoided or treated promptly for the patients under treatment with derivates of digitalis. Hypopotassemia can precipitate the hepatic come for those with advanced cirrhosis, due to the increase of the renal synthesis of Ammonium, can induce fast and frequently modifications in the renal function, this includes the resistance to ADH with the alteration of the urinary concentration, increases the production of Ammonium and alters the renal reabsorption of bicarbonate and sodium.

The level of alarm is under 3 mEq/l and causes ventricular fibrillation, especially if the decrease of the concentration of the Potassium is sudden. The hyperkalemia represents the increase of the potassemia over the limit of 5,0 mEq/L. The most frequent causes are the increase of the Potassium release from cells and the scarce renal excretion.

Not corrected the hyperkaliemia produces progressive cardiac dysfunction and finally a systole. The hyperkaliemia can be treated by the administration of insulin, glucose and sodium bicarbonate.

CONCLUSIONS

The increase and decrease of the concentration of plasmatic Potassium are determined by perturbations of the external and internal balance of Potassium.

Leukocytosis >100000/μL can determine the false hyperkaliemia, if the sample is not processed in an hour (the leucocytes release K^+). The patients with thrombocytosis from some chronic myeloprofilerative syndromes can have values falsely increased, due to the great number of thrombocytes that release Potassium during the coagulation. For these patients is recommended the performing of the Potassium from plasma (blood sample collected on heparin). The intense physical activity, the infusion of glucose associated with insulin determines hypotassemia. The stress, the myocardia infarction, the bronchia asthma, determines the hypopotassemia by the discharge of catecholamines, that determine the passing of K^+ from the extracellular compartment in the intracellular one by stimulating the β-adrenergic receptors.

In the presence of infectious mononucleosis but also as a consequence of an inherited defect, the leucocytes and erythrocytes can lose fast K^+ *in vitro*.

The decrease of the temperature of the total blood determines the increase of plasmatic K^+ as a consequence of the release from cells.

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