POTASSIUM AND ANAESTHESIA

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Potassium is the major intracellular cation in the body and has several roles, the most important being the generation of the resting cell membrane potential and the action potential, as well as protein synthesis, acid-base balance and maintenance of intracellular osmolality. The total amount of potassium present in the body is approximately 3200mmol, 90% of which is intracellular, and this is regulated by a variety of homeostatic mechanisms.

Physiology of Potassium

Of total body potassium, approximately 135-150mmol/l is present intracellularly, compared to plasma levels of 3.5-5.5mmol/l. The daily requirement is about 1mmol/kg/day, which is absorbed from the small intestine by diffusion.

Potassium Homeostasis

The kidney is the main organ involved in potassium regulation. There is, however, some secretion of potassium in the colon, some of which is reabsorbed in exchange for H^+. Both of these are under the control of aldosterone which is the main hormone involved in potassium regulation. In the kidney the majority of filtered potassium is reabsorbed in the proximal tubules. The distal tubules then secrete potassium, and normally the amount secreted is equal to potassium intake so balance is maintained.

There are several factors that can alter the amount secreted in the distal tubules:

- The rate of flow of fluid in the tubules - potassium secretion is proportional to the rate of flow. Increasing the flow limits the build up of potassium in the tubule which would otherwise increase and inhibit further secretion. This is the main reason why the plasma potassium level is seen to fall in patients having a large diuresis.
- Electrical coupling in the distal tubules means that potassium secretion is also dependent on sodium reabsorption. The greater the amount of sodium reabsorbed, the more potassium is secreted. This means that potassium secretion will increase when there is decreased sodium in the distal tubular fluid. Potassium secretion will also increase when sodium absorption is increased, which is under control of aldosterone and Atrial Natriuretic Peptide and hence also dependent on intravascular volume and blood pressure.
- High potassium levels stimulate aldosterone secretion and act as negative feedback to maintain levels. Sodium is also reabsorbed in exchange for H^+, however, so the amount of potassium secreted is also affected by acid-base balance. Potassium secretion is decreased when there is increased H^+ secretion, for example when a patient is acidotic.
- Via a similar mechanism, acidosis also results in a shift of potassium from the intracellular compartment to the extracellular compartment.

Insulin and catecholamines are also involved in potassium regulation. They both reduce the extracellular potassium concentration by increasing uptake into cells by an action on Na^+/K^+ ATPase and $\beta 2$ receptors respectively. These mechanisms can be quite potent and the release of adrenaline during a stress response can cause an acute decrease in plasma potassium by 0.5-0.6mmol/l.

Potassium and the Membrane Potential

One of the most important roles of potassium is the resting membrane potential and in the repolarisation phase of action potentials. The normal cell membrane is relatively permeable to potassium ions, and impermeable to sodium and anions. The anions generate a negative intracellular potential. Because of the relative permeability of the cell membrane to potassium compared to sodium, this results in holding potassium intracellularly against

its chemical gradient, although there is a small leak of potassium down its concentration gradient which is balanced by the action of the Na^+/K^+ ATPase pump which in turn maintains the resting membrane potential. The end result is a resting membrane potential, the size of which is primarily due to the relative intracellular to extracellular concentration of potassium. It is the ratio of theses concentrations rather than the actual potassium concentrations themselves that is more important for maintaining the membrane potential. As a result, chronic disorders of potassium balance that involve a total body deficit or excess of potassium where the ratio is relatively well maintained, are frequently better tolerated and result in less symptoms to patients than acute disorders of potassium balance where the total body potassium may be relatively normal and the ratio disturbed. This is important when deciding whether or not to operate on a patient with an abnormal potassium level and some thought must be given to whether the disorder is chronic or acute, as well as the absolute plasma potassium level.

Hypokalaemia

This is typically taken as being a potassium level of less than 3.5mmol/l, though symptoms may not occur until the level is less than 2.5mmol/l. The total body deficit may be up to 500mmol. On average plasma potassium decreases by 0.3mmol/l for each 100mmol reduction in total body stores.

Causes may be acute or chronic.

Acute causes can be divided into two groups:

- Those that cause a shift of potassium intracellularly such as alkalosis, excessive use of β2 agonists, administration of insulin and glucose and hypothermia
- Those that result in a loss of potassium such as vomiting, diarrhea and losses in bowel fistulae, IV fluid therapy without potassium, diuresis due to solutes such as glucose or mannitol and the diuretic phase of acute renal failure.

Chronic causes of hypokalaemia tend to be those that result in a decrease in total body potassium such as dietary insufficiency, malabsorbtion, diuretics, Cushing's syndrome and hyper-aldosteronism.

Effect of Hypokalaemia

An acute decrease in the extracellular potassium concentration will result in an increase in the intracellular/extracellular ratio. This causes the membrane potential to become more negative and results in muscle and nerve cells becoming less excitable. This results in weakness and increased sensitivity to non-depolarising neuromuscular blocking drugs. In the heart, atrial, ventricular and conduction cells are all affected to a different extent resulting in increased automaticity, increased excitability and arrhythmias such as tachyarrhythmias and extrasystoles, and decreased cardiac contractility. Cardiac arrest may also occur. Patients may also develop impaired renal concentrating ability, increased toxic effects of digoxin, muscle weakness, hypotonia and alkalosis.

ECG changes: S-T segment depression, P-R and Q-T prolongation, T wave inversion and U waves.

Hypokalaemia and Anaesthesia

It used to be thought that because of the risk of developing arrhythmias, a healthy patient undergoing surgery with a potassium level of less than 3.0mmol/l should have their operation postponed if possible and have replacement therapy to normalize the plasma potassium. But there is a morbidity and mortality associated with replacement therapy. Several studies have shown no increased incidence of intraoperative cardiac arrhythmias in asymptomatic patients with chronic hypokalaemia so this is no longer the current view. Some authors have suggested that levels as low as 2.6-2.9mmol/l may be acceptable in otherwise healthy patients. There is little evidence to suggest an absolute level at which replacement therapy should be undertaken before surgery. Preoperative arrhythmias rather than plasma potassium has been shown to be a stronger predictor of intraoperative arrhythmias.

More important is taking each patient on an individual basis and assessing the following:

- The urgency of the operation,
- The type of surgery being undertaken
- The cause and time course as well as the degree of hypokalaemia,

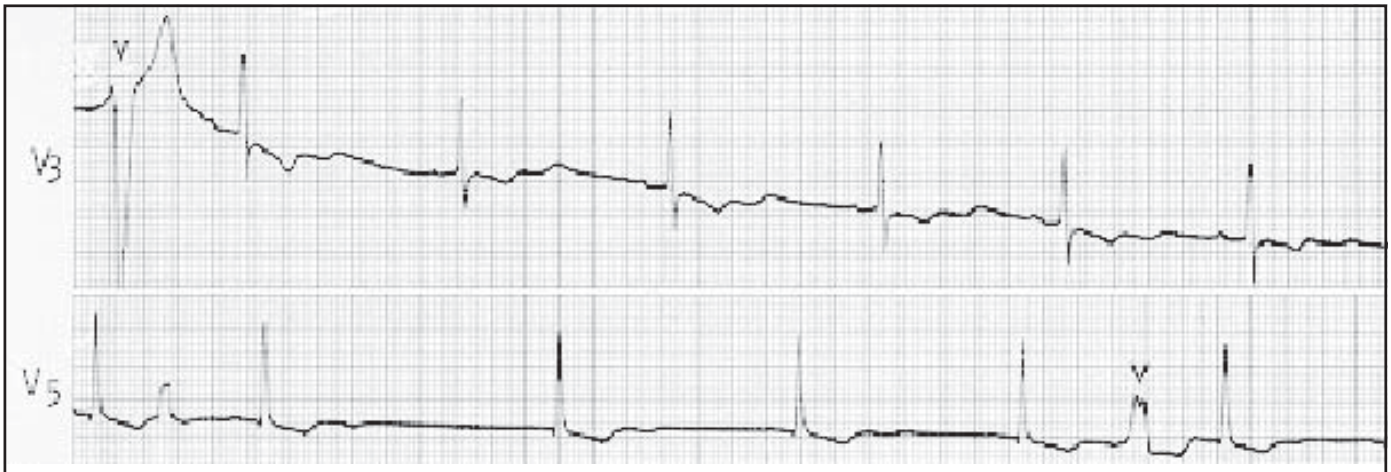

Figure 1. ECG showing T wave inversion and U waves

- Presence of arrhythmias, ECG changes or other symptoms,
- Cardiovascular risk factors such as myocardial ischaemia, heart failure or left ventricular hypertrophy
- Concurrent medications such as digoxin

Patients with acute or symptomatic hypokalaemia, preexisting cardiac disease and those taking digoxin are more likely to need replacement therapy if the level is less than 3.5mmol/l. Prior thought must be given to correction of the underlying disorder first, however, as replacement therapy may result in hyperkalaemia once the underlying cause has been corrected. Patients with chronic hypokalaemia may have a normal ICF/ECF ratio, and together with the lack of evidence that there is any increased risk due to chronic hypokalaemia, serious thought needs to be given as to whether there would be any benefit from delaying the operation and treating the hypokalaemia, which may cause problems by altering a normal membrane ratio.

The acute treatment involves first correcting the underlying disorder. If replacement therapy is deemed necessary it should be guided by estimation of total body deficit, and consists of:

- IV potassium chloride up to 40 mmol over 2 hours which needs to be given via a central vein and with full ECG monitoring, unless there is a metabolic acidosis in which case potassium bicarbonate is more suitable.
- If it does not need to be corrected quickly, oral supplementation can be used.

Caution must also be taken when correcting a chronic disturbance rapidly because this may result in an imbalance in the ICF/ECF ratio and arrhythmias. If possible, slow correction with oral potassium is better.

Anaesthetic management consists of preventing a further increase in hypokalaemia by allaying anxiety, avoiding dextrose solutions, maintaining a normal $PaCO_2$ and the use of a nerve stimulator to assess neuromuscular blockade.

Hyperkalaemia

This is defined as a plasma level of greater than 5.5mmol/l. Acute causes may be due to:

- potassium shift, such as in acidosis, rhabdomyolysis, trauma, malignant hyperpyrexia, suxemethonium (worsened by burns and nerve injury), familial periodic paralysis
- increased intake such as over-supplementation and blood transfusion.

The chronic causes are renal failure, Addison's disease and drugs such as potassium sparing diuretics, ACE inhibitors and cyclosporin.

Effect of hyperkalaemia

Acute hyperkalaemia causing a decrease in the ICF/ECF ratio will result in the resting membrane potential becoming less negative. The effect of this in muscle and nerve cells is that the membrane potential is closer to the threshold potential and so more excitable. If this continues, fatigue occurs resulting in muscle weakness. In the heart, excitability is decreased and as the activity decreases the threshold to ventricular fibrillation decreases so cardiac arrest in diastole may occur.

ECG changes: The earliest ECG change is a tall peaked T-wave, followed by prolongation of the P-R interval, widening of the QRS complex, absent P waves and slurring of S-T segments into T waves. Other symptoms include nausea and vomiting.

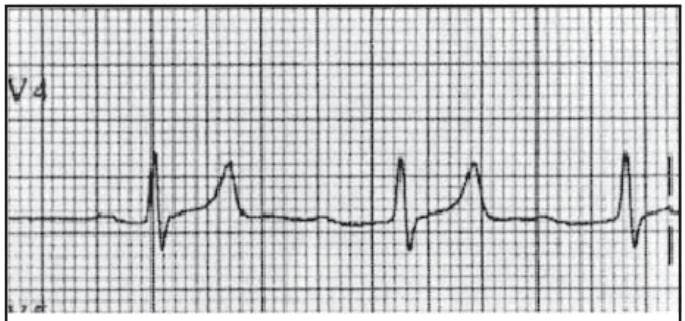

Figure 2. ECG showing peaked T waves

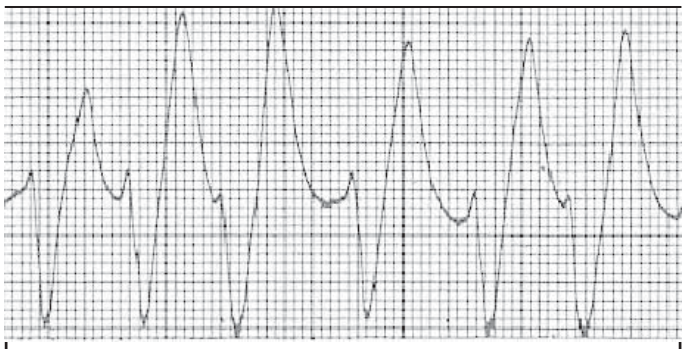

Figure 3. ECG showing wide QRS complexes

Hyperkalaemia and Anaesthesia

The decision to treat hyperkalaemia is easier and is based on the degree of elevation and the symptoms and signs present. If there are ECG changes or the concentration is greater than 6.5mmol/l the incidence of serious cardiac compromise is high and rapid intervention is necessary. A plasma potassium of less than 5.9mmol/l has been suggested before an elective operation. The cause should be investigated and corrected if possible.

Acute treatment may include the following:

- Insulin 5-10 units in 100ml of 10-20% dextrose IV over 30-60 minutes
- Salbutamol either 5mg nebulised or 50mcg bolus followed by 5-10mcg/min IV infusion
- Causing an alkalosis either by giving 50mmol IV bicarbonate or by increasing minute ventilation to cause a respiratory alkalosis if patient is ventilated.

The above all act by causing a shift of potassium intracellularly.

- Potassium exchange resins such as calcium resonium 15gPO/ 30gPR tds
- Dialysis/haemofiltration - these treatments act to remove potassium from the body.

- Calcium 5-10ml of 10% calcium gluconate IV if severe, or if there are cardiac manifestations. Calcium antagonizes the reduced conduction and improves myocardial contractility.

Further anaesthetic management consists of maintaining a normal or low $PaCO_2$ and avoidance of suxamethonium if possible.

Summary

Potassium is the major intracellular cation and is intimately involved in maintenance of the resting membrane potential. Hyper- and hypokalaemia can result in serious cardiac compromise, and treatment should be guided not only on the absolute levels but also on the presence of symptoms and other risk factors.

References

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