Singapore Med J 1998; Vol 39(11):511-516

Potassium and Anaesthesia

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INTRODUCTION

Potassium is the major intracellular cation with 98% of body K^+ being located in the cells. The total body K^+ stores in a healthy adult are approximately 3000 to 4000 mmols (50 to 55 mmol/kg body weight)[1]. Intracellular potassium concentration is around 150 mmol/L and that of extracellular concentration is 4 – 5 mmol/L only.

Potassium has two major physiologic functions[2,3]

1. Potassium participates in the process of protein and glycogen synthesis in the cell.
2. The ratio of potassium concentration in the cell and the extracellular fluid (ECF) is the major determinant of the resting membrane potential (Em) across the cell membrane. This function is energy dependent (Na^+/K^+ - ATPase) which actively pumps sodium out of the cell and potassium into the cells.

It is the resting potential that sets the stage for the generation of the action potential that is essential for normal neural and muscular function. Thus both hypokalemia and hyperkalemia can result in cardiac arrhythmias and muscular weakness leading to paralysis.

Potassium and excitable cells

A. Nerve cells

The resting membrane potential is about - 90 mV. In acute hyperkalemia, the ratio of intracellular to extracellular K^+ is decreased. The gap between the resting membrane potential to the excitability threshold is decreased and the nerve conduction is initiated more easily. If this continues it progresses to weakness of muscles[4,5]. Gradual hyperkalemia, as in chronic renal failure, will cause an increase in both the intracellular and extracellular concentration with little change in the ratio. Hence, the signs and symptoms related to hyperkalemia are less intense. The converse occurs in acute hypokalemia. Hyperpolarisation of the resting membrane leads to decreased nerve cell excitability. This results in generalised weakness, and ultimately paralysis (Paralytic ileus, respiratory insufficiency). In chronic hypokalemia, there will be minimal or no change in the K^+ gradient and no change in the resting membrane potential[6].

B. The Heart

Changes in the potassium level affect the heart at two locations[6]. Sino-atrial node: In hyperkalemia the excitability is decreased and as the activity decreases, the threshold to ventricular fibrillation decreases[7]. Conducting System: Hypokalemia increases excitability and predisposes the myocardium to catecholamine induced arrhythmia or ventricular fibrillation[8].

C. Skeletal and smooth muscle

Hypokalemia results in decreased resting membrane potential. The hyperpolarisation makes depolarisation more difficult and decreases muscular excitability. Hyperkalemia on the other hand, moves the resting membrane potential closer to the threshold. Spontaneous activity occurs more easily, but recovery is slower. With regard to smooth muscle, paralytic ileus is the end result of hypo- or hyperkalemia[6].

Potassium regulation

Most of the potassium ingested is absorbed in the small intestine and redistributed mainly to the skeletal muscle.

Renal regulation

Approximately 90% of the ingested K^+ is excreted by the kidneys. The major site of potassium secretion is the cortical collecting tubule as shown in Fig 1. An active Na^+- K^+-ATPase pump transports two K^+ into the cell in exchange of three Na^+ ions. The Na^+- K^+-ATPase pump is strongly influenced by the plasma aldosterone level[9]. Aldosterone also increases potassium permeability at the luminal membrane, stimulates sodium reabsorption and increases the uptake of potassium ions by skeletal cells[10]. After entry into the cell, potassium is secreted through a channel that is selective for potassium and this is influenced by electrochemical gradient.

Extrarenal mechanisms (Fig 2)

A. Insulin

Insulin promotes the entry of K^+ into skeletal muscle and the liver by increasing Na^+-K^+-ATPase activity[3,11,12].

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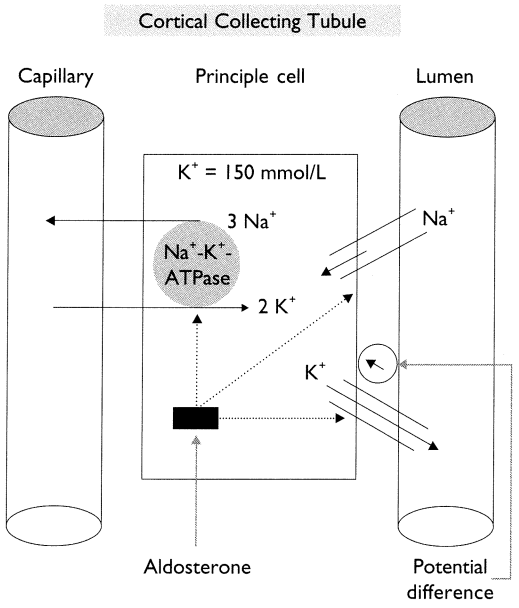

Fig 1 – Cellular mechanism of potassium secretion in the cortical collecting tubule. Note potassium entry into the cell is an active process involving Na+-K+-ATPase, whereas secretion of potassium into the lumen is related to electrochemical gradient.

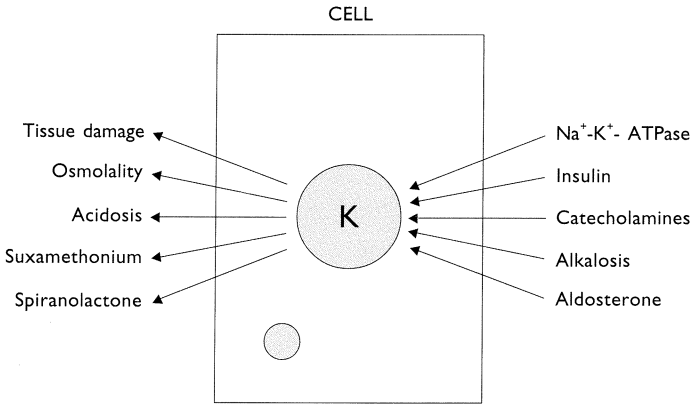

Fig 2 – Factors influencing the distribution of potassium between the cell and the extracellular fluid.

B. Catecholamines

Increase in the catecholamine levels results in an increased uptake of K^+ by the cells. This is mediated by B_2- receptors. At the same time, renal secretion of K^+ is decreased. The release of epinephrine during a stress response can acutely lower the plasma K^+ by 0.5 – 0.6 mmol/L[13-15].

C. Aldosterone

Aldosterone exerts its effect on potassium at the cortical collecting duct. Aldosterone also promotes intracellular uptake of potassium in skeletal muscles[9,10,16]. Increase or decrease in the level of serum K^+ can change production of aldosterone in the same direction.

D. Acid-base

In acidosis hydrogen, ion enters the cell and are exchanged for potassium ions, causing an increase in serum potassium. During alkalosis, the reverse occurs[1,17,18]. During acidosis, the change in plasma K^+ varies from 0.2 to 1.7 mmol/L for every 0.1 unit fall in the pH[17]. During hyperventilation for each 0.1 pH unit, the serum K^+ will decrease rapidly by about 0.2 mmol/L[1]. Bicarbonate decreases potassium in acute hyperkalemia by 0.1 mmol/L for each 1 mmol/L increase in bicarbonate concentration[18].

Potassium and anaesthesia

Every anaesthetist while evaluating a patient with potassium disorder, needs to ask, "What is the minimum or maximum potassium levels at which anaesthesia can safely be administered for elective or emergency surgery". It seems the answer is not straight forward.

Pre-operative evaluation should include history of vomiting, diarrhoea, acid-base disorder, and history of drugs since many of them are known to cause potassium imbalance[6]. If the cause is not known urinary potassium estimation may be useful (Table I).

The decision about proceeding with surgery and anaesthesia in the face of acute or chronic depletion or excess of potassium depends on many factors[19-21].

a. the cause of potassium disturbance
b. the urgency of operation
c. the degree of abnormality
d. the medication
e. acid-base balances

Hypokalemia and anaesthesia

Chronic hypokalemia is often associated with a total body K^+ deficit between 600 – 1000 mmol/L[22]. It is advisable not to correct chronic hypokalemia by intravenous infusion of potassium, as it can disturb the electrophysiology of a well compensated homeostasis[21] and there is no risk of arrhythmias peri-operatively from chronic hypokalemia[6,19,20,23-25]. It may take up to 2 days to replace the potassium loss completely[23].

Table I – Drugs that are known to cause hypo- or hyperkalemia

Hypokalemia	Hyperkalemia
Bronchodilators (Salbutamol)	Propranolol
Diuretics	Spiranolactone
Calcium channel blockers	Ace inhibitors
Insulin	Digoxin intoxication
Glucose	Non-steroidal anti-inflammatory drugs
Bicarbonate	Potassium containing Penicillin
Laxative	Fresh frozen plasma
Aminoglycoside	Heparin

The major danger in anaesthetising patients who have disturbance in serum potassium is related to myocardium in terms of arrhythmia's and poor contractility[20,26,27]. The concern of hypokalemia worsening dysrhythmias is based on the fact that hypokalemia favours the development of serious cardiac arrhythmias in patients with ischaemic heart disease, digitalis toxicity or history of serious cardiac arrhythmias[28]. There seems to be no universal agreement about the lowest level of serum K^+ where anaesthesia can be safely administered. Some have warned about the high incidence of dysrhythmias in the face of hypokalemia and heart disease[29,30]. On the other hand, others claim that the problem is not as dangerous as previously assumed[19,20,24]. The level of serum K^+ at which elective surgery can be performed varies from 2.6 – 2.9 mmol/L[19,20,24]. The range of safe potassium levels is arbitrary and has changed from 3.3 in 1979, to 3.1 in 1986 to 2.9 in 1990 on the lower side and from 5.6 in 1979 to 5.7 in 1986 and to 5.9 in 1990 on the upper side[19,20,24].

The incidence of intraoperative cardiac dysrhythmias is not increased in asymptomatic patients with chronic hypokalemia (2.6 to 3.5 mmol/L) undergoing elective surgery[19,24,32]. Cancellation of surgery for chronic hypokalemia may no longer be indicated[19,20,24]. Severe hypokalemia less than 2.5 mmol/L has been associated with the development of dangerous ventricular tachyarrhythmias even in the absence of heart disease or digitalis therapy.

Acute lowering of serum potassium may be more arrhythmogenic than chronic hypokalemia. Wong[25] suggested that serum K^+ level of < 2.7 mmol/L for elective surgery should be considered "risky" regardless of whether the patient is symptomatic or not. Symptomatic pre-operative dysrhythmias, rather than hypokalemia per se is a stronger predictor of intraoperative dysrhythmias[24,25].

Hirsch et al[19] in a prospective study of 447 patients undergoing major cardiac or vascular procedures, reported no association between the frequency or severity of arrhythmias and the pre-induction serum K^+. Based on serum K^+ levels measured immediately before surgery, 57% of patients were normokalemic ($\geq$ 3.6 mmol/L), 34% hypokalemic (3.1 – 3.5 mmol/L), and 9% severely hypokalemic ($\leq$ 3.0 mmol/L). No arrhythmias occurred intraoperatively in 63% and minor arrhythmia's which required no treatment occurred in 16%. Frequent or complex ventricular ectopy appeared before and during operation in 21% but was not related to pre-operative K^+ level. There was no relationship between potassium level and morbidity.

Vietz et al[24] prospectively evaluated the incidence of dysrhythmias intraoperatively in 150 patients undergoing surgery. There were 62 patients who were chronically hypokalemic (2.6 – 3.4 mmol/L). They found the incidence of intraoperative dysrhythmias were the same between hypokalemic and normokalemic patients. The only factor that correlated with intraoperative dysrhythmias was the presence of pre-operative dysrhythmias.

Management of anaesthesia premedication

It is advisable to repeat serum K^+ measurement just before induction of anaesthesia in a patient with hypokalemia, since plasma K^+ measured just before induction of anaesthesia is often lower than the value measured 24 hours earlier[19,23,31,32]. This acute decrease in potassium is due to beta 2-adrenergic receptor stimulation[32]. Pre-operative anxiety is known to cause an increase in the plasma adrenaline levels[14,32,33]. McCleane[14] prospectively studied 200 patients posted for elective surgery. They measured serum potassium on the day of admission and on the day of surgery. They demonstrated small decrease in serum K^+ (0.13 mmol/L) in patients who had increased anxiety. Kharasch and Bowdle measured serum potassium in a wide range of surgical patients and found an average decrease serum potassium of 0.5 mmol/L and this could be attenuated by beta 2- adrenoceptor antagonist[32].

Induction and maintenance

Chronic hypokalemia is associated with reduced myocardial contractility and postural hypotension. These patients may be sensitive to cardiac depressant anaesthetic drugs or hyperventilation[22]. During surgery, dextrose containing solutions should be avoided, as hyperglycaemia could contribute to hypokalemia[22]. Induction of anaesthesia has very little effect on serum K changes, provided $PaCO_2$ remains normal[23]. Intravenous calcium injections which are often recommended during massive blood transfusion may worsen the hypokalemia[34].

Non-depolarising muscle relaxants (NDMR)

Acute hypokalemia enhances non-depolarising muscle relaxant block[35]. Chronic hypokalemia is more likely to be associated with a normal ratio of intracellular to extracellular potassium such that response to muscle relaxants are not altered[22]. A prudent-approach is to reduce the initial muscle relaxant dose to 25% to 50%[35,36]. Subsequent administration of NDMR should be guided by a peripheral nerve stimulator. The action of neostigmine to reverse the neuromuscular blockade is lessened in the presence of hypokalemia[36,37]. Hyperventilation should be avoided as this may decrease potassium level[22]. It is important to monitor the ECG continuously during the intraoperative and post-operative periods. Capnography and arterial blood gases should guide proper ventilation.

Local anaesthesia

Local anaesthesia is safe in the presence of hypokalemia. Local anaesthetic with epinephrine can decrease the serum K^+ level, because of epinephrine induced hypokalemia[13,33]. Hahn[38] found a mean decrease in the serum potassium of 0.4 mmol/L (range 0 – 1) in 45 patients after epidural anaesthesia with 2% mepivacaine with epinephrine. Subsequently Toyoda et al[39] prospectively evaluated 30 healthy adults undergoing elective surgery under axillary blockade and found that patients who received axillary block using 1% lidocaine with epinephrine 1:100,000

(n=15), showed a significant decrease in serum K^+ levels to a maximum of 2.9 mmol/L at 30 min after the blockade. But patients who received propranolol 2 mg intravenously (n=15) before blockade did not show any significant reduction in serum K^+ levels. Based on the data available, it is advisable to avoid epinephrine in local anaesthesia for patients with hypokalemia. If epinephrine has to be used in the local anaesthetic, prior treatment with beta blocker is advocated[39].

Post-operative period

Hypokalemia in the post-operative period may be related to peri-operative problems. Rewarming the patient quickly may result uptake of K^+ into the cell resulting in hypokalemia[40]. Infusion of ionotropic agents like epinephrine and dopamine may worsen hypokalemia[21].

Hyperkalemia and anaesthesia

The major cause of pre-operative hyperkalemia are renal failure, infusion of fresh frozen plasma, transfusion of old blood, tissue damage, drugs, and hypoxia[23]. Plasma potassium should be below 5.9 mmol/L before subjecting patients to elective operation[20]. Hyperventilation will help in reducing serum K^+ (10 mmHg decrease in $PaCO_2$ reduces plasma K^+ concentration by about 0.5 mmol/L[41]).

In normal patients, the maximum increase in serum potassium is 1 mmol/L, when succinylcholine in a dose of 1 mg/kg is given intravenously[42]. But there are conditions where succinylcholine can cause serious rise in potassium.

Burns: The serum potassium may increase to dangerous levels if given succinylcholine. This susceptibility to hyperkalemia exists between about 7 and 60 days post burn[20,43-45]. If the patient has infection, the 60-day rule should be extended in view of tissue degeneration.

Trauma: Birsch et al[46] found significant increase in serum potassium after one week when given succinylcholine. The response could be prevented by pre-treatment with d-tubocurarine. The patient as in the case of burns, is susceptible for 2 months following trauma.

Renal failure: Suxamethonium is safe in the presence of renal failure associated with potassium level less than 5.5 mmol/L. But serum potassium more than 5.5 mmol/L in the presence of uremia requires postponement of the operation[47].

Paraplegia: Patients who are hemiplegic or paraplegic are vulnerable to hyperkalemia within the first 6 months after the onset. Succinylcholine administration may result in an increase in serum potassium to dangerous levels. The degree of hyperkalemia correlates well with the severity of muscle wasting[48]. Succinylcholine should be used carefully in other conditions like tetanus[49] and muscle dystrophies[50].

Since there is no effective way to prevent release of potassium after administration of succinylcholine, (including pre-treatment with a subparalysing dose of non-depolarising muscle relaxant), it is advisable to avoid administering succinylcholine to patients with elevated serum potassium. Interestingly, pre-treating the patients with magnesium sulfate 60 mg/kg intravenously prevented the rise in serum K^+[51]. The mechanism of action is probably related to prevention of potassium efflux from the myocardial cells. Nevertheless, hyperventilation before the administration of succinylcholine may provide some degree of protection[22].

Appearance of classical ECG changes of hyperkalemia after administration of succinylcholine should be immediately countered by hyperventilation with increased oxygen in addition to using drugs, to antagonise the effect of hyperkalemia[23].

Non-depolarising muscle relaxants (NDMR)

Response to non-depolarising muscle relaxant in the presence of altered serum potassium is unclear. The dose of pancuronium was directly related to plasma potassium[35,52]. A high serum potassium may antagonise the action of pancuronium and the dosage needs to be increased by as much as 25%[35,36]. The dose of d-tubocurarine needs to be increased as much as 36%[35]. Presence of skeletal muscle weakness pre-operatively would suggest the possibility of decreased muscle relaxant requirements intraoperatively. It is advisable to monitor patients with the help of a peripheral nerve stimulator and titrate the dose[22].

Maintenance

The requirement of volatile anaesthetic agents does not alter in the presence of hyperkalemia[53]. Pre-operative and perioperative intravenous fluid must be carefully selected depending upon the serum potassium level of the patient[22]. Ringer's lactate contains 4 mmol/L of potassium. Hypoventilation during anaesthesia should be avoided, because a drop in the pH from 7.4 to 7.3 can increase the serum potassium levels from 5.5 to 6.5 mmol/L[26,27].

Factors which are important during the induction of anaesthesia continue to play a role in maintaining serum concentration of K^+ (Table II). Malignant hyperthermia can result in dangerous elevations of potassium.

Drugs such as calcium gluconate, sodium bicarbonate, glucose – insulin must be readily available to treat hyperkalemia.

Table II – Factors responsible for change in serum potassium during maintenance of anaesthesia

Hypokalemia	Hyperkalemia
Hyperventilation (1)	Hypoventilation (1)
Metabolic alkalosis (17)	Metabolic acidosis (17)
Sodium bicarbonate (18)	Hypothermia (40)
Blood transfusion (54)	Blood transfusion (54)
Epinephrine (13)	Potassium contaning fluids (23)
Alpha adrenergic blockade	Hypoxia (40)

Hyperkalemic periodic paralysis

Patients with hyperkalemic periodic paralysis who undergo anaesthesia require special attention to prevent anaesthetic induced paralysis. In these patients, one should avoid potassium releasing anaesthetic drugs like succinylcholine and ketamine. Temperature should be maintained and carbohydrates in the form of glucose should be supplied[55].

Post-operative period

Acute hyperkalemia in the post-operative period is usually associated with renal failure and after liver and renal transplantation[56].

CONCLUSION

Based on the available data, anaesthesia may be safely undertaken in patients with chronic hypo- or hyperkalemia (2.9 – 5.9 mmol/L) without any pre-existing cardiac abnormality. It is not clear at present whether this low potassium is safe in patients with pre-existing cardiac disease who are on diuretics and digitalis. Serum potassium up to 5.9 mmol/L is well tolerated during anaesthesia as long as the anaesthetic technique per se does not predispose the patient to hyperkalemia. The safe anaesthetic management is to avoid iatrogenic hypo- or hyperkalemia during anaesthesia.

ACKNOWLEDGEMENT

The author wishes to thank Ms Anelia Manzanilla for her secretarial work.

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