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Potassium Ion and Anaesthetic Implications

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SUMMARY

Potassium is the major intracellular cation of our body. Its role in excitability of vital cell membranes like nerve, skeletal muscle and cardiac muscle is crucial and undisputable. It is directly involved in the function of the kidney. It is a catalyst in a few enzymatic reactions and it influences the control of osmotic pressure. As potassium has assumed a major role in perioperative & critical care settings the knowledge about this ion becomes a necessity for practicing anaesthesiologists. Hence in this review, we have tried to detail the normal potassium physiology, the deficit (hypokalemia) and the excess (hyperkalemia) of this ion, the clinical implications and correction of these disorders and the practical problems of influence of potassium ion during administration of anaesthesia.

HANDLING OF POTASSIUM BY THE BODY

Potassium (K+) is the principal intracellular cation. Approximately 3500 m mol of potassium is present in the body. (i.e.) 50 m mol/kg.[1] Potassium is the most abundant exchangeable cation in the body. It exists predominantly in the intracellular fluid at concentrations of 140 to 150 meq/liter and in the extra cellular fluid at concentrations of 3.5 to 5 meq/liter. The maintenance of the serum potassium concentration is a complex bodily function and results from the balance between intake, excretion, and distribution between intracellular and extra cellular space. Ingested potassium is virtually completely absorbed from and minimally excreted through the intestine under nonpathologic circumstances. Renal excretion of potassium, which is the major chronic protective mechanism against abnormalities in potassium balance, depends on filtration, reabsorption, and a highly regulated distal nephron secretory process. Factors regulating potassium secretion include prior potassium intake, intracellular potassium, delivery of sodium chloride and poorly reabsorbable anions to the distal nephron, the urine flow rate, hormones such as aldosterone and beta catecholamines, and the integrity of the renal tubular cell. The maintenance of distribution between the inside and outside of cells depends on the integrity of the cell membrane and its pumps, osmolality, pH, and the hormones insulin, aldosterone, beta 2-catecholamines, alpha-catecholamines, and prostaglandins. Both distributions across cell membranes and/or renal excretion of potassium may be altered by pharmacologic agents such as diuretics, alpha- and beta-catechol antagonists and agonists, depolarizing agents, and digitalis.[2,3]

The distribution of body potassium is as below.

Table 1

Showing potassium distribution in body fluids.

	Conc. of K+	Total K+
Extra cellular fluid (ECF)	5 m mol /litre	30-70 m mol Plasma – 15 m mol Interstial fluid- 35 m mol
Intra cellular fluid (ICF)	150 m mol / litre	2500-4500 m mol Muscle – 2650 m mol Liver – 250 m mol RBC – 250 m mol Others - (urine, sweat, stool and bone)

1. The cell membrane conductance for potassium is higher than sodium and anions. (i.e.) Cell membrane is more permeable for potassium.

2. The Na+K+ ATPase pump actively transports K+ in and Na+ out of the cell[4] but in a ratio of 2:3.

The above two facts account for the differences in ion concentrations in ICF and ECF and the maintenance

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of – 90 mv resting membrane potential across membranes. The ratio of ICF potassium and ECF potassium is around 30:1(150 mmol: 5 mmol) and it is clinically significant as any alteration in this ratio causes symptoms rather than actual deficit or excess of potassium.[5] The ratio rather than the actual concentrations is essential for the maintenance of membrane potential. Hence in chronic disorders of potassium balance that involves an excess or deficit of total body potassium, the ratio is well maintained to cause minimal symptoms. On the other hand, acute changes involving disturbance of the ratio produce symptoms. This assumes significance when deciding whether or not to operate on a patient with an abnormal potassium level and consideration should be given to whether the abnormality is acute or chronic.[6]

The daily requirement of potassium is around 1 mmol/kg[5] which is absorbed in the small intestine through diffusion. Food provides a major source potassium intake.

Table 2

Showing potassium content of some foods.[5]

Highest content	Very high content	High content
25 mmol/ 100 gm	12.5 mmol/ 100 gm	6.2 mmol/ 100 gm
Dried figs Molasses	Dried fruits (dates) Nuts Avocados Wheat germ	Spinach Tomatoes Carrots Potato Cauliflower Banana Oranges.

The renal excretion of potassium is the major route of elimination of dietary and other sources of excess potassium. Out of the filtered load of potassium 90% is reabsorbed by the proximal convoluted tubule and the loop of Henle. Proximally potassium is reabsorbed passively with sodium and water while Na+K+2Cl- co transporter mediates potassium uptake in the thick ascending limb of loop of Henle. Hence potassium delivery to cortical collecting duct (CCD) approximates dietary intake. It is the secretion by the principal cell[7] which maintains the potassium balance in either an excess or a deficient potassium setting. When the ECF potassium is increased the distal secretion is increased to effect a kaliuresis.This distal secretion is influenced by many factors.[8]

1. Aldosterone, ECF potassium and increased urine flow can enhance potassium secretion.
2. Electrolytes.

The luminal potential in the CCD varies from – 50 mv to 0 mv. Hence the gradient from the cell (-90 mv) to the lumen varies from 40 to 90 mv. As the luminal sodium increases (i.e. increased Na excretion) the gradient increases to favour a potassium secretion. The reabsorbable anion chloride decreases the gradient while nonabsorbable anions like sulfate, bicarbonate increase the gradient to alter the potassium secretion. Within the distal nephron magnesium dependent Na+K+ ATPase plays a crucial role in potassium regulation. Hence Mg depletion can lead on to potassium wasting. Unlike other electrolytes as secretion is the major regulator of potassium, the kidneys manage to excrete the dietary intake even if the GFR is around 8 ml /min [9, 10]

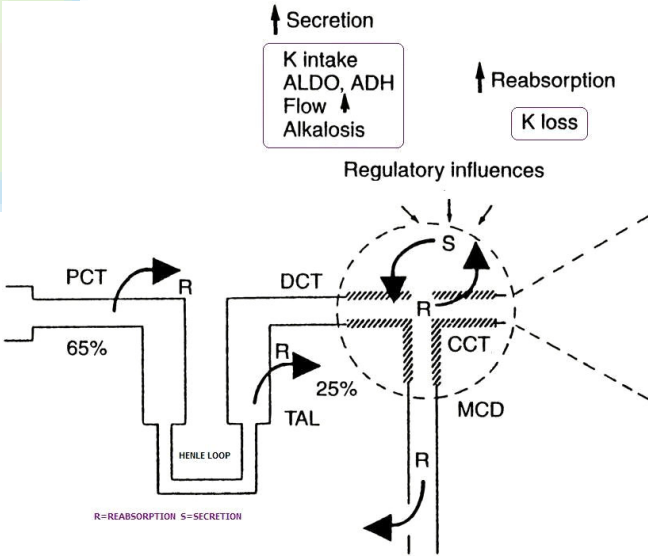

Figure 1

Showing renal regulation of potassium excretion

EXTRA RENAL REGULATION

The Gastrointestinal tract contributes to the elimination of K+.When the dietary intake is normal and climate temperate, approximately 10% passes through the bowel and a small amount in sweat. In chronic renal failure the stool potassium increases by additional colonic secretion [11, 12] and [13]

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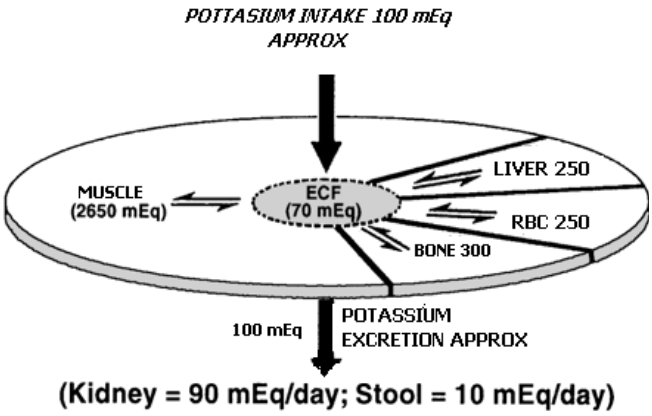

Figure 2

Showing the normal potassium profile.

POTASSIUM AND EXCITABLE CELLS

Depolarization of nerve cells occurs due to selective increase in sodium permeability. The rapid increase in intracellular sodium is reversed by outward movement of potassium. Neuromuscular excitability is largely influenced by ICF /ECF potassium ratio. Alteration in potassium concentration influence nerve cell function as they alter the ratio. As ICF depot is much higher, the ECF potassium will have more impact on the ratio. In the heart, alteration of the ratio can influence in various locations like pacemaker sites, conducting systems and the contractile states. Acute decrease in the ECF potassium increase excitability and make the myocardium more prone for ventricular fibrillation and catecholamine prone arrhythmias. This assumes significance due to the fact that blunting of intraoperative catecholamine surge lies in the hands of anaesthesiologists.Potassium alters muscle function by its influence on muscle activity. With decreased extra cellular potassium the resting membrane potential becomes more negative to produce hyper polarization which in turn decreases muscle excitability. With increased extra cellular potassium spontaneous depolarization occurs more easily which results in slower recovery after action potential4. Both hypokalemia and hyperkalemia can cause ileus of the smooth muscle. Hence the knowledge of potassium and muscle excitability is imperative for practicing anaesthesiologists who commonly play with muscle relaxants and deal with postoperative ileus of the abdomen.

HORMONAL INFLUENCE ON POTASSIUM HANDLING

1. Aldosterone:

Aldosterone seems to increase inward sodium conductance from the collecting duct to favour a potassium secretion. It also causes increased ATPase activity.[9,14] High serum concentration of K+ induces aldosterone secretion while hypokalemia suppresses it.

2. Glucocorticoids:

They influence renal potassium secretion by direct action on the renal parenchyma. Dexamethasone is said to increase glomerular filtration and thereby increased flow through CCD to favour a kaleurisis. Glucocorticoids increase renal Na+K+ ATPase activity[15] and hence stimulate potassium secretion. An increase in postoperative steroids may be avoided to attain a better electrolyte status.

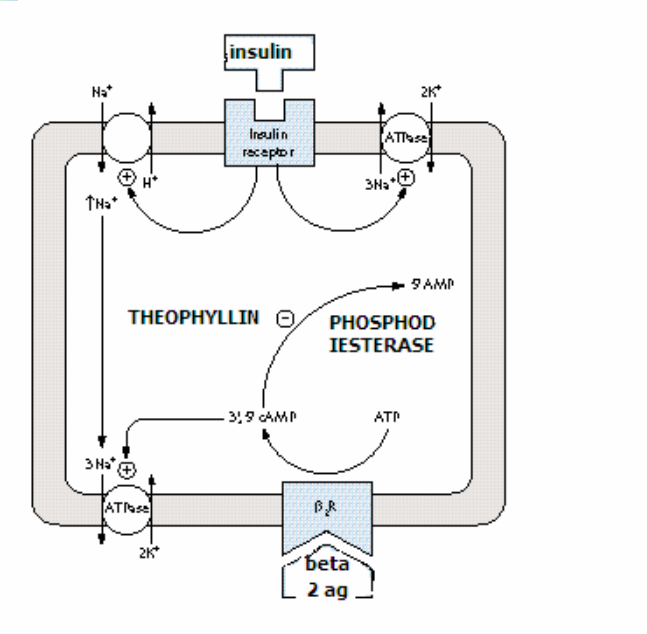

Figure 3

Showing hormonal influences on potassium handling

3. Catecholamine

They decrease renal secretion by acting on the distal collecting system. They effect an intracellular movement of potassium also.[16] Chronic administration of catecholamine can oppose potassium secretion.

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4. Antidiuretic hormone: (ADH)

This increases the secretion of K+ in the CCD.[17] This is mediated by increased sodium reabsorption and increased activity of the potassium channel of the luminal cell. It accentuates the action of aldosterone. This hormone may function as the potassium regulator with reference to total body water content.

5. Insulin

The effect of acute insulin rise is to drive potassium intracellular mainly to the muscle, liver and the general ICF.[18] Acute increase in potassium may stimulate the release of both glucagon and insulin. In hypokalemia,[19] glucose intolerance develops which usually gets corrected with potassium therapy.[20]

ACID BASE AND POTASSIUM

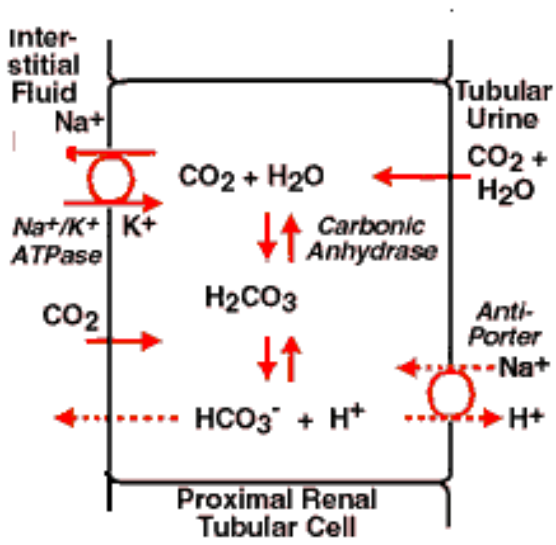
Renal secretion of potassium is influenced by acute pH changes.[21] (See figure below) Alkalosis stimulates and acidosis inhibits K+ secretion. An increase in pH of 0.1 results in a decrease of serum potassium by 0.6 mmol/l and vice versa. Both respiratory and metabolic acidosis is associated with hyperkalemia which probably occurs as a compensatory mechanism. Acidosis causes excess of extra cellular H+ ions. This induces an H+ shift into the cell followed by a K+ shift outward to produce hyperkalemia. Metabolic acidosis can be caused by either an increase in mineral acid (NH_4Cl, HCl) or organic acid (lactate, beta hydroxy butyric acid). Mineral acid decreases urinary K+ excretion while organic acids tend to cause less hyperkalemia because of its insulin secreting action.[6]

Potassium loss causes a metabolic alkalosis as follows:

When potassium ions are lost from the cell, sodium and hydrogen ions move from the extra cellular water into the cell. But for every 3 potassium ions lost, only 2 sodium ions enter the cell the other one being the hydrogen ion. This causes a decreased extra cellular hydrogen ion to produce an alkalosis. The second mechanism is that under normal circumstances, the secretion of potassium ions from the principal cells in the urine occurs in association with reabsorption of sodium ions from the tubular urine to the cells and thereby into the blood. These processes take place at different anatomical sites. If there is hypokalemia the exchangeable potassium ions of the tubular urine is decreased. Hydrogen ions instead of potassium ions will be excreted for reabsorption of sodium ions. This urinary loss of hydrogen ions causes acidic urine in the presence of metabolic alkalosis due to hypokalemia. Alkalosis conversely causes hypokalemia. Potassium ions move into the cells to push hydrogen outside. This increases extra cellular hydrogen ions to counter alkalosis.[6] In respiratory alkalosis, the effect of hyperventilation is to drive K+ intra cellular and this can cause clinical problems if there is significant intraoperative hyperventilation to produce hypokalemia.

Figure 4

Showing acid base changes and potassium



DRUGS AND POTASSIUM

Drugs cause hypokalemia by three mechanisms.

1. Intracellular movement of potassium.

Beta agonists like salbutamol, terbutaline epinephrine (used in Bronchial Asthma and Premature labour.)Insulin and theophylline[22,23,24] and [25] produce hypokalemia.

2. Gastrointestinal loss

Chronic laxative abuse.[26]

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3. Renal loss

Diuretics like thiazides, loop diuretics, large doses of penicillin, amino glycosides, levodopa, and prostaglandins (used to induce abortion) can cause kaliuresis by different mechanisms[27, 28] and [29].

Drugs cause hyperkalemia by various methods.

1. Extra cellular redistribution

Succinyl choline, digitalis, beta blockers, amino acids.[30, 31, 32]

2. Aldosterone interference

Nonsteroidal anti-inflammatory drugs (NSAIDs), Angiotensin converting enzyme (ACE) inhibitors, Angiotensin receptor blockers, azole antifungals, heparin including low molecular weight heparins. cyclosporine, flourides and spironolactone. [33,34,35,36,37,38,39]

3. Diminished potassium excretion

Amiloride, trimethoprim, pentamidine, potassium in higher doses especially in renal impairment.[40]

4. **Hemolysis.** Packed cell transfusion.

Angiotensin converting enzyme system : Renin is an enzyme that acts on angiotensinogen to catalyze the formation of the decapeptide angiotensin I. This decapeptide is then cleaved by ACE to yield the octapeptide angiotensin II. Angiotensin II is known to have several effects that are considered to be clinically significant, the major ones being altered peripheral resistance, altered renal function and altered cardiovascular structure. These effects form the basis for majority of therapeutic uses of ACE inhibitors.

The indications include.

- Hypertension.
- Congestive cardiac failure.
- Diabetic nephropathy.
- Myocardial infarction.
- Atherosclerosis.
- Stroke.
- Migraine.
- Raynaud's phenomenon

Hyperkalemia: Hyperkalemia can develop in patients on ACE inhibitors, especially in those with renal impairment or DM and those receiving drugs that can increase serum potassium concentration (e.g. Potassium sparing diuretics, potassium supplements, potassium containing salt substances).As indications of ACE inhibitors are becoming wide, the possibility of patients coming for anaesthesia is increasing. Hence in such patients, perioperative monitoring of electrolytes becomes a necessity.[42,43]

The clinically significant drug interactions of ACE inhibitors with relation to potassium and anaesthesia are showin in Table 3.

Table 3

S.No.	Drugs	Outcome	Onset	Mechanism	Management
1.	Potassium	Hyperkalemia	delayed	Lowered aldosterone level	Monitor potassium level and renal dysfunction.
2.	NSAIDs	Decreased antihypertensive effects.	delayed	Inhibition of prostaglandins.	Monitor for potassium renal dysfunction & blood pressure
3.	Amiloride	Increased antihypertensive effects	delayed	Lowered aldosterone level	Monitor potassium
4.	General anaesthetics	Marked hypotension			
5.	Cox2 inhibitors	Decreased antihypertensive, natriuretic effects.	delayed	Inhibition of production of vasodilator and natriuretic prostaglandins	Monitor for potassium renal dysfunction

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HYPOKALEMIA

A serum potassium of less than 3.5 mmol/l is termed hypokalemia.Patients with serum K+ of 3-3.5 mmol/l often have no symptoms. With more severe hypokalemia nonspecific symptoms like fatigue, muscle weakness, lassitude and constipation can occur.[43] Potassium loss impairs the ability of the kidneys to concentrate urine and thirst and polyuria ensue. With less than 2 mmol/l, ascending paralysis may follow. The likelihood of symptoms depends more on the rapidity of decrease than the intensity of decrease. Smooth muscle dysfunction (paralytic ileus) may manifest. Hypokalemia induced Nacl reabsorption may have a hypertensive effect. One of the serious difficulties in studying potassium excess or deficit is that it is mainly an intra cellular ion. Serum levels indicate the extra cellular status and for this reason ECG changes are preferred to study potassium changes on the assumption that there are no local alterations in potassium content of heart muscle cell due to injury or strain. ECG changes are the cornerstone in the diagnosis of hypokalemia. This may be low and broad T waves, prominent U wave, depressed ST segment and lengthened QT interval. There may be increase in P wave amplitude, widening of QRS interval. Numerous arrhythmias including sinus bradycardia, prolonged PR interval, Wenckeback's type, PAT with block are possible.[44,45]

The major causes of hypokalemia are:

Table 4

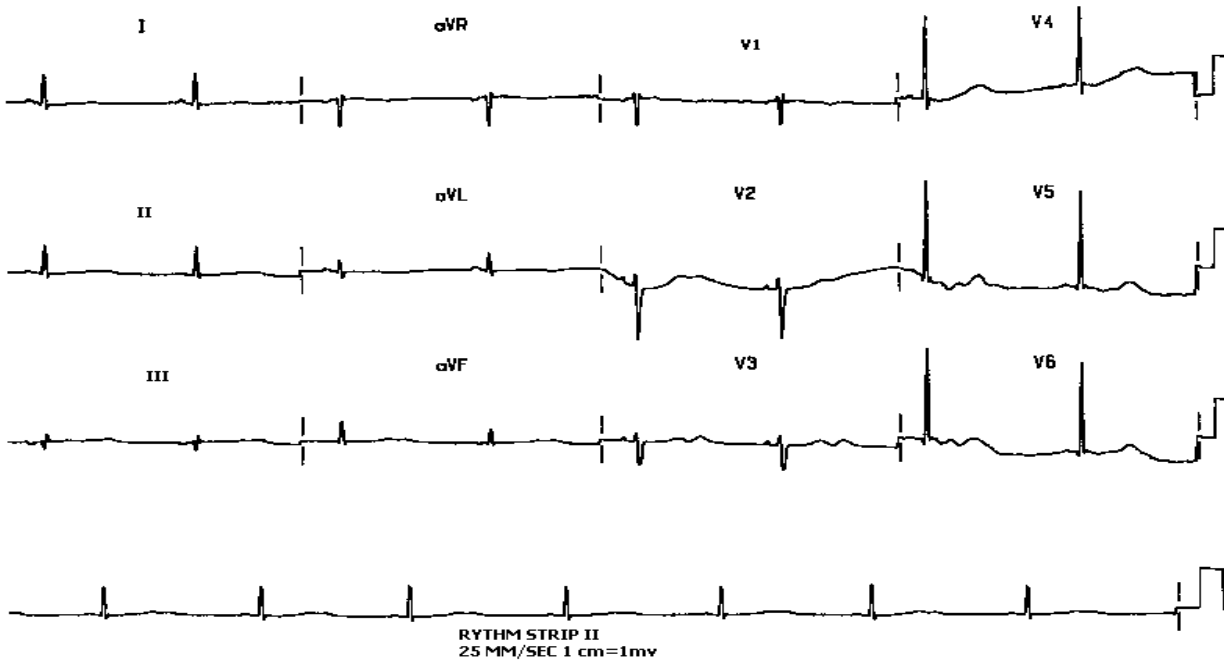
Causes	Mechanisms
Inadequate intake	Starvation, alcoholism, Anorexia nervosa, mineralocorticoid excess.
Excessive renal loss	Diuretics, Barrter's syndrome, chronic metabolic alkalosis, drugs like penicillin, ticarcillin.
Gastrointestinal losses	Vomiting, diarhoea. Villous adenoma
Shift from ECF to ICF.	Beta 2 agonists, acute alkalosis, insulin, B_{12} therapy

Table 5

Showing potassium deficit and serum level

Potassium deficit (body)	Decrease in serum level
200-400 mmol	4.0 to 3.0
600 mmol	3.0 to 2.0

FIGURE SHOWING HYPOKALEMIA AND ECG CHANGES



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The therapeutic goals of hypokalemia are

1. Treatment of the cause.
2. Stop ongoing loss.
3. Correct deficit.

The correction of deficit can be either by oral or parenteral potassium.

Oral potassium

Three salts are available for repletion of body stores.

1. Potassium chloride.(KCl)
2. Potassium phosphate.
3. Potassium bicarbonate.

Potassium phosphate is reserved for combined potassium and phosphate losses while potassium bicarbonate is suited for a clinical setting of acidosis and potassium loss. KCl is used routinely and is available in liquid and tablet forms. The liquid form is less expensive and but have an unpleasant taste. The tablets are available as slow release and are well tolerated with a rare association of ulceration and bleeding of the gastrointestinal tract. Ideally 40-100 mmol of potassium is needed to replace and maintain potassium balance in patients receiving diuretics. If hypokalemia persists, addition of a potassium sparing diuretic like amiloride is the answer. Potassium salts in the market contain 12 mmol/gm and are effective but hyperkalemia remains a threat. Intravenous potassium is reserved for correction of cardiac arrythmias, periodic paralysis and myopathies. Intravenous (IV) potassium should be used cautiously not more than 20 mmol/hour with ECG monitoring.[46] Shifting the paediatric patient to the theatre with flowing electrolyte solution and increasing the rate for intraoperative losses can be dangerous. Reduced sodium intake corrects hypokamia of hyperaldosteronism while magnesium supplementation corrects hypokalemia of hypomagnesemia.[47] In patients with acidosis and hypokalemia such as Diabetic ketoacidosis, potassium administration should be scientific to avoid an early hyperkalemia and a delayed hypokalemia[5,6]

The approximate rise of serum potassium after 0.5 meq/kg of IV potassium therapy is given in Table 6.[48]

Table 6

Routine	0.6
Patients on beta blockers	0.9
Patients on catecholamine.	0.1

HYPERKALEMIA

It is defined as a plasma concentration of more than 5.0 meq/litre.It occurs as a result of either a potassium release from cells or a decreased renal loss. Tissue trauma, transfusion of large amount of bank blood, enthusiastic administration of IV potassium, acute renal failure are some of the important causes Increased potassium intake is rarely the sole cause of hyperkalemia. Drugs causing hyperkalemia are detailed above. Pseudohyperkalemia represents an artifactually elevated plasma potassium concentration. The settings are

Causes of actual potassium values lower than Laboratory values

1. Drawing blood during potassium infusion.
2. Laboratory error.
3. Hemolysis.
4. Leukocytosis.
5. Thrombocytosis.
6. Repeated clenching of the fist before drawing blood.
7. Traumatic venipuncture.
8. Familial pseudohyperkalemia.

The causes of hyperkalemia may also be classified as 1.acute 2.chronic

Acute causes are due to shifts such as acidosis, drugs, depolarizing muscle relaxants and periodic paralysis. Chronic causes are due to renal insufficiency conditions. Hyperkalemia can occur with a low or normal potassium stores. The clinical features point to cardiac toxicity. It includes arrythmias, conduction abnormalities and finally cardiac arrest. These effects are more pronounced in hyponatremia, hypocalcaemia and acidemia. Ascending muscle paralysis can occur if plasma potassium exceeds 7 meq/l. This may progress to aphonia and respiratory arrest. The ECG changes described are elevated ST segments, tall or inverted T waves absent P waves and an irregular idioventricular rhythm.[49-56]

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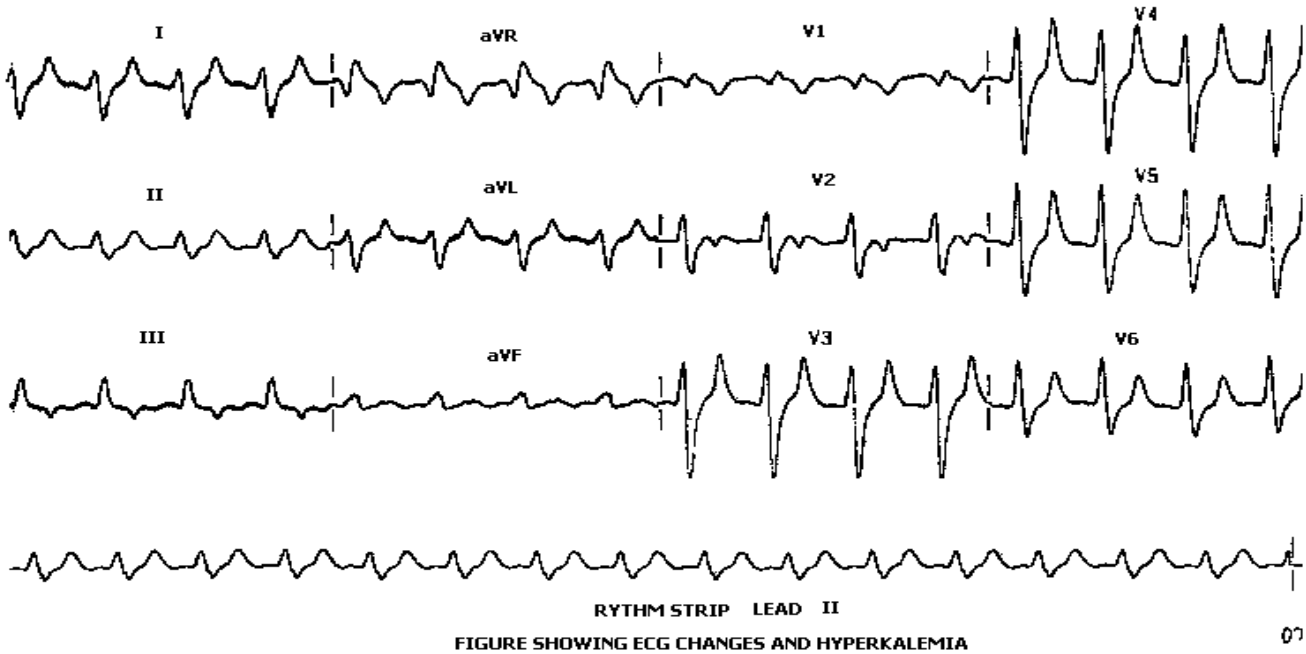
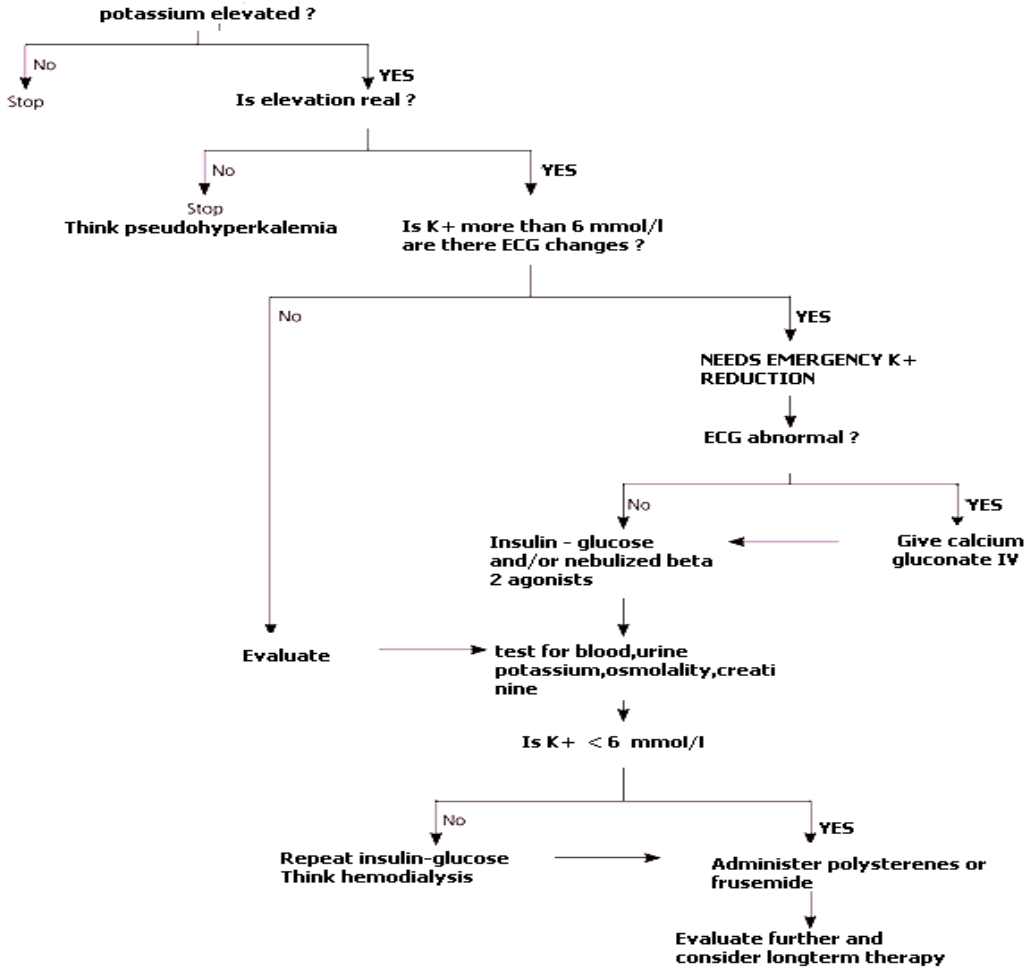


FIGURE SHOWING ECG CHANGES AND HYPERKALEMIA



LGORITHM FOR MANAGEMENT OF HYPERKALEMIA

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Table 7
Showing management of hyperkalemia.[57,58,59]

	MECHANISM	ONSET	DURATION
Calcium gluconate 10-20 ml IV (10%solution)	Direct antagonism	Rapid	15-30 minutes
Hyperventilation (PaCO2 25-30 mmHg)	Intracellular shift	Rapid	
Hemodialysis	Remove	Rapid	
Sodium bicarbonate. 50-100 meq IV	Intracellular shift	15-30 minutes	3-6 hours
Glucose(25-50gm) + insulin(10-20 units) infusion or inhaled beta 2 agonists	Intracellular shift	15-30 minutes	3-6 hours
Oral Resins (sodium polystyrene sulfonate) 25-50 gm with 100 ml 20%sorbitol	Exchange of Na With K+ in GIT	1-2 hours	4-6 hours
Peritoneal dialysis	Remove	Immediate	
Diuretics(frusemide in 40 mg doses)	Remove	Immediate	4-6 hours

Management includes shifting potassium into cells, antagonism of its deleterious effects on heart and removal of excess potassium from the body. The safe and effective use of insulin dextrose in the treatment of hyperkalemia is highlighted with particular emphasis placed on the dose of insulin in the designated insulin syringe. The administration of sodium bicarbonate should be cautious in the absence of accompanying acidosis. Extreme vigilance with ECG monitoring is a must during administration of calcium with special emphasis in patients on digoxin.[60,61]

ANAESTHETIC RELEVANCE

Preanaesthetic considerations:

- Preoperative drug effects on potassium levels should be borne in mind.
- Ryle's tube aspiration and aggressive bowel preparation may cause hypokalemia.
- It was earlier thought that because of risk of intra operative arrhythmias, a healthy patient undergoing surgery with a serum level of less than 3 mmol/l should have their surgery postponed and correction instituted. Some authors have even suggested that levels of 2.6mmol/l are acceptable in healthy patients. Still there is no exact evidence to suggest an absolute level before surgery. There is evidence that preoperative arrhythmia is a better indicator of intraoperative arrhythmias rather than potassium level.[62,63,64] Hence postponement of surgery is not warranted in a healthy patient with plasma potassium of less than 3 mmol/l. Patients with acute or symptomatic hypokalemia, pre existing cardiac disease, patients on digoxin are likely to need potassium supplements and either oral or intravenous is considered according to the needs of the situation.
- Plasma potassium measured in a blood sample obtained before surgery is often less than the value two days prior. Hence allaying anxiety by appropriate premedication is certain to decrease sympathetic stimulation and concurrent hypokalemia.It is reported that clonidine premedication is said to prevent preoperative hypokalemia.[65]
- Individual clinical decisions are important before preoperative potassium replacement after considering the following.

1. Urgency of surgery.
2. Type of surgery.
3. Cause and time course of hypokalemia.
4. Presence of arrhythmias.

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5. Cardiovascular risk factors.
6. Concurrent medications like digoxin.

- Plasma potassium of less than 6 mmol/l is suggested for elective surgery. The cause of hyperkalemia should be investigated and corrected if possible.
- Preoperative exhaustive exercise66 may increase K+ up to 2 mmol/l.

INTRAOPERATIVE CONSIDERATIONS

- Succinylcholine, a depolarizing muscle relaxant, is the most common agent used for intubation in both the controlled and emergency settings. Succinylcholine is the current favorite muscle relaxant for intubation because it has a rapid onset of 40 to 60 seconds and a short duration, lasting only 6 to 10 minutes. To understand the hyperkalemic response of Succinyl choline we shall touch upon the basics of neuromuscular transmission. The nerve synthesizes and stores acetylcholine in vesicles. Stimulation of the nerve mobilizes acetylcholine (Ach) to the cleft between nerve and muscle. Receptors in the endplate respond to Ach by opening channels for ion flow. When channels open, sodium and calcium flow from outside to inside while potassium flows from inside to outside. The net current is depolarizing thereby creating an endplate potential which in turn causes the muscle to contract. Succinyl choline acts in the same way as Ach but the ways in which the body gets rid of the drugs vary. Ach is immediately destroyed by acetyl cholinesterase to bring the junction to the resting state but Succinyl choline stays longer to be destroyed by plasma pseudo cholinesterase. Depolarization opens voltage gates to flow sodium ions. The time gate closes and this will open only when the voltage gate closes. Persistent depolarization causes voltage gates to open constantly so that the time gate never opens. This sets block in transmission of depolarization to the perijunctional area to cause a block. The above described classical path is only for junctional receptors. Normally a neural influence prevents the expression of extrajunctional receptors. When there is avulsion of a nerve, there is proliferation of extrajunctional receptors on the muscle membrane. These receptors are activated by lower concentration of agonists and ion flows are higher. Hence in bedridden patients and patients with stroke, spinal cord injuries amyotrophic lateral sclerosis, burns, extrajunctional receptors proliferate to get stimulated on Succinyl choline administration to cause more ion flows and consequent hyperkalemia. It gets a week for the development of extrajunctional receptors to form and it takes around 60 days for the healing of either a burn or muscle injury to get completed .This is the basis of avoiding succinyl choline in these potential days (1 week to 60 days) where there is a possibility of dangerous hyperkalemia. Hyperkalemia is reported in patients with renal failure and intra abdominal sepsis. The neuropathy associated with renal failure expresses the system to manufacture the extrajunctional receptors. In sepsis and some cases of burns there is impaired glucose tolerance and insulin resistance. This causes a decreased intra cellular glucose which in turn suppresses sodium pump of the cell. These events result in cells prone for potassium exuberance outward and subsequent hyperkalemia. To be precise, Succinyl choline causes a rise in serum potassium of about 0.5-1 mmol/l in normal patients within 1-7 minutes of injection. The rise can exceed 2 mmol/l in patients with massive trauma, burns, neurological lesions and certain myopathies. The hyperkalemia may last 10-15 minutes. Pretreatment with nondepolarizers decreases the response in some instances. Hence administration of Succinyl choline should be absolutely scientific in patients with possible electrolyte problems even though there are scant reports of safe use of succinyl choline in hyperkalemic patients.[67-71]
- Incidences of hyperkalemia have been reported after administration of hypertonic mannitol during craniotomy. Schwartz et al reported unsuspected hyperkalemia in children with pyloric stenosis coming for surgery.[72,73]
- Monitoring of electrolyte values, ECG, neuromuscular function, capnography are necessary additions. Hypokalemia and hypocapnia

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may prolong neuromuscular blockade.[74,75] Willibanks et al showed exaggerated neuromuscular blockade is possible in patients with hyperkalemia.[76] Liu et al reported a case of unsuspected intra operative hyperkalemia during cerebral angiography.[77] Naoki et al reported 6 patients which presented with perioperative hyperkalemia after stopping Inj. Ritodrine for premature labour. Intraoperave hypoventilation and acute respiratory acidosis can cause hyperkalemia but Natalini et al countered with reports that acute respiratory acidosis does not cause hyperkalemia in normokalemic anaesthetized patients. Hence a careful monitoring of electrolytes, ventilation and concomitant drugs used are mandatory.[78,79]

- Intravenous fluids with no dextrose are selected during surgery if hypokalemia is possible.
- In a study of 268 children undergoing liver transplantation, the incidence of intraoperative potassium (K+) disturbances and the risk factors for hypokalemia in the preperfusion and postreperfusion periods were studied. Overall, hypokalemia was the predominant disturbance, occurring in 72.0% of pediatric patients during liver transplantation. Hypokalemia was more common during the postreperfusion period than the prereperfusion period. Hyperkalemia, though a commonly cited complication, was infrequent during pediatric liver transplantation.[80]
- Tissue trauma like major surgery itself pushes potassium out of cells and hence in patients with multiple trauma and blood transfusion, monitoring of electrolytes should be done.
- It is better to avoid hypoventilation and hypoxia because accompanying acidosis may aggravate hyperkalemia.[81]
- Potassium is rarely used in combination with local anaesthetics for epidural and nerve blockades.[82]
- Elevations of pCO2 and plasma potassium have been reported after tourniquet release.[83]
- Transfusion: Serum potassium levels may be as high as 19-20 meq/l in blood stored for 21 days. Although hyperkalemia is occasionally reported, banked blood must be given at the rate of 120ml/ minute to produce significant hyperkalemia. Hence calcium administration is warranted only on diagnostic signs of hyperkalemia like peaked T waves on electrocardiogram.[84]
- Epinephrine used along with local anaesthetics during administration of blocks may cause hypokalemia.[85]
- Hypokalemic periodic paralysis is a familial autosomal dominant trait. The paralytic attacks are precipitated by large carbohydrate-rich meals, cold, mental or surgical stress, infections, exercise, drugs, electrolytes and endocrine abnormalities. Death may occur from respiratory arrest, infections, aspiration or cardiac arrhythmias. Anesthesia and surgical procedures may induce an attack and complicate the perioperative patient condition. Guidelines for anaesthetic management should include preventive measures i.e. reduce mental stress and carbohydrate intake and correction of electrolytes and endocrine abnormalities. During the operation, measures should include maintanance of ideal temperature, reduction of stress and monitoring of muscle relaxants and ECG. When paralysis is diagnosed, slow (30-40 mEq/hr) i.v. infusion of potassium is suggested, while monitoring plasma levels of potassium, ECG, and facial nerve conduction. Intravenous anaesthesia, intravenous regional anaesthesia and spinal anaesthesia have been successfully used while careful and monitored general anaesthesia is also acceptable.[86,87]
- Anaesthesia in patients with Hyperkalemic periodic paralysis was associated with perioperative paralytic episodes. But some measures like preoperative potassium depletion, avoidance of carbohydrate depletion, maintenance of normothermia and avoidance of potassium releasing drugs can be strictly followed for the smooth conduct of anaesthesia in patients with Hyperkalemic periodic paralysis.[88]

POSTOPERATIVE CONSIDERATIONS

- Postoperative ileus is seen after laparotomies the common culprit being hypokalemia. Hence

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monitoring and supplementation of necessary potassium is to be considered in all patients with abdominal surgeries.[89]

- Acute tubular necrosis is commonest cause of post surgical oliguria. Appropriate fluid management and treatment of hyperkalemia is often necessary in the recovery room. Usually dialysis is not needed.[81,90]
- Reports of postoperative hyperkalemia are reported after removal of aldosterone producing adenomas.[91]
- Postoperative use of Non steroidal anti-inflammatory drugs should be scientific in selected patients.[92]

POTASSIUM AND CARDIOPULMONARY BYPASS:[93-96]

Myocardial protection and preservation during CPB forms the essence of success of cardiac surgeries in many instances. Cold cardioplegic myocardial protection is the commonest technique used. The goals of cardioplegia are

- Immediate and sustained electromechanical quiescence.
- Rapid, homogenous and sustained cooling.
- Maintenance of therapeutic additives in effective concentrations.
- Periodic washout of metabolites.

Even though there are numerous described methods producing cardioplegia, the most common agent used to produce electromechanical quiescence is potassium. The principal effects of increased extra cellular potassium are complete diastolic arrest, inhibition of spontaneous phase-4 depolarization in pacemaker cells and maximal energy conservation.

The mechanism is as follows:

The resting membrane potential of the myocardial cell is largely determined by the trans membrane potassium gradient. When the gradient is decreased by elevated extra cellular potassium, membrane potential becomes less negative. (More positive) During membrane depolarization from a less negative (e.g. – 50 instead of – 90) the inward sodium currents are slowed to an extent of almost inactivation. Hence an action potential is not propagated. The ideal potassium concentration is 30-40 mEq/l. The potassium concentration of more than 50 mEq/l tends to open calcium channels to produce a calcium influx and consequent myocardial contraction. This effect increase wall tension and consequent higher energy utilization. Additions of beta blockers, calcium channel blockers to cardioplegia solutions have been successfully used. Steroids and prostaglandins as an adjunct to myocardial protection have conflicting evidences.

The electrolyte changes concerning potassium during CPB may be either hypokalemia or hyperkalemia. Hypokalemia may be due to hemodilution with non potassium containing priming fluids or large doses of diuretics. This may be corrected with increments of 5-10 mEq of KCl over 1-2 minute interval directly given into the pump/oxygenator by the perfusionist and serum values may constantly monitored. Hyperkalemia may be due to systemic uptake of potassium containing cardioplegia, hemolysis, tissue damage and acidosis. Routinely described methods can be used to lower potassium if warranted. Potassium is the critical ion that may present acute changes during CPB. Others ions excluding calcium rarely show significant changes and their correction is less demanding for the weaning to take place. Potassium has pronounced effects on cardiac conduction, and hyperkalemia may result in atrioventricular block. After adoption of blood cardioplegia a higher potassium level is more commonly seen at the end of perfusion. In the presence of a normal renal function, a mild hyperkalemia represented by a serum level of 6 mEq/L will not require special treatment and resolve spontaneously. If a higher potassium level exists in the presence of a heart block or bradycardia a more regular rhythm should be secured with temporary pacemaker wires and the hyperkalemia should be treated with insulin, glucose and furosemide .Hypokalemia if present should be treated to avoid the risks of atrial and ventricular arrhythmias. Hence during CPB it is preferable to monitor potassium and institute appropriate corrective measures for successful weaning from CPB. The preoperative correction of potassium before cardiac surgery resulted in better outcomes in

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cardiac surgery. Glucose Insulin potassium infusion might be beneficial in patients with decreased cardiac reserve and poor LV function. A recent study by Lazar et al found better cardiac performance after bypass grafts in a group of forty diabetic patients but others suggested that GIK infusion may not decrease the myocardial damage during bypass graft surgery. Variable results are available with the use of Glucose Insulin potassium infusion for heart protection during cardiac surgery.

To conclude, potassium, a major intra cellular cation is intimately involved in the maintenance of resting membrane potential. Hypo or hyperkalemia can cause disturbances of muscle, nerve and cardiac cell excitability. These three excitable cells are closely associated with anaesthesia. The absolute levels of potassium, presence of symptoms, acute or chronic disturbance and associated risk factors should be borne in mind before perioperative correction of serum potassium levels.

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