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Hyperkalaemia: causes, electrocardiographic changes and management

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ABSTRACT Hyperkalaemia is an ever present threat in dialysis units and is encountered frequently on general wards, particularly in patients with renal failure. It is important because it can cause life threatening cardiac dysrhythmia, with death by asystole or ventricular fibrillation commonly the first clinical manifestation.1 Preferred therapy varies, reflecting the lack of clear evidence for an optimal regimen, but broadly speaking the choice of treatment is based upon the serum potassium level, the ECG and the likelihood that potassium will rise further. In this review we highlight the causes, sequelae and evidence for the treatment of hyperkalaemia. Our starting point is a serum potassium of 6 mmol/l. Thereafter, we suggest it may be useful to distinguish two groups of patients on the basis of their ECG: those whose ECG is normal, whom we consider to have severe hyperkalaemia, and those with ECG evidence of raised serum potassium or life threatening hyperkalaemia.

KEYWORDS Acute renal failure, causes of hyperkalaemia, chronic renal failure, electrocardiographic changes, hyperkalaemia, management of hyperkalaemia

LIST OF ABBREVIATIONS Acute renal failure (ARF), chronic renal failure (CRF), dual mode, dual pacing, dual sensing (DDD), electrocardiogram (ECG), Randomised Aldactone Evaluation Study (RALES)

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POTASSIUM HOMEOSTASIS

Factors influencing potassium homeostasis are shown in Figure 1. Dietary potassium intake is generally 50–100 mmol/24 hours. Normal serum potassium levels are between 3·5 and 5·2 mmol/l. Total body potassium is about 50 mmol/kg or approximately 3,500 mmol for a 70 kg adult. Ninety-eight per cent of this is intracellular where the concentration is around 150 mmol/l. The extracellular/intracellular gradient is maintained by the Na–K ATPase pump, the activity of which is regulated by beta 2 adreno-receptors, insulin and acid-base status. Ninety to ninety-five per cent of potassium losses occur through the kidney under the influence of aldosterone, the remainder in faeces. Importantly, in renal failure, gut elimination is increased and then accounts for up to 25% of daily potassium losses, unless constipated.[2]

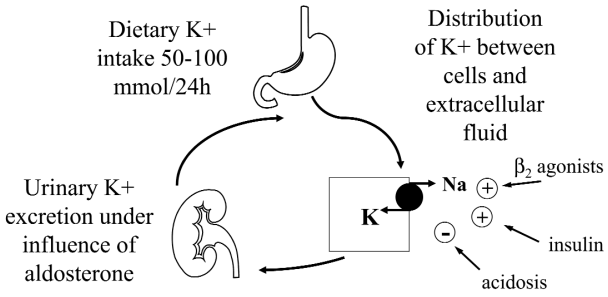

FIGURE 1 Potassium homeostasis showing dietary intake, distribution of potassium between cells and extra cellular fluid, and urinary potassium excretion under influence of aldosterone.

CAUSES OF HYPERKALAEMIA

These may be derived from the potassium homeostasis diagram (see Figure 1). Common causes of hyperkalaemia are given in Table 1. This list is not intended to be exhaustive. It does not include the common artefactual hyperkalaemia that occurs when there is a delay between venepuncture and separation of serum in the laboratory, or rare inherited disorders such as Gordon's syndrome or hyperkalaemic periodic paralysis. Hyporeninaemic hypoaldosteronism is not uncommon in elderly type 2 diabetics with mild renal insufficiency whose serum potassium appears disproportionately high to the level of serum creatinine. Hyporesponsiveness to both renin and aldosterone secretion can be demonstrated and leads to hyperkalaemia. The treatment of choice is frusemide.[3] Medical students and junior doctors may find it helpful to remember that many of the important causes of hyperkalaemia begin with A: Acute renal failure, Addison's disease, Acidosis, Artefact, ACE inhibitors, Angiotensin receptor blockers, Aldosterone antagonists, Anti-inflammatory drugs, Added K.

TABLE 1 Common causes of hyperkalaemia.

High potassium load*
- High potassium foods.
- Potassium supplements.
- Salt substitutes containing potassium.
- Upper gastrointestinal bleed.
- Large haematoma.

Redistribution of potassium between cells and extracellular fluid
- Acidosis.
- Tissue necrosis including crush injuries, rhabdomyolysis, burns, tumour lysis.
- Drugs including digoxin intoxication, betablockers.

Impaired renal excretion
- Acute and chronic renal failure.
- Addison's disease.
- Hyporeninaemic hypoaldosteronism.
- Drugs including ACE inhibitors, angiotensin receptor blockers, non steroidal anti-inflammatory drugs, potassium sparing diuretics, cyclosporin, tacrolimus, trimethoprim, heparin.

*High potassium load does not cause hyperkalaemia unless renal excretion of potassium is impaired.

Drug induced causes of hyperkalaemia merit special consideration. Patients taking ACE inhibitors and angiotensin receptor blockers for whatever reason can become severely hyperkalaemic with acute renal failure during intercurrent illnesses, characterised by severe dehydration, especially with vomiting and diarrhoea.[4, 5] Severe hyperkalaemia in patients with heart failure who are taking spironolactone and ACE inhibitors or angiotensin receptor blockers is more likely with advanced age, doses of spironolactone more than 25 mg per day, reduced renal function at start of therapy and type 2 diabetes.[6] Increasing use of spironolactone following the publication of the RALES has led to a measurable rise in hyperkalaemic related deaths.[7] Patients co-prescribed renin angiotensin system blockers, potassium sparing diuretics and non steroidal anti-inflammatory drugs may be particularly likely to develop hyperkalaemia – the so called 'triple whammy'.[8] Hyperkalaemia is a feature of digoxin intoxication because digoxin blocks the Na–K ATPase pump on cell membranes. Other drugs that may cause hyperkalaemia include betablockers, the calcineurin inhibitors cyclosporin and tacrolimus, trimethoprim, heparin, and of course, K supplements.

The most important causes of hyperkalaemia are ARF and CRF. Hyperkalaemia in a setting of ARF poses the greater threat to life particularly if the patient is oligo-anuric and hypercatabolic, circumstances which mean that potassium is only likely to rise further. Hyperkalaemia in a patient with stable CRF more often represents a steady state and need not trigger quite such an energetic treatment response, provided of course that there are no life threatening features on the ECG. This is not to imply that hyperkalaemia in CRF can be ignored, merely to state that a serum potassium of 6·5 mmol/l in an oliguric, hypercatabolic patient with ARF is likely to be more dangerous than the same level of serum potassium in a patient with stable chronic renal failure.

SYMPTOMS AND ECG CHANGES

Hyperkalaemia is usually asymptomatic until severe when the patient may complain of paraesthesias or weakness, progressing in extreme cases to a flaccid paralysis. Death by asystole or ventricular fibrillation is commonly the first clinical manifestation. The ECG is the single most important investigation in hyperkalaemia and will usually give important clues that the patient is at risk of cardiac arrest. T waves that are taller than the R wave in the same lead, have a narrow base and a shortened QT interval, are the earliest change and may occur when serum potassium exceeds 5·5 mmol/l.[9] As the serum potassium concentration increases further, P waves flatten and then eventually disappear. Between 6·5 and 8 mmol/l the QRS complex widens (see Figure 2A). These changes become progressively more common as the serum potassium rises further but are not always present even in severe hyperkalaemia.[10] The final changes may include bradycardia leading to asystole, or a sine wave pattern in which the widened QRS complex merges with the T wave,[11] or ventricular fibrillation. Ventricular tachycardia can occur in hyperkalaemia but is more often associated with hypokalaemia. Pulseless electrical activity has also been reported.[12]

A difficulty for clinicians is that it is possible to have severe hyperkalaemia without ECG changes[13, 14] and for the changes of hyperkalaemia to mimic other ECG morphologies. For example, the broad QRS complex may bear a superficial resemblance to bundle branch block. A patient with no P waves, a broad QRS complex and a tachycardia may appear to have ventricular tachycardia,[9] whereas if the rate is slow, as is more commonly the case, the appearances may be confused with complete heart block.[15] True second and third degree atrioventricular block have been described in hyperkalaemia,[16–18] but are less common because the P wave usually disappears before such advanced AV block occurs. A bizarre form of AV block has been recorded in a patient with hyperkalaemia and a DDD pacemaker.[19] A pseudo myocardial infarction pattern with ST elevation in the anteroseptal leads has also been described.[20, 21]

TREATMENT OF HYPERKALAEMIA

Given the magnitude of the threat to life, and the fact that several different treatments are available, it is perhaps surprising that the first systematic review of the treatment of hyperkalaemia was not published until

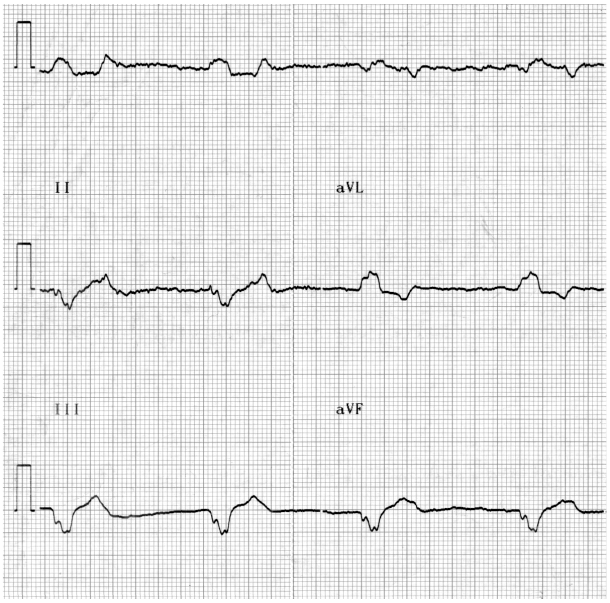

FIGURE 2A Electrocardiogram of 72-year-old woman who presented with diarrhoea and vomiting while on an ACE inhibitor. Serum potassium was 8·5 mmol/l with blood urea 21·0 mmol/l and serum creatinine 625 umol/l. The bradycardia, absent P waves and broad QRS complexes all suggest a diagnosis of life threatening hyperkalaemia.

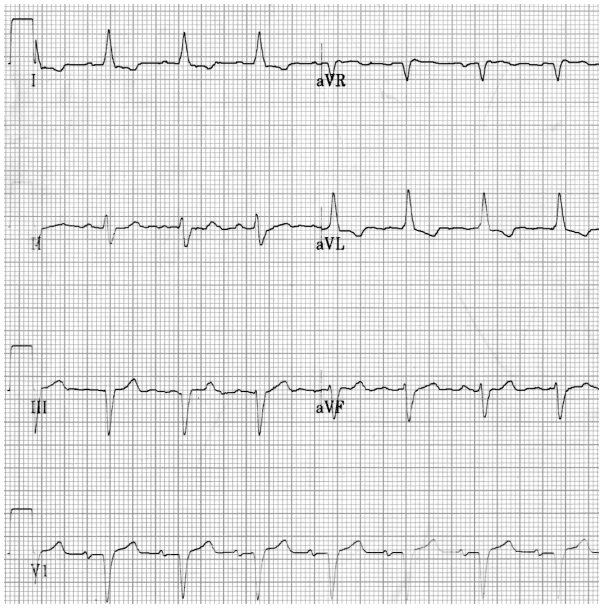

FIGURE 2B The QRS complex narrowed and P waves returned after 10 mls calcium chloride. Serum potassium fell to 5·2 mmol/l following insulin with dextrose, nebulised salbutamol, sodium bicarbonate, then emergency haemodialysis. At clinic review 2 months later, blood pressure was 142/78 with serum potassium 3·8 mmol, blood urea 4·9 mmol/l and serum creatinine 111 umol/l.

2005.[22] Using this and other sources,[1, 2, 23, 24] it is evident that the emergency management of hyperkalaemia can be considered under three headings (see Figure 3). It is clearly important to stop taking the offending drugs and treat the underlying disorder, but these measures may take some days to lower serum potassium and are not a substitute for the emergency treatment we describe below. It should also be borne in mind that serum potassium may rebound as the effects of the emergency measures wear off.[2] It is therefore essential to monitor the serum potassium after initial treatment, to consider the cause of hyperkalaemia and address all precipitating factors in order to prevent recurrence.

1. REDUCE RISK OF ARRHYTHMIA

Intravenous calcium

Calcium acts directly to antagonise the membrane actions of potassium but not to lower the serum level. It is rapidly effective over 1–5 minutes and has a duration of action of approximately one hour.[25] Calcium should definitely be given whenever hyperkalaemia is associated with bradycardia, absent P waves or broad QRS complexes, and probably also in the presence of tall T waves,[23] but is not indicated in the absence of ECG changes. Calcium chloride 10% contains more calcium (6·8 mmol in 10 ml) than calcium gluconate 10% (2·2 mmol in 10 ml) and is preferred for this reason. 10 ml of 10% calcium chloride should be given intravenously over 5 minutes using the largest available vein as extravasation may cause tissue necrosis. This can be repeated every 2–3 minutes until the bradycardia resolves, P waves return, and the QRS complex narrows, to a maximum of 30 or 40 ml (see Figure 2B). Caution should be exercised in the digitalised hyperkalaemic patient as calcium administered too quickly in the setting of digoxin toxicity may induce arrhythmia or cardiac arrest.[26] 10 mls 10% calcium chloride made up to 100 mls with 5% dextrose and infused over 30 minutes is recommended in patients taking digoxin for this reason.[26]

2. DRIVE K^+ INTO CELLS

Intravenous insulin with dextrose

Insulin with dextrose has long been used in the treatment of hyperkalaemia. Insulin drives potassium into the cells while the glucose prevents hypoglycaemia. Insulin causes a fall in serum potassium of 0·5–1 mmol/l that begins within minutes and peaks at 30–60 minutes.[27] Ten units of soluble insulin with 50 ml 50% dextrose over 5–10 minutes intravenously is the usual regimen. This can be followed, if necessary, by a continuous infusion of 10 units soluble insulin in 500 ml 10% dextrose at a slower rate. Combination of insulin with nebulised salbutamol leads to a greater and more prolonged fall in serum potassium of 1·0–1·5 mmol/l, peaking at 1–3 hours.[28] It must be emphasised that these measures only buy time until a means of eliminating potassium from the body can be established; also that blood glucose should be checked every 15 minutes for a minimum of 2 hours.

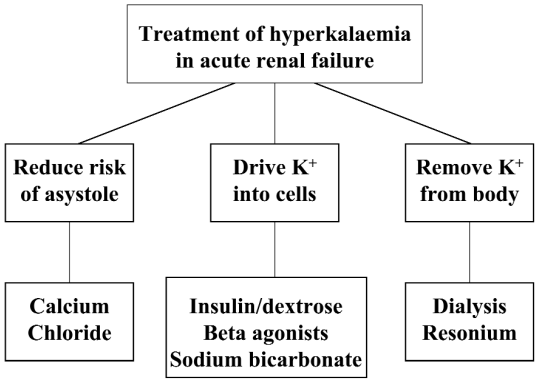

FIGURE 3 Principles of treatment of hyperkalaemia in acute renal failure.

Hypoglycaemia is not uncommon after insulin and dextrose, particularly in patients with renal failure,[29] but is attenuated by co-administration of salbutamol.[30]

Nebulised salbutamol

This is usually given concurrently with IV insulin and not on its own as up to 40% patients are unresponsive to salbutamol alone.[27] The dose required to drive potassium into cells is greater than the dose used for acute severe asthma. Salbutamol is usually well tolerated but should be used with caution if tachycardic. Ten milligrams of nebulised salbutamol will lower serum potassium from around 30 minutes with peak reduction 0·5 mmol/l at 1–2 hours. A fall of around 1 mmol/l can be achieved with 15–20 mg nebulised salbutamol[31, 32] (see Figure 4). Five hundred micrograms of intravenous salbutamol can be given with similar effects[33] but nebulised therapy is much easier to administer in the emergency setting.

Intravenous sodium bicarbonate

Bicarbonate causes hydrogen ions to be released from cells and potassium ions to move into cells to maintain electro-neutrality, but should never be used as sole therapy for hyperkalaemia as it is not particularly effective. Infusion of 8·4% bicarbonate at 4 mmol/min for one hour, followed by 1·4% at 0·5 mmol/min for 5 hours (a total of 390 mmol) in 12 mildly acidotic CRF patients whose serum bicarbonate was 17·5 mmol/l and serum potassium 6·04 mmol/l, led to a fall in serum potassium of less than 0·3 mmol/l at 2 hours.[34] Benefits may be greater in patients who are more acidotic than this,[35] and there is some evidence that sodium bicarbonate potentiates the action of insulin on serum potassium.[36] The usual dose is 500 ml of 1·26% sodium bicarbonate (75 mmol) IV over 60 minutes, which is considerably less than that given in the trials. Sodium bicarbonate should not be administered through the same line as calcium chloride as a precipitate of calcium carbonate may

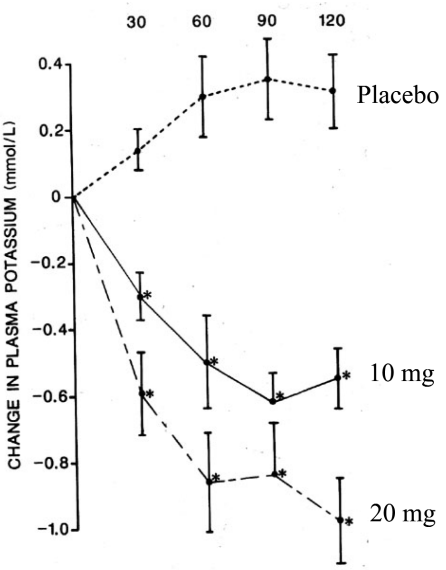

FIGURE 4 Fall in serum potassium between 30 and 120 mins after 10 and 20 mg nebulised salbutamol.[31]

form. Unless the patient has already arrested, 8·4% bicarbonate should be avoided in order to reduce the risk of tissue necrosis from extravasation.

3. REMOVE K^+ FROM BODY

Dialysis

The most effective ways of removing potassium from the body in an anuric catabolic patient are haemodialysis or haemofiltration. These are the treatments of choice in life threatening hyperkalaemia after first protecting the heart and then driving potassium into cells. Haemodialysis is preferred to peritoneal dialysis as it removes potassium more rapidly, unless of course the patient is already on peritoneal dialysis. Typically when dialysing against a 1 mmol/l K^+ bath, serum potassium will fall by 1 mmol/l in the first 60 minutes and by a further 0·5 mmol/l in the next 2 hours.[37] More potassium is lost by using potassium free dialysate[37] and at higher blood flow rates.[38]

Cation exchange resins

These are cross-linked polymers that exchange calcium for potassium across the gut wall. They do not bind dietary potassium and do not therefore require that a patient be eating in order to be effective. The evidence of benefit is not strong, however. No differences in serum potassium were observed at four hours when a single dose of resin was compared with placebo.[39] Two studies have reported around 0·5 mmol/l reduction in serum potassium at 24 hours, but the extent to which this was dependent on the induction of diarrhoea by the co-prescription of laxatives was unclear.[40, 41] Subsequent work has suggested that most potassium losses are due to the

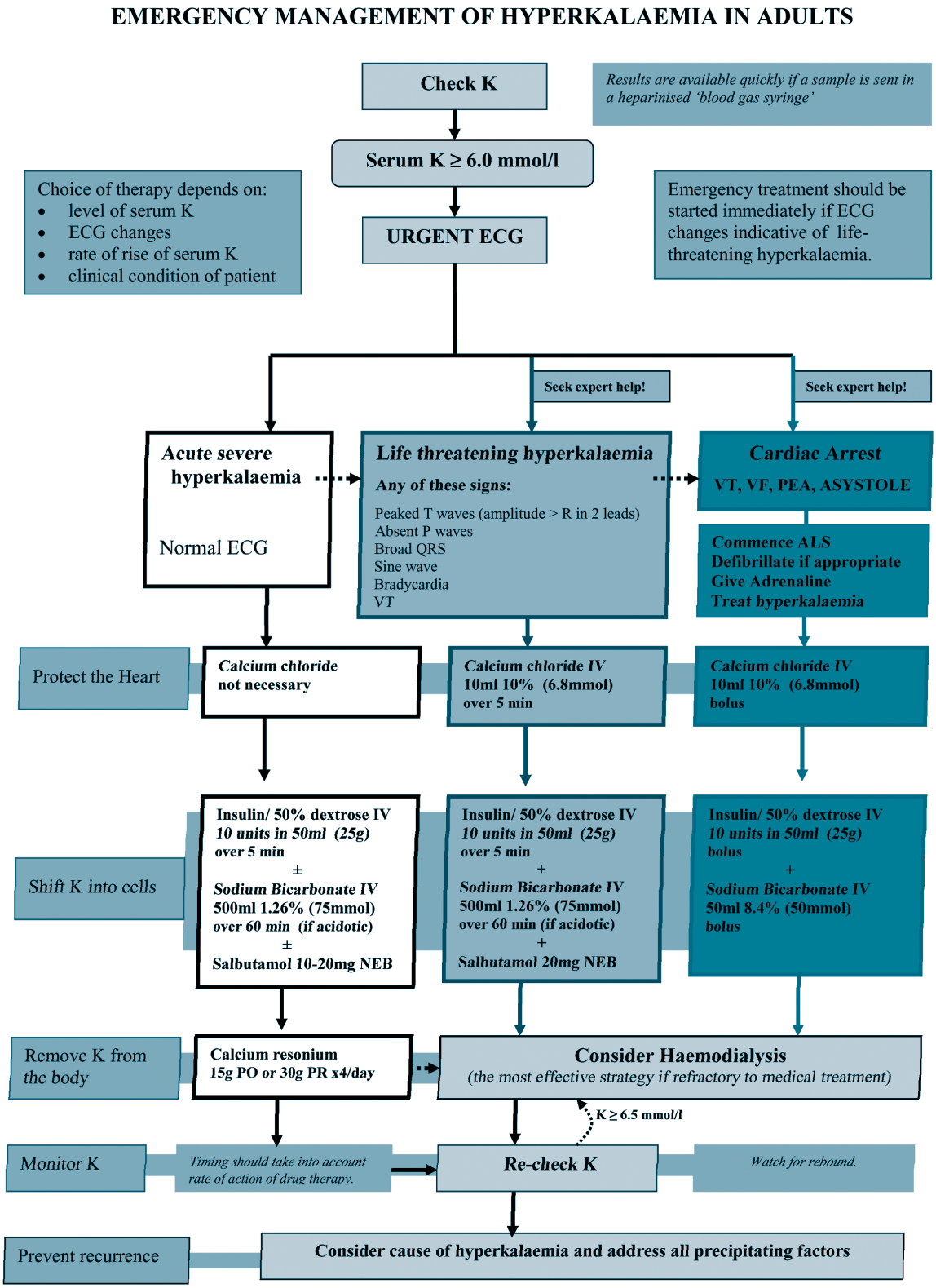

FIGURE 5 Treatment plan for adult patients with severe and life threatening hyperkalaemia.

TABLE 2 Recommended drug doses.

Calcium chloride
10 ml 10% IV over 5 minutes, repeated every 2–3 minutes until QRS complex narrows or bradycardia resolves. Rapidly effective with duration of action around one hour. Calcium protects heart but does not lower serum potassium. Gluconate is acceptable if chloride is not available but contains three times less calcium, so adjust dose. Give calcium speculatively if ECG is available before serum K and shows any form of arrhythmia (bradycardia or tachycardia) with absent P wave and broad QRS complex. Risk of tissue necrosis with extravasation. Infuse over 30 mins if patient is taking digoxin. Never give calcium and bicarbonate through the same access as risk of precipitation.

Insulin with dextrose
10 units soluble insulin with 50 ml 50% dextrose over 5 minutes will lower potassium by about 1 mmol/l. Effect peaks at 30–60 mins and lasts 4–6 hours. Can cause hypoglycaemia so check glucose every 15 mins. Insulin and dextrose can also be infused by adding 10 units actrapid to 500 ml 10% dextrose if necessary.

Salbutamol
10–20 mg nebulised salbutamol will lower potassium by about 1 mmol/l. Effect peaks at 1–2 hours and lasts 4–6 hours. Not recommended as monotherapy as up to 40% patients are unresponsive to salbutamol alone. A good addition to insulin/dextrose if patient has bradycardia. Avoid or reduce dose in presence of tachycardia.

Sodium bicarbonate
500 ml 1·26% sodium bicarbonate over 30–60 mins if venous bicarbonate <15 mmol/l, but not as monotherapy. May lower potassium by up to 0·5 mmol/l at around 2 hours and lasts 4–6 hours. 1·26% preferred, as risk of tissue necrosis with 8·4% sodium bicarbonate. Never give bicarbonate and calcium through same access as risk of precipitation.

Calcium resonium
15 g qds orally with senna and lactulose, or 30 g qds rectally provided this is washed out by colonic irrigation between doses. Will lower potassium by around 0·5 mmol/l starting after 4 hours with peak effect at 24 hours. Not recommended for acute hyperkalaemia given delayed onset of action but may have a limited role in chronic hyperkalaemia.

induction of diarrhoea.[39] Resins are not recommended for acute hyperkalaemia given their delayed onset of action, but may have a limited role in chronic hyperkalaemia. The usual regimen is Calcium Resonium 15 g three or four times a day by mouth with senna and lactulose to prevent constipation/induce diarrhoea. This drug can also be given as a 30 g enema in patients who are nil by mouth. Resonium must be washed out by irrigation using a rectal catheter between doses. The use of cation exchange resins and other measures in the management of hyperkalaemia is summarised in Figure 5.

Intravenous saline

We mention this for completeness. Although dextrose is required to reduce risk of hypoglycaemia when insulin is given to drive potassium into cells, intravenous saline is preferred to dextrose as replacement fluid for hyperkalaemic patients who are passing urine and are not volume overloaded. Potassium excretion takes place at the distal tubule in exchange for sodium, and in the setting of saline depletion insufficient sodium may reach the distal tubule for this to occur. Similarly, intravenous frusemide 40–80 mg can be used to promote kaliuresis in patients who are still passing urine by blocking the reabsorption of sodium in the thick ascending limb of Henle. Following administration of frusemide, more potassium is exchanged for sodium in the distal tubule.

TREATMENT OF HYPERKALAEMIA DURING CARDIO-PULMONARY ARREST

Figure 5 also gives recommendations for the emergency management of hyperkalaemia during cardio-pulmonary arrest. This may be necessary when cardio-pulmonary arrest follows unsuccessful treatment of known hyperkalaemia or when unsuspected hyperkalaemia is discovered during resuscitation. The advice differs only slightly from that of life threatening hyperkalaemia pre-arrest. Adrenaline 1 mg IV should be given for its powerful beta adreno-receptor agonist activity, together with calcium chloride 10% 10 ml IV. 50 ml 8·4% sodium bicarbonate should be given on the grounds that the benefits of the concentrated preparation are likely to out-weigh the risk. Complete recovery after hyperkalaemic arrest following cardio-pulmonary resuscitation of up to 3·5 hours duration has been recorded.[42, 43] The treatment of hyperkalaemia during cardio-pulmonary arrest is reviewed in more detail elsewhere.[26, 44]

SUMMARY OF TREATMENT RECOMMENDATIONS

It is not easy to provide hard and fast rules for treating hyperkalaemia, because the threshold for intervention and the choice of therapy varies with the clinical circumstances. Thus it is not only the actual level of serum potassium that triggers a therapeutic response, but also the ECG changes that are present and the likelihood that serum potassium will rise further. We believe it may be useful to distinguish severe hyperkalaemia from life threatening hyperkalaemia, in the same way that it helps to distinguish severe asthma from life threatening asthma when managing patients who present with an acute exacerbation of their airways disease. The most important investigation in the assessment of hyperkalaemia is the electrocardiogram. The presence of ECG changes suggesting hyperkalaemia should lead to the immediate administration of calcium chloride in the same way that an asthmatic who stops wheezing should trigger

a call for urgent help from the anaesthetist. Figure 5 suggests a treatment plan for patients with severe and life threatening hyperkalaemia, particularly when these are associated with renal failure, and Table 2 summarises the different drugs used and their doses.

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