The Shrewsbury and Telford Hospital NHS Trust

Management of Hyperkalaemia

Clinical Guide

Ref. No. 2197

Additionally refer to:

SaTH DKA MANAGEMENT PATHWAY JUNE 2010

SaTH Guideline for the Management of Diabetic Ketoacidosis

SaTH Acute Kidney Injury (AKI) Quick reference guide (Oct14)

SaTH Acute Kidney Injury (AKI) Pathway for Adult Patient

SaTH Cardiopulmonary Resuscitation Policy (CG22)

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Version history

Version	Date	Author	Status	Comment – include reference to Committee presentations and dates
V1	20.05.2015	Craig Lammas	Draft	28.05.2015 Circulation of policy (see additional SaTH circulation checklist) 18.06.2015 Amendments made, endorsement by Safe medicines Group 17.06.2015 01.07.2015 appendix A5 updated with new dietary advice 20.09.2015 additional hyperlink to treatment algorithm to inserted on pg4
V1.1	23/05/2019	Robin Long		08/08/2019 Patient Safety Alert NHS/PSA/RE/2018/006 Alert presented at Medicines Management Governance and Unscheduled Care Group Medical Governance. No policy changes required.
V1.2	06/04/2021	Suresh Ramadoss		5.2.5 added - Sodium Zirconium Cyclosilicate and Patiromer, two new oral potassium lowering drugs Appendix 1 updated (Emergency management of Hyperkalaemia in Adults) Nebulised salbutamol dose changed from 10-20 mg to 10mg in severe hyperkalaemia

Contents

1 Document Statement

Clinical staff need to know how to recognise and promptly manage acute hyperkalaemia in an Adult patient (inpatient and outpatient) as delay can lead to the development of cardiac arrhythmias and subsequence cardiac arrest

2 Overview

This policy focuses on the recognition and emergency treatment of acute hyperkalaemia in adults in secondary care settings. It is applicable to clinicians in all specialties.

This guideline does not comprehensively cover the treatment of hyperkalaemia in;

i. ***The management of hyperkalaemia in Diabetic ketoacidosis (please refer to Diabetes mellitus – DKA Pathway)***

ii The management within out-patient or primary care settings.

3 Definitions

3.1 Cardiac arrhythmias

Also known as cardiac dysrhythmia, is a problem with the rate or rhythm of the heartbeat, it may be too fast, too slow or irregular.

3.2 Cardiac Arrest

Cardiac arrest is the sudden cessation of mechanical cardiac activity, confirmed by unresponsiveness, apnoea or agonal gasping respiration, and the absence of a detectable pulse.

3.3 Haemodialysis

A medical procedure to remove fluid and waste products from the blood and to correct electrolyte imbalances. This is accomplished using a dialysis machine

3.4 Haemofiltration

The patient's blood is passed through a set of tubing (a filtration circuit) via a machine to a semipermeable membrane (the filter) where waste products and water (collectively called ultrafiltrate) are removed by convection. Replacement fluid is added and the blood is returned to the patient

3.5 Hyperkalaemia

There is no universal definition of hyperkalaemia, but a serum K+ ≥ 5.5 mmol/L is widely used

3.6 Pseudohyperkalaemia

A spurious elevation of the serum concentration of potassium occurring when potassium is released in vitro from cells in a blood sample collected for a potassium measurement. This may be a consequence of disease or as a result of improper collection technique

4 Duties

All clinical staff need to know how to recognise and promptly manage acute hyperkalaemia in an Adult patient

5 Policy detail (follow hyperlink below for summary treatment algorithm)

A1 Renal Association Hyperkalaemia Treatment Algorithm

5.1 Assessment

Is the Result correct?

Is the result unexpected or an isolated finding? To exclude pseudohyperkalaemia (See Appendix 2 for causes) promptly assess the patient

(ABCDE approach), review prior blood results and arrange repeat serum potassium sampling.

To aid prompt clinical decision making and treatment the European Resuscitation Council have stratified hyperkalaemia as;

Mild (5.5-5.9 mmol/L),

Moderate (6.0-6.4 mmol/L)

Severe (≥6.5 mmol/L)

The UK Renal Association and Resuscitation Council also endorse these range values

ALL patients with potassium (K+) greater than 6.0mmols MUST have an urgent 12lead ECG and signs of hyperkalaemia excluded

Typical features of hyperkalaemia include:-*

Tented T waves
Prolonged PR interval
Loss of P Waves
Broadening QRS complexes
QRS complexes merging with T waves

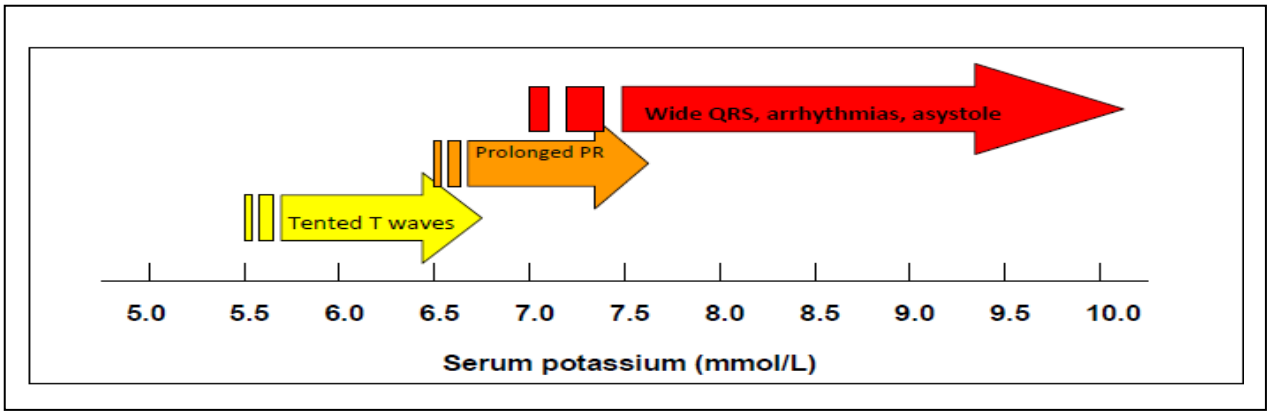

Figure 1 Progressive changes in ECG with increasing severity of hyperkalaemia
(Re-produced from Clinical practice guideline treatment of acute Hyperkalaemia in Adults – UK Renal Association 2014)

****Severe hyperkalaemia can still be present in patients without obvious ECG changes.***

ECG changes may be also be minimal in haemodialysis patients and atypical and more suggestive of myocardial ischaemia in Diabetic Ketoacidosis (1)

Patients with pacemakers there may be widening of the paced QRS complex and increased atrial and ventricular pacing thresholds with or without increased interval between the pacemaker stimulus and the onset of the paced beat. (1)

Where ECG changes are evident, emergency treatment should be initiated, even without confirmation of the repeat serum potassium result. In parallel to the emergency treatment consider point-of care blood gas analysis measurement via arterial/venous sampling

For example ECG changes please refer to Appendix 7

5.2 Treatment (K+ >6.0mmols) – Protect - Shift – Remove (See appendix 1 for Algorithm)

5.2.1 Protect the Cardiac membrane (use of IV Calcium)

Hyperkalaemia decreases the resting membrane potential of the myocardial cells, thus they are more likely to spontaneously depolarise and trigger arrhythmias.(2) To counter and antagonise the depolarisation effect Intravenous calcium salts should be administered thus prompt IV cannulation of a large vein should be secured.

10mls 10% IV Calcium Chloride
OR
30mls 10% IV Calcium Gluconate should be administered over 5-10minutes

Once administered onset of effect should be within 3minutes and have a lasting duration of approximately 30-60minutes.

Should there be no sign of changes in the ECG features of hyperkalaemia after the first initial dose a repeat dose should be considered within 5-10minutes until the ECG is normalising.

NB Calcium Gluconate may require even further sequential boluses, continued every 10minutes until a normalising ECG is observed.

Intravenous Calcium is only 'buying time' and IS NOT treating the underlying hyperkalaemia

5.2.2 Potassium Shifting (Insulin & Glucose)

Through activating increased ATPase activity at the cell membrane, insulin increases the movement of potassium ions away from the bloodstream and into the cells. (3) However, the sole administration of Insulin to a patient with normal glucose levels would in most instances induce hypoglycaemia. ***Thus unless known hyperglycaemia (>15mmols) the insulin should always be co-administered with hypertonic glucose as follows:-***

10units of insulin (actrapid ®) + 50mls 50% glucose administered over 15-30minutes
The Renal Association and the Resuscitation Council (UK)

Onset of action occurs within 15minutes with a peak reduction of 0.6 – 1.0mmols/L potassium being observed at 60minutes.

With the administration insulin hypoglycaemia is a significant risk, renal patients especially can experience delayed hypoglycaemia up to 6hrs after an infusion therefore blood glucose monitoring should be closely monitored within the 6hour period (See refer to Appendix 1 treatment algorithm).

5.2.3 Potassium Shifting (Beta-antagonist – Salbutamol)

The b-2 adrenoceptor agonist Salbutamol also stimulates ATPase activity thereby contributes to the shifting of potassium into cells.

Unfortunately, in some patient groups (end stage renal disease and patients on beta-blockers) it is evidently ineffective so it should only be considered an adjunct therapy in the management of hyperkalaemia. (1)

Used in conjunction with insulin + glucose administration efficacy of potassium shifting is increased.

The nebulised route is the easier and safer route of administration

In moderate hyperkalaemia consider use based again ECG changes and rate of K+ rise

In severe hyperkalaemia give - 10 mg of nebulised salbutamol

The effect of salbutamol is dose-dependent, but its onset of action is within 30minutes with peak effect within 60minutes.

Caution and cardiac monitoring should be observed in patients with ischaemic heart disease and/or tachyarrhythmias, the lower 10mg dose is also recommended.

The administration of insulin + glucose and nebulised salbutamol may lower the K+ within the first 30-60minutes and may have a lasting effect for 4-6hrs they *may not*. It is therefore imperative to repeat serum K+ sampling within the first 2hours to determine a reduction has occurred.

A 'rebound' elevation of K+ levels is often observed 4-6hrs thus further serum sampling is required at these times. (See appendix for algorithm)

5.2.4 Potassium Removal

Calcium salts/insulin-glucose/beta-2 agonists are not definitive therapies - they simply buy time for re-evaluation and definitive removal therapy

There may be a combination of factors contributing to the hyperkalaemia; there may be underlying hypoxia, cardiac failure, chronic kidney disease (CKD), Acute Kidney injury (AKI) and/or underlying Sepsis. It may be as a result of prescribed drugs or an interaction between them, so after initial protection and shifting a prompt and thorough clinical assessment and review of the patient, their medical and drug history should be undertaken. (See appendix for precipitating factors)

Advice should be sought early from the on call Consultant (Telephone through switchboard) / ITU if K+ is >6.5mmols and/or the cause or condition despite initial treatment may require dialysis (haemodialysis/haemofiltration) or further organ support.

i. Stop any drugs that are nephrotoxic.
ii. Stop any potassium containing drugs
iii. Consider temporarily omitting drugs that limit renal potassium excretion(medications that have been associated with precipitating hyperkalaemia include non-steroidal anti-inflammatory agents, trimethoprim, pentamidine, azole antifungals, and heparin)
iv. Stop IV fluids containing potassium additives
v. Stop oral potassium supplements
vi. Monitor fluid balance

vii. Consider urinary catheterisation in oliguric patients
viii. Consider Calcium Resonium
ix. Commence low potassium diet and refer to Dieticians.

5.2.5

Two new oral potassium lowering drugs (Sodium zirconium cyclosilicate and Patiromer) have recently been approved for specific management of hyperkalaemia by the National Institute for Health and Care Excellence (NICE) but Insulin and glucose infusion remains the most effective emergency treatment. Sodium zirconium cyclosilicate and Patiromer can be used for adults with chronic kidney disease (CKD 3b-CKD 5) or patient on heart failure treatment either: In emergency care alongside standard care for acute life-threatening hyperkalaemia or for patient having persistent asymptomatic hyperkalaemia (K - > 6.0) when they on long term use of ACE/RAAS inhibitors or spironolactone. Data from preclinical and clinical studies show that a fixed dose of patiromer has a greater serum potassium–reducing effect when the patient has higher serum potassium values than lower serum potassium values and the onset of action for patiromer is 4 to 7 hours after administration and that the summary of product characteristics states that it should not replace emergency treatment for life-threatening hyperkalaemia. The NICE states that Sodium zirconium cyclosilicate and Patiromer could have a role in treating life-threatening hyperkalaemia alongside usual care. The clinical experts feel that it would not replace intravenous insulin and glucose, but it might replace calcium resonium. The NICE feels that managing acute life-threatening hyperkalaemia and chronic hyperkalaemia differed and that patiromer had a potential role in both.

Any long-term dialysis patient admitted with hyperkalaemia requires management acutely as mentioned in the above commentary. Contact must then be made with the on call consultant with regards to organising a haemodialysis session.

6 Training Needs

There is no mandatory training associated with this guidance. If staff has queries about its operation, they should contact their line manager in the first instance.

7 Review process

This document will be reviewed in 3 years of approval date, or sooner if required. The document will be reviewed in light of feedback and learning from any adverse incidents. In order that this document remains current, any of the appendices to the policy can be amended and approved during the lifetime of the policy without the document having to return to the ratifying committee.

8 Equality Impact Assessment (EQIA)

No negative impact to any person(s) or group(s) is anticipated.

9 Process for monitoring compliance

Aspect of compliance or effectiveness being monitored	Monitoring method	Responsibility for monitoring (job title)	Frequency of monitoring	Group or Committee that will review the findings and monitor completion of any resulting action plan
Incidence management adheres to ascribed practices within policy.	*Datix submission*	*Patient Safety*	*Annual*	

10 Associated Documentation

References

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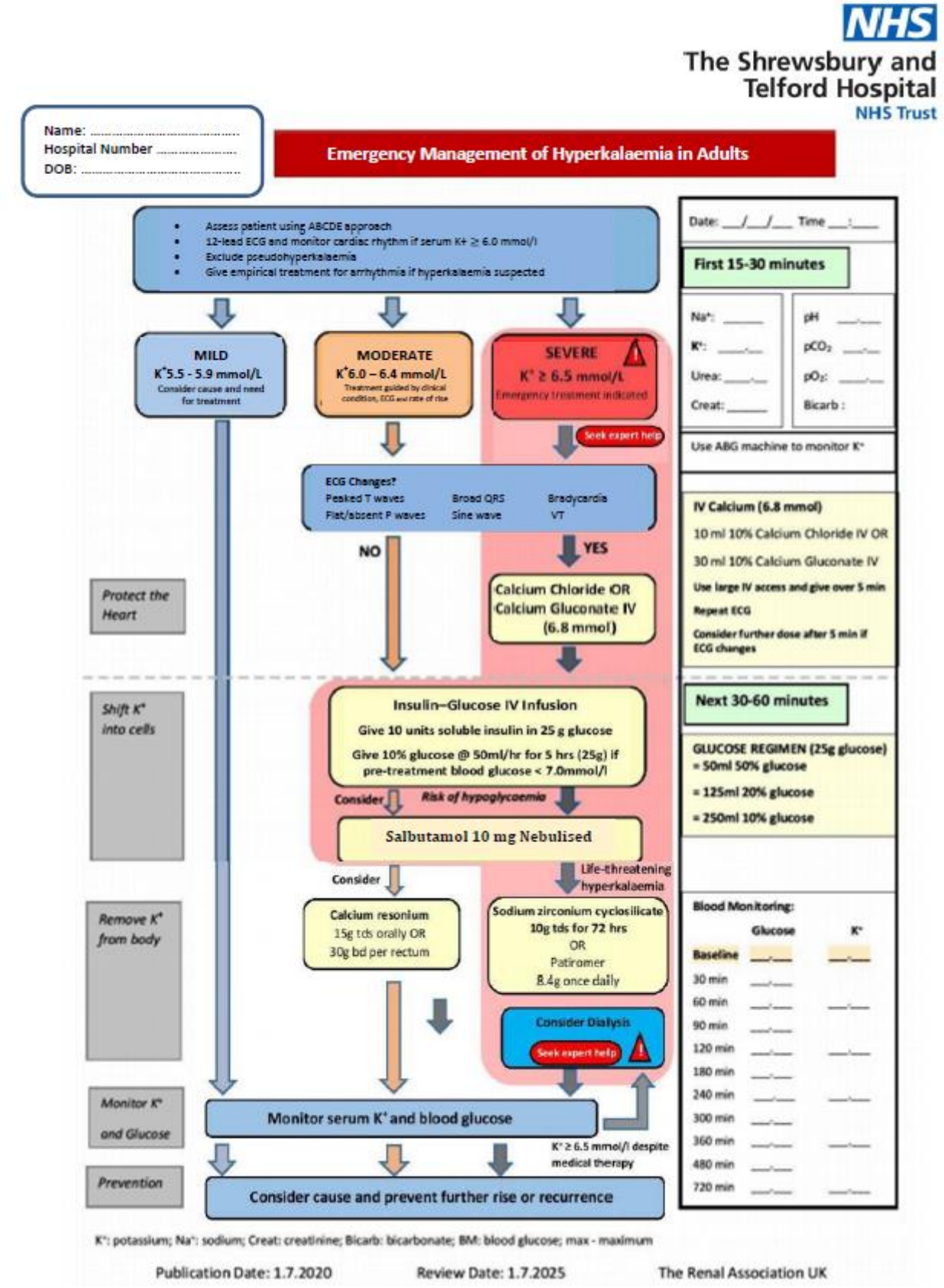
Appendices

A1 Renal Association Hyperkalaemia Treatment Algorithm
A2 Causes of Pseudohyperkalaemia
A3 Precipitating factors
A4 Drugs associated with Hyperkalaemia
A5 Low Potassium Diet (Basic guide) – seek Dietician advise
A6 Pharmacology (A6a,b,c,d)
A7 Life-threatening ECG changes

A1



Modified for SaTH NHS Trust 2021

A2 Causes of Pseudohyperkalaemia

- **Sample taken from limb with IV therapy infusing**

- **Prolonged tourniquet application**

- **Small needle or excessive pressure while sampling**

- **Haemolysis** *in vitro*
  Difficult sample collection, inappropriate handling (e.g. shaking tube)

- **K- EDTA contamination of the sample from an FBC tube (purple tube)**

- **Delayed sample separation**
  Potassium starts to rise in un-separated samples after 6hrs at room temperature (15-25ºC) and is unsuitable for analysis >12hrs. Always remember to indicate time and date on the sample & request forms to allow the laboratory to spot this

  Always try to ensure samples are dispatched to the laboratory as soon as possible and within 5hrs.

- **Storage at cold temperature**
  At low temperatures there is a rapid leak of potassium out of the red bloods cells therefore **NEVER** refrigerate samples

- **Thrombocytosis (high platelets)**

- **Severe leucocytosis / Fragile white cells**

A3 Precipitating Factors

Please note this is NOT a comprehensive List and other causes may need to be considered.

- **Acute Kidney Injury**
- **Chronic Kidney Disease (CKD 4&5) / End Stage Renal Disease on dialysis**
- **Hypoxia**
- **Metabolic Acidosis**
- **Diabetic Ketoacidosis (please refer to separate DKA Pathway)**
- **Mineralocorticoid deficiency (e.g. Addison's)**
- **Endogenous (tumour-lysis syndrome, rhabdomyolysis, trauma, burns)**
- **Non-compliance with dietary potassium restriction**
- **Drugs (see A4)**

A4 Drugs associated with Hyperkalaemia

Nyirenda MJ,Tang JI, Padfield PL, et al. Hyperkalaemia. BMJ 2009; 339: 1019-1024.

Drugs that alter transmembrane potassium movement

β blockers

Digoxin

Potassium-containing drugs

Potassium supplements

Salt substitutes

Hyperosmolar solutions (mannitol, glucose)

Suxamethonium

Intravenous cationic amino acids

Stored red blood cells (haemolysis releases potassium)

Herbal medicines (such as alfalfa, dandelion, horsetail, milkweed, and nettle)

Drugs that reduce aldosterone secretion

ACE inhibitors; Angiotensin II receptor blockers

NSAIDs

Heparins

Antifungals (ketoconazole, fluconazole, itraconazole)

Ciclosporin

Tacrolimus

Drugs that block aldosterone binding to mineralocorticoid receptor

Spironolactone

Eplerenone

Drospirenone

Drugs that inhibit activity of epithelial sodium channel

Potassium sparing diuretics (amiloride, triamterene)

Trimethoprim

Pentamidine

A5 Low Potassium Diet

The following is only a BASIC guide for initial management of inpatients that need a low potassium diet.

As soon as possible a **Dietitian** referral **must** be made by selecting renal advice on the PSAG board.

The dietary advice will need to be individualised based upon their current intake and clinical condition.

	Avoid	**Alternative**
Drinks	Coffee	Tea – all types
	Drinking chocolate, malted drinks (Ovaltine, Horlicks etc)	Water, tonic water, soda water and flavoured water
	Hi Juice squashes and Ribena	Cordials, Squash, Barley Water
	Fruit/vegetable juices or smoothies	Fizzy drinks – lemonade, Lucozade (original only)
	Oxo or Bovril	
Fruit	Bananas, plums, kiwi and dried fruit	Limit to one of these fresh fruit a day: Apple , Pear, Peach, Nectarine Satsuma, Tangerine or 1 small handful of grapes
Snacks	Chocolates, toffee, fudge and liquorice	Boiled sweets, mints and peppermints
	Crisps and potato based snacks	Turkish Delight and Jelly sweets
	Twiglets	Marshmallows
	All cakes, biscuits or cereal bars containing dried fruit, nuts or chocolate	Plain biscuits e.g. Rich Tea or Digestives Plain cake
Meals	All soups including build-up	
	Chips and Jacket potatoes	Boiled, mash potatoes, rice or pasta

A6a **Drug Pharmacology**

IV CALCIUM CHLORIDE 10% Injection (10mls contains 6.8mmol/L calcium)

IV CALCIUM GLUCONATE 10% Injection (10mls contains 2.26mmol/L calcium)

Administration	
Dosage	10mls 10% IV Calcium chloride **OR** 30mls 10% IV Calcium gluconate Administered intravenously over 5-10minutes into a large vein
Action	Effective within 3minutes and duration of action is 30-60minutes * Further sequential boluses, continued every 10minutes until a normalising ECG is observed.
SAFETY	12 Lead ECG is required BOTH before and AFTER administration Continuous cardiac monitoring is required during infusion Intravenous Calcium salts are irritants to veins and can cause tissue necrosis if extravasation occurs. Therefore ***ensure*** IV is patent and watch for signs of irritation ***DO NOT*** administer sodium bicarbonate IV simultaneously via same IV access due to risk of formation of insoluble calcium salts. ***DO NOT*** mix with ANY drugs due to incompatibilities

Adapted from:- The Renal Association (2014). Clinical practice guideline treatment of acute hyperkalaemia in Adults.Hampshire: Renal Association

A6b

INSULIN + GLUCOSE INFUSION

Administration	
Dosage	10units soluble insulin (e.g. Actrapid™) in 50mls 50% glucose (or 125mls 20% glucose) given INTRAVENOUSLY over 15-30minutes into a large vein.
Preparation	Measure 10units soluble insulin (e.g. Actrapid™) using an ***insulin syringe*** (10units is 0.1mls) Inject insulin into the 50mls 50% glucose (or 125mls 20% glucose) and invert and mix Withdraw insulin / dextrose mix into 50ml Syringe Complete identifying additive label and adhere to 50ml Syringe Actions above ***MUST*** be witnessed/cross-checked and counter-signed by a **registered** colleague (i.e. GMC, HPC, NMC)
Action	Effective within 15-30minutes and duration of action is 4-6hrs May be repeated as necessary
SAFETY	***INCORRECT*** measurement of insulin can be FATAL Blood glucose monitoring is ***ESSENTIAL*** for at least 6hrs post administration Hyperosmolar glucose (i.e. 50%) should NOT be used in hyperkalaemia in association with diabetic ketoacidosis.

Adapted from:- The Renal Association (2014). Clinical practice guideline treatment of acute hyperkalaemia in Adults.Hampshire: Renal Association

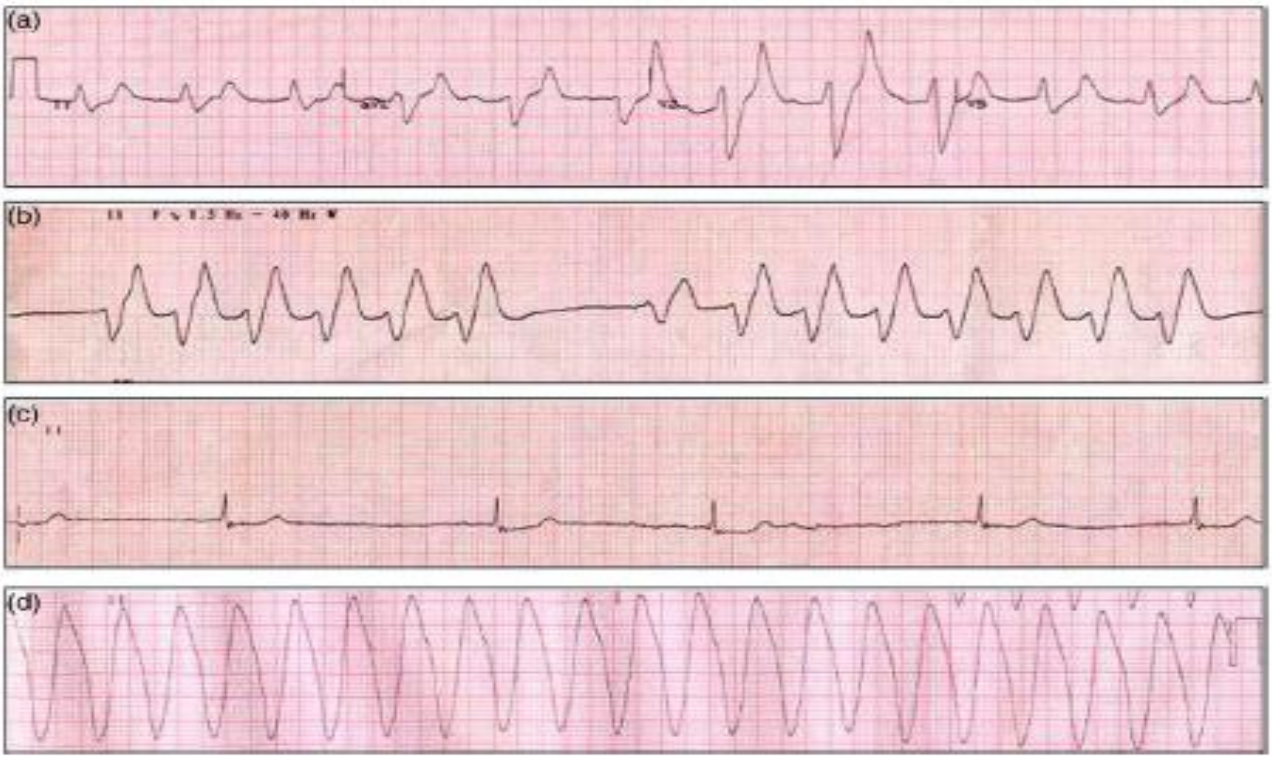
A6c

Salbutamol

Administration	
Dosage	10mg via nebuliser via mouthpiece or close fitting mask.
Action	Onset of action 30-60minutes and duration of action is 4-6hrs
SAFETY	***CAUTION*** with Ischaemic heart disease (give 10mg) ***CAUTION*** if tachyarrhythmia present ***CAUTION*** in acute angle glaucoma Tremor and tachycardia are common side effects Upto 40% patients will not respond to this treatment. Therefore salbutamol should ***NOT*** be used as a monotherapy.

Adapted from:- The Renal Association (2020). Clinical practice guideline treatment of acute hyperkalaemia in Adults

A7 Life-threatening ECG changes in Hyperkalaemia



(a) Illustrates tented T waves, loss of P waves and a wide QRS complex in a patient presenting with paralysis and acute renal failure (serum K+ 9.3mmols/L)
(b) Illustrates a sine wave pattern in a patient with acute renal failure and digoxin toxicity (serum K+ 9.3mmols/L
(c) Illustrates a severe bradycardia at a rate of 28bpm in a haemodialysis patient presenting with syncope (serum K+ 8.1mmols/L)
(d) Illustrates ventricular tachycardia in a haemodialysis patient presenting with generalised weakness (serum K+ 9.1mmols/L).
Paper speed is 25mm/s

Reproduced from

Alfonzo A, Isles C, Geddes C & Deighan C (2006) Potassium disorders - Clinical spectrum and emergency management. Resuscitation 2006; 70: 10-25.