GUIDELINES FOR THE TREATMENT OF HYPERKALAEMIA IN ADULTS
This document has been produced by the Clinical Resource Efficiency Support Team (CREST), which is a small team of health care professionals established under the auspices of the Central Medical Advisory Committee in 1988. The aims of CREST are to promote clinical efficiency in the Health Service in Northern Ireland, while ensuring the highest possible standard of clinical practice is maintained.

The guidelines have been produced by a sub-group of health care professionals chaired by Professor Gary McVeigh.

Membership of the CREST Hyperkalaemia sub-group:

Professor Gary McVeigh, Consultant Physician, BCH
Dr Kieran Fitzpatrick, Consultant Anaesthetist, BCH
Professor Peter Maxwell, Consultant Nephrologist, BCH
Dr Tom Trinick, Consultant Chemical Pathologist, UCHT

CREST wishes to thank them, the members of the Clinical Governance Pharmacist Team and all those who have contributed in any way to the development of these guidelines.

Further copies of this booklet and wall-chart may be obtained from:

Gary Hannan
CREST Secretariat
Room D1 Castle Buildings
Stormont
BELFAST BT4 3SQ
Telephone 028 9052 2028

Or you can visit the CREST website at:

www.crestni.org.uk

ISBN 1-903982-15-4
HYPERKALAEMIA

The reported incidence of hyperkalaemia in hospitalised patients is between 1 and 10%. **The vast majority of cases are related to patients prescribed angiotensin converting enzyme inhibitors (ACE) or angiotensin II receptor blockers (ARBs) in conjunction with spironolactone with pre-existing or new renal failure.** Most other cases are related to potassium supplementation and prescription of diuretics/drugs with potassium-sparing properties.

Aetiology of Hyperkalaemia

Pseudohyperkalaemia

- Prolonged tourniquet time
- Test tube haemolysis
- Marked leucocytosis and thrombocytosis (measure plasma not serum concentration in these disease states)
- Sample taken from a limb infused with IV fluids containing potassium

Transcellular shift (intracellular to extracellular compartment)

- Acidosis (including diabetic ketoacidosis)
- Drugs (digoxin poisoning, succinylcholine, arginine, β-blockade)
Renal causes

- Acute or chronic renal failure
- Hyperkalaemic renal tubular acidosis (type IV)
- Mineralocorticoid deficiency (hypoaldosteronism states)
- Drugs that interfere with potassium excretion (amiloride, spironolactone)
- Drugs that interfere with the renin-angiotensin system (angiotensin converting enzyme inhibitors, angiotensin II receptor blockade, nonsteroidal anti-inflammatory agents, heparin)

Increase circulating potassium - Exogenous or Endogenous

- Exogenous (potassium supplementation)
- Endogenous (tumour lysis syndrome, rhabdomyolysis, trauma, burns)

ASSESSMENT OF THE PATIENT

Is this “true” hyperkalaemia?

A repeat serum potassium should be ordered urgently, especially if hyperkalaemia is an unexpected or isolated finding and there are no ECG signs of hyperkalaemia, to exclude pseudohyperkalaemia.
How severe is the hyperkalaemia?

Hyperkalaemia is classified as –

- mild ($K^+ 5.5 - 6.0$)
- moderate ($K^+ 6.1 - 6.9$) or
- severe ($K^+ \geq 7.0$) or if ECG changes or symptoms (muscle weakness or flaccid paralysis, paresthesias) occurring at ANY level or serum potassium $\geq 5.5$mmol/l especially if associated with hypoxia

Situations associated with a rapid rise in potassium (acute renal failure, rhabdomyolysis) and hypoxia of any cause are more strongly associated with the development of cardiac conduction disturbances. Mild hyperkalaemia is common and often well tolerated in patients with chronic renal failure.

Is urgent treatment required?

Urgent treatment is required if the serum potassium is $\geq 7$ mmol/l OR hyperkalaemia is accompanied by ECG changes or above symptoms - even in the presence of mild hyperkalaemia ($K^+ 5.5 - 6.0$).

Why has the patient got hyperkalaemia?

A thorough medical history focussing on a history of renal disease and determination of the medications or fluids prescribed will often reveal the cause of the hyperkalaemia. Examine for bladder distension and prostatic hypertrophy. Catheterise if appropriate.
MONITORING THE PATIENT

- A 12-lead ECG and cardiac monitoring is mandatory in patients with hyperkalaemia
- The ECG does not always demonstrate changes, even in the presence of severe hyperkalaemia, so a normal ECG does not obviate the need for therapy. However, the presence of ECG findings should be a strong impetus for urgent action
- The most worrying findings are decreased or absent P-waves, PR prolongation, QRS widening, sine wave QRST, AV dissociation or asystole. It is often difficult to judge if T-waves are truly peaked and this finding on its own should not be an automatic indication for urgent therapy

Monitor urea, electrolytes and glucose at regular intervals. Additional blood work including creatinine kinase and blood gas analysis are performed if appropriate.

TREATMENT OF HYPERKALAEMIA

Stop further potassium accumulation

Stop all potentially offending drugs immediately. These include ACE inhibitors, angiotensin receptor blockers, potassium retaining diuretics eg spironolactone, amiloride – (in Frumil®), NSAIDs and K+ containing laxatives (Movicol®, Klean-Prep®, Fybogel®). Beta-blockers and digoxin should also be stopped as they prevent intracellular buffering of potassium and reduce the effectiveness of insulin-glucose and beta-2 agonists.
Place the patient on a low potassium diet. It is imperative that whilst waiting for this diet that the patient does not consume fruit juice, fruits, chocolate, fruit gums, biscuits, coffee or potatoes.

Protect the cardiac membrane

Give 10ml of calcium gluconate 10% intravenously over 2 minutes

- This intervention will not lower the potassium, but if ECG changes are present, there should be improvement seen within 1 to 3 minutes
- If improvement does not occur a further 10ml of calcium gluconate 10% can be given intravenously every 10 minutes until the ECG normalises (patients may require up to 50ml). The effect of this intervention is transient (approximately 30 minutes)
- It is important to note that if the patient is taking digoxin and the decision is made that calcium gluconate is required, it should be given *slowly* over 20 minutes mixed in 100ml of glucose 5% as rapid calcium administration may precipitate myocardial digoxin toxicity
- Digoxin toxicity can cause hyperkalaemia and arrhythmias and urgent haemodialysis or the administration of digoxin antibody (Fab) fragments may represent the preferred approach. Consult with senior colleagues

Shift the potassium from the blood into the cell

- Withdraw 10 units of Actrapid® insulin using an INSULIN syringe. *Always* obtain a check of volume from a senior nurse before proceeding. Add to 50ml glucose 50% and administer by slow IV injection over 5 minutes (see Appendix 1)
• The onset of the hypokalaemic action occurs within 15 minutes and lasts at least 60 minutes. The reduction in potassium observed ranges from 0.6 to 1.0mmol/l
• If the serum glucose is $\geq 15$mmol/l glucose administration with insulin is not required
• The effects of administering insulin/glucose are observed in 15 minutes and last 4-6 hours
• Monitoring – blood glucose should be measured 30 minutes after starting the infusion and then hourly up to six hours after completion of the infusion as delayed hypoglycaemia is commonly reported when less than 30g of glucose is administered with insulin

**Administer 10mg of nebulised salbutamol**

• Salbutamol for nebulisation is normally 2.5mg/2.5ml strength and the nebuliser chamber will hold 10ml i.e. 10mg salbutamol. This will lower the potassium by 0.5 to 1.0mmol/l by 15-30 minutes with the effect lasting at least 2 hours
• 20mg of nebulised salbutamol may be more effective than a 10mg dose at 2 hours. The lower dose is preferable in patients with ischaemic heart disease. There is no difference in the maximum hypokalaemic effect when nebulised salbutamol is compared with salbutamol (0.5mg) administered intravenously
• Salbutamol may not lower potassium in all patients and some studies show that up to 40% of dialysis dependent patients are resistant to these agents. The hypokalaemic response is also attenuated in patients taking beta-blockers and digoxin. Therefore salbutamol is not recommended as a single agent to treat hyperkalaemia
• There is evidence that the combination of nebulised salbutamol and insulin/glucose display additive effects in lowering the serum potassium, with attenuation of the hypoglycaemic action of insulin
These interventions buy time for more definitive therapy as they do not remove potassium from the body.

**Sodium bicarbonate - not recommended.** While this has been a traditional treatment for hyperkalaemia, many studies show that sodium bicarbonate fails to lower the serum potassium. A reduction in potassium will not occur within 60 minutes of administration. There are also potential risks in giving sodium bicarbonate in terms of volume and sodium overload and tetany in patients with chronic renal failure and co-existent hypocalcaemia. The risks outweigh any potential benefit.

**REMOVAL OF POTASSIUM FROM THE BODY**

**Haemodialysis**

If despite the above measures the potassium remains greater than 7mmol/l or if pathological ECG changes/symptoms persist, the renal team should be contacted to arrange urgent dialysis if appropriate.

- This is the most effective and definitive but invasive method in treating hyperkalaemia. It is strongly considered if hyperkalaemia is severe (level debated but ≥7.0mmol/l) and other first-line agents have been unsuccessful, or if there is ongoing tissue damage and continued release of intracellular potassium is expected.
- It is important to enlist the help of nephrology at an early stage in these circumstances.
Use the gut

- Calcium polystyrene sulphonate resin (Calcium Resonium®) enema 30g followed with 15g orally 4 times daily with regular lactulose will increase gut losses of potassium
- When given rectally the calcium resonium must be retained for 9 hours followed by irrigation to remove resin from the colon to prevent faecal impaction. Bowel perforation can be a complication
- The onset of action is slow (≥2 hours) and other measures should be employed in the interim to lower potassium levels. Do not add Calcium Resonium® to fruit juice which has a high potassium content
- One gram of resin exchanges 1mmol/l Na for 1mmol/k

Drugs administered for the treatment of hyperkalaemia are prescribed on the Kardex. The term Units must not be abbreviated when prescribing insulin.

Clinical pearls

- Always consult with the senior doctor responsible for the patient with hyperkalaemia
- Always stop drugs/food and fluids that exacerbate hyperkalaemia (ACE/ARBs, spironolactone, K⁺ sparing diuretics, digoxin, NSAIDs)
- Careful cardiac monitoring and repeated blood testing including glucose is mandatory
- A negative ECG does not negate the need for calcium gluconate and insulin/glucose in severe cases
- Digoxin toxicity (probable in renal failure) can increase K⁺.
Calcium gluconate **MUST** be administered slowly over 20 minutes mixed in 100ml glucose 5% to prevent myocardial digoxin toxicity. Alternatively, and perhaps safer, urgent dialysis and administration of digoxin antibody (Fab) fragments is preferred. Consult with senior colleagues

- Insulin can be administered as a single agent without 50ml/glucose 50% if glucose >15mmol/l
- Beta-2 agonists may not lower K⁺ especially in dialysis patients or those taking beta-blockers or digoxin. Not recommended as a single agent
- Calcium gluconate/insulin/beta-2 agonists are not definitive therapies - they simply buy time for more definitive therapy
- Ensure that the patient is placed on a “low potassium diet” and ban the patient from consuming food with a high potassium content e.g. chocolate, fruit juices, until a dietetic assessment has been undertaken
REFERENCES


Appendix 1

Safe administration of insulin and glucose in the treatment of hyperkalaemia

A second check must be performed at every stage of the preparation and administration.

Two practitioners must be involved, one of whom must be a senior nurse on duty.

1. Select the following:
   - 1 x 50ml vial glucose 50%
   - 1 x 50ml IV syringe and needle
   - 1 x vial of Actrapid® or Humulin S® (soluble insulin)
   - 1 x insulin syringe with needle

2. Measure 10 units of insulin using the insulin syringe:
   - draw the plunger back to the 10 unit mark on the syringe
   - note 10 units of insulin is contained in 0.1ml

3. Inject the insulin into the glucose 50% vial.

4. Invert the glucose vial to mix.

5. Withdraw the contents of the glucose vial into the 50ml syringe.

6. Administer into a large vein by slow IV injection over 5 minutes

7. Monitor blood glucose and serum potassium levels according to treatment guidelines.
GUIDELINES FOR THE TREATMENT OF HYPERKALAEMIA IN ADULTS