### HYPERKALEMIA

#### Definition

In serum:
- **Neonates** \( K^+ > 6.0 \text{mmol/L} \)
- **Child and adult** \( K^+ > 5.5 \text{mmol/L} \)

Patients with chronic kidney disease (CKD) and low GFR (less than 30) may have persistent or recurrent hyperkalemia and may be tolerant of higher levels of potassium than those with sudden onset hyperkalemia. For CKD patients with potassium of 5.5 – 6.5 mmol/L, discuss promptly with the paediatric nephrologist on call.

**NOTE:**

Capillary blood collections will increase haemolysis of cells and increase serum potassium. To exclude the effect of haemolysis, an elevated serum potassium collected by capillary method must be repeated with a running venous blood specimen. Pseudo-hyperkalemia can also be seen in disorders with extremely high white or red cell counts.

#### Causes

**Excessive load:**

1. Large packed red cell transfusions
2. Medications – high dose penicillin G
3. Errors in IV KCl dose calculation
4. Liquid formulas and foods (salt substitutes)
5. Excessive cellular damage from trauma, burns

**Transcellular \( K^+ \) shift**

1. Acidosis
2. Insulin deficiency (NB: fasting in renal failure patients)
3. Hyperosmolality (mannitol)
4. Non specific \( \beta_2 \) Blockers due to blocking of catecholamine induced uptake
5. ACE inhibitors (ACEI), Angiotensin Receptor Blockers (ARBs), aldosterone antagonists

**Reduced renal excretion**

1. Renal failure
2. Inherited or acquired tubular dysfunction
3. Mineralocorticoid deficiency
4. Drug effect – spironolactone, NSAIDs. ACEI and ARBs, trimethoprim
5. Hypovolemia
Hyperkalemia

Investigations for Chronic Hyperkalemia

1. Complete serum biochemistry including creatinine, urea, blood gas
2. Urine potassium
3. Other investigations if chronic hyperkalemia considered – first morning plasma renin, aldosterone, urine electrolytes (K⁺, osmolality, creatinine)
4. Calculate Transtubular K gradient (TTKG) –
   \[ TTKG = \frac{\text{Urine K} \times \text{Urine Osmolality}}{\text{Plasma K} \times \text{Plasma Osmolality}} \]
   
   TTKG <7 in presence of hyperkalemia suggests impaired excretion due to deficiency or resistance to mineralocorticoid (this is not accurate if urine is more dilute than serum or contains very little Na⁺)

Therapeutic Approach

1. Recognise acute adrenal insufficiency due to abrupt cessation of steroids (treat with hydrocortisone, volume replacement)
2. Start continuous ECG monitoring
3. Stop all medications, and IV fluids that may contain potassium
4. Assess cardiac effects with urgent ECG (12 lead) when K⁺ > 6.0-6.5mmol/L
5. If ECG changes of hyperkalemia are present treat immediately – see below.
6. Potassium lowering measures
   a) Potassium shifting therapies - sodium bicarbonate, salbutamol, insulin & dextrose
   b) Potassium removing therapies – resonium, acute dialysis

ECG Changes of Hyperkalemia

Narrow peaked T waves, U waves, shortened QT interval, prolonged PR interval, prolonged QRS interval, loss of P wave, sine waves and finally ventricular fibrillation

ECG CHANGES DO NOT NECESSARILY PROGRESS IN ORDER OF SERIOUSNESS OR SEVERITY, IN RELATION TO THE SEVERITY OF HYPERKALEMIA.

Hyperkalemia with severe ECG changes (not isolated peaked T waves) is a Medical Emergency:

- Notify PICU
- Give IV calcium gluconate urgently if there is widening of QRS complex or loss of P waves. In this situation, a registrar can administer IV calcium gluconate through a large peripheral fasting running vein such as in the antecubital fossa.
- Nebulised salbutamol can be started while IV dextrose and insulin is set up.

Hyperkalemia without ECG changes must still be treated urgently with nebulised salbutamol which can be started while IV dextrose and insulin is set up.
### Medications in Hyperkalemia

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mechanism</th>
<th>Onset/Duration</th>
<th>Dose</th>
</tr>
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<tbody>
<tr>
<td><strong>Calcium gluconate 10%</strong>&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Stabilises membrane protect myocardium</td>
<td>Immediate / 30min</td>
<td>0.5mls/kg (max 20ml)</td>
</tr>
<tr>
<td><strong>Salbutamol</strong>&lt;sup&gt;2,3&lt;/sup&gt;</td>
<td>$K^+$ shift intracellularly</td>
<td>20min / 2-4hrs</td>
<td>2.5-5mg neb</td>
</tr>
<tr>
<td><strong>Insulin</strong>&lt;sup&gt;4&lt;/sup&gt;</td>
<td>$K^+$ shift intracellularly</td>
<td>20min / 4-6hrs</td>
<td>If have central IV access 0.1u/kg actrapid iv bolus plus 2mls/kg 50% dextrose iv bolus</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>No central IV access 0.1u/kg iv bolus plus 5 mls/kg 10% dextrose iv bolus</td>
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<td></td>
<td>In both cases check dextrose stick at 15 and 30 minutes”</td>
</tr>
<tr>
<td><strong>Resonium</strong>&lt;sup&gt;5,6&lt;/sup&gt;</td>
<td>Removes K by exchange across colon</td>
<td>1-2hrs / 4-6hrs</td>
<td>1gm/kg PR</td>
</tr>
<tr>
<td><strong>Frusemide</strong>&lt;sup&gt;7&lt;/sup&gt;</td>
<td>Inhibits K reabsorption in Nephron</td>
<td>10min</td>
<td>1-2mg/kg IV stat</td>
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</table>

**Comments**

1. Hyperkalemia with severe ECG changes (not isolated peaked T waves) is a **medical emergency** (see above).
2. Salbutamol is the treatment of first choice because of ease of administration while other treatments are being set up.
3. Salbutamol and IV insulin have additive effect. **The magnitude of potassium lowering effect is unpredictable and not all patients will benefit. These are only short term treatments while definitive treatment is being organised**
4. Insulin and $\beta_2$ adrenergic receptors stimulate Na-K+ ATPase pump activity
5. Sodium resonium contains a large amount of sodium and can result in volume overload
6. Resonium has its effect principally through potassium exchange in colon, therefore rectal administration is preferred. If there are specific contraindications to rectal use (e.g. after large bowel surgery or suspected bowel obstruction) then oral administration is acceptable.

Coagulopathy should not be a contraindication unless they have active rectal bleeding, e.g. in patients with GVHD or severe mucositis.

SORBITOL should NOT be used to make up resonium mixture as it has been associated with colonic necrosis

**Neonates should not be given resonium due to reduced gut motility**

7. Frusemide is only effective if renal function is relatively normal.

References


Kiessling SG, Goebel J, Somers MJG. Pediatric Nephrology in the ICU. 2009. Springer