Hyperkalaemia – Emergency Management in Children

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Author/custodian	Senior Medical Officer, Emergency Department			**Review date**	06/03/2028
Supersedes	V1.0				
Applicable to	All staff involved in the care and emergency management of children with hyperkalaemia				
Authorisation	Executive Director Clinical Services				

Purpose

This purpose of this guideline is to guide the care and management of a paediatric patient with acute hyperkalaemia in Queensland.

Hyperkalaemia presentations require management using a stepwise approach and this guideline places an emphasis on the recognition of patients with potentially life-threatening cardiac toxicity.

Scope

This guideline is intended to assist all clinical staff to care and manage hyperkalaemia appropriately for paediatric patients in Queensland. It is not intended to be a substitute for specific professional or clinical advice, or to replace consultation with senior staff.

This material is published by Queensland Health with the intention of providing a guideline for use at Queensland Children's Hospital. Anyone wishing to use this guideline outside QCH should refer to their local Medicines Committee before using.

Guideline

Definition

Hyperkalaemia is defined as a serum potassium of greater than 5.5 mmol/L in a child or greater than 6.0 mmol/L in a neonate.

ALERT

Severe hyperkalaemia (potassium greater than 7 mmol/L or greater than 6.5 mmol/L with ECG changes) is a medical emergency.

Whilst less common, patients with mild (potassium 5.5 – 6.0 mmol/L) and moderate hyperkalaemia (potassium 6.1 – 7.0 mmol/L) may also have potentially life-threatening ECG changes.

Causes

Mechanism	Cause
Pseudohyperkalaemia (Factitious hyperkalaemia)	Collection technique (haemolysis) Significant thrombocytosis (platelets > 1,000 x 10^9/L) Significant leucocytosis (WCC >70 x 10^9/L)

Impaired potassium excretion	Acute renal failure
	Chronic kidney disease
	Hypoaldosteronism
	  Primary adrenal insufficiency
	Tubular unresponsiveness to aldosterone
	  Obstructive nephropathy
	  Sickle cell disease
	Medications affecting Na+/K+ exchange
	  Potassium sparing diuretics
	  Angiotensin converting enzyme inhibitors
	  Angiotensin II receptor blockers
	  Trimethoprim
	  NSAIDs
	  Calcineurin inhibitors (tacrolimus and cyclosporin)

Redistribution of potassium from the intracellular to extracellular space	Acidosis
	Familial hyperkalaemic periodic paralysis
	Hypertonicity
	  Hyperglycaemia
	  Mannitol
	Medications
	  Succinylcholine
	  Beta blockers
	  Digoxin

Addition of potassium into extracellular space	Potassium supplements or potassium containing IV fluids Rhabdomyolysis Crush injury Tumour lysis syndrome Haemolysis

Assessment

Hyperkalaemia is usually asymptomatic. Where clinical signs and symptoms do exist, cardiac and neurological features tend to predominate.

ALERT

Hyperkalaemia may be life-threatening. Management of the elevated potassium should always take priority over any diagnostic evaluation.

History

Signs and symptoms of hyperkalaemia:

- Nausea and vomiting
- Fatigue
- Paraesthesia, muscle weakness, paralysis
- Respiratory distress and failure
- Palpitations, syncope, cardiac arrest

Aetiology:

- Past medical history
- Full medication history (including any recent intravenous fluids and infusions)

Examination

The examination should be focused to identify the underlying aetiology and identify the complications of severe hyperkalaemia.

Examination findings suggestive of severe hyperkalaemia include haemodynamic instability, arrhythmia, diminished deep tendon reflexes, muscle weakness/paralysis and hypoventilation.

Investigations

ECG

An ECG should be urgently performed to assess for conduction abnormality or arrhythmia.

ECG changes seen in hyperkalaemia include tall/peaked T waves (seen across all leads), prolonged PR interval, loss of P waves and widening of the QRS complex which is often described as 'broad and bizarre'. These changes can progress to sine waves, ventricular arrhythmias and asystole. These ECG changes occur in a typical pattern.

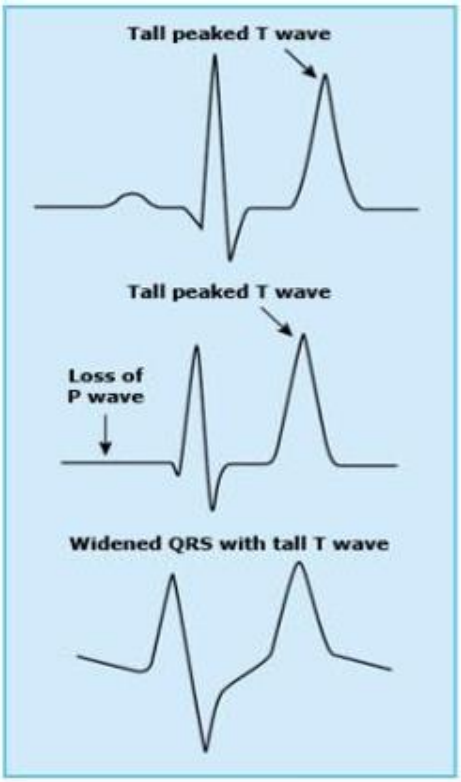
It is important to note that there is no definite correlation between serum potassium level and ECG changes or risk of arrhythmia, and hyperkalaemic patients may develop arrhythmias despite a normal baseline ECG.

Other electrolyte or biochemical abnormalities such as hypocalcaemia, acidosis and hyponatraemia can enhance cardiac toxicity and precipitate arrhythmias at lower potassium levels.

Patients with chronic hyperkalaemia may not have ECG changes despite high serum potassium levels.

Figure 1 – Typical electrocardiographic features of hyperkalaemia

From UpToDate

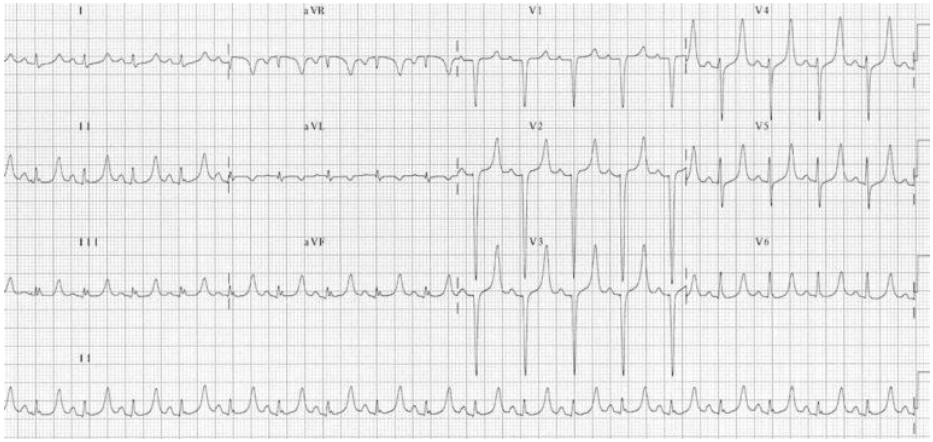


Serum potassium	Major change
5.5-6.5	Tall peaked T waves
6.5-7.5	Loss of P waves
7.0-8.0	Widening of QRS
8.0-10.0	Sine wave, ventricular arrhythmia, asystole

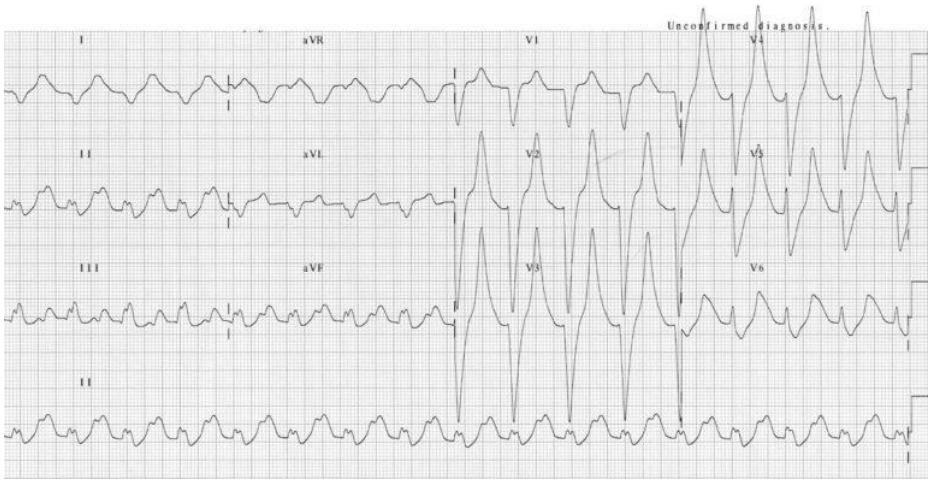
Adapted from: Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. Am J Emerg Med 2000; 18:721.

ECG examples

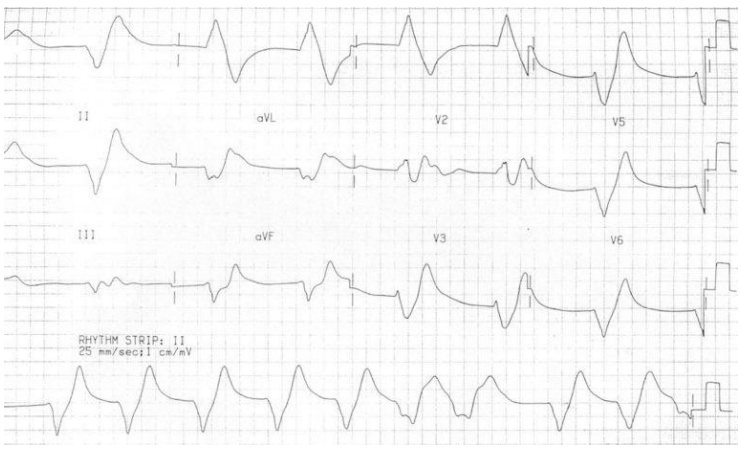
From Life in the Fast Lane



Peaked T waves



Peaked T waves
Prolonged PR interval
Broad and bizarre QRS



Sine wave

Venous blood gas/iSTAT

Urgently perform a venous blood gas or iSTAT to confirm the potassium level. This should not delay the initiation of treatment, especially in severe hyperkalaemia.

ALERT

In patients with renal dysfunction, you should assume the potassium level is correct until proven otherwise.

Renal function

Investigations to determine underlying aetiology should be guided by history and examination findings.

Management

The management of hyperkalaemia can be broken down into a series of interventions. Whilst the steps and principles are the same, there is significant variation within each step, dependent on the serum potassium level and clinical picture.

For the purpose of management, hyperkalaemia can be divided into:

- Cardiac arrest
- Severe: potassium >7.0 or >6.5 with ECG changes
- Moderate: potassium 6.1-7.0 without ECG changes
- Mild: potassium 5.5-6.0 without ECG changes in an asymptomatic patient

Clinicians should review the Children's Resuscitation Emergency Drug Dosage manual (CREDD) for guidance on how to prepare any drug listed below.

ALERT

In acidosis, potassium shifts from the intracellular space to the extracellular space. This means that patients with hyperkalaemia and acidosis may have a total body potassium deficit. Treat as per the guidelines below but ensure close monitoring of potassium levels as the acidosis corrects.

ALERT

In cases of crush injury or tumour lysis syndrome, the potassium will continue to rise due to ongoing cell breakdown. The threshold for treatment is therefore lower in such cases.

If Tumour Lysis Syndrome is suspected, refer to the Tumour Lysis Syndrome Guideline.

Cardiac arrest

ALERT

It is extremely rare for hyperkalaemia to be the primary cause of cardiac arrest in children. Hyperkalaemia seen in cardiac arrest is more likely to occur because of multi organ dysfunction and cell death. Regardless of the cause, treat as detailed below.

Step 1 – Prioritise patient care

- Activate a CODE BLUE and notify ED SMO
- Liaise with PICU consultant regarding potential for Extra-corporeal Life Support (ECLS) and/or dialysis
- Continue CPR as per the Advanced Paediatric Life Support algorithm
- Obtain peripheral large vein IV access or place an IO
- If Tumour Lysis Syndrome suspected, refer to the Tumour Lysis Syndrome Guideline.
- If adrenal insufficiency suspected, give hydrocortisone 2mg/kg

Step 2 - Protect the myocardium

10% Calcium gluconate	0.11 mmol/kg (maximum 4.4 mmol) Infuse over 3 to 5 minutes via central access, large vein, or IO **Do not give with sodium bicarbonate** Dose can be repeated after 15mins if ECG still abnormal	Stabilises myocardium Reduces risk of arrhythmias Does not lower potassium **Contraindicated in digoxin toxicity or hypercalcaemia** Risk of injury (Irritation/tissue necrosis) is high if infiltration/extravasation occurs Onset of action: 5 minutes Duration of effect: 30 – 60 minutes

Step 3 - Lower the serum potassium level

Insulin/Glucose bolus dosing regimen (ARREST only)	Glucose 10% 5 mL/kg followed by Insulin (Actrapid) 0.1 unit/kg	Give glucose as a slow push followed by insulin dose HIGH RISK OF HYPOGLYCAEMIA Monitor BGL every 15 minutes Onset of action: 10 – 20 minutes Maximum effect: 30 – 60 minutes Duration of effect: 4 – 6 hours Lowers potassium by: 0.5 – 1.2 mmol/L
Sodium bicarbonate	1 mmol/kg intravenous (via large vein) Infuse over 5 minutes Do not administer with other drugs	Use only in significant metabolic acidosis Monitor pH to avoid alkalosis Onset of action: within 1 hour Duration of effect: up to 2 hours Effect small and inconsistent

Step 4 - Promote the elimination of potassium from the body

ECLS/Dialysis		Consider in life-threatening hyperkalaemia Early liaison with PICU

Once return of spontaneous circulation occurs, manage as severe hyperkalaemia.

Severe hyperkalaemia: potassium greater than 7 mmol/L or greater than 6.5 mmol/L with ECG changes

ALERT

Severe hyperkalaemia may be life-threatening.

Step 1 – Prioritise patient care

- Notify senior medical staff
- Manage in a resuscitation area
- Instigate full cardiac monitoring
- Cease any potassium containing feeds/fluids
- Cease any medications that increase serum potassium or reduce potassium excretion
- Obtain central IV access where readily available (e.g. in patients with a port) or where achievable *without significant delay to treatment*. If central access not possible, obtain peripheral large vein IV access.
- Consider early discussion with renal and/or PICU
- If Tumour Lysis Syndrome suspected, please refer to the CHQ-GDL-01483 Tumour Lysis Syndrome Guideline.
- If adrenal insufficiency suspected, give hydrocortisone 2mg/kg

Step 2 - Protect the myocardium

10% Calcium gluconate	0.11 mmol/kg (maximum: 4.4 mmol) Infuse over 3 to 5 minutes via central access (preferred) or a large vein **Do not give with sodium bicarbonate** Dose can be repeated after 15mins if ECG still abnormal	Stabilises myocardium Reduces risk of arrhythmias Does not lower potassium **Contraindicated in patients with Tumour lysis syndrome (unless unstable arrhythmia present), digoxin toxicity or hypercalcaemia** Risk of injury (Irritation/tissue necrosis) is high if infiltration/extravasation occurs Onset of action: 5 minutes Duration of effect: 30 – 60 minutes

Step 3 - Lower the serum potassium level

Salbutamol	Less than 6 years – 2.5 mg nebulised Greater than 6 years – 5 mg nebulised Repeat 1-2hrly as required	Onset of action: 5 – 30 minutes Maximum effect: 90 minutes Duration of effect: 3 hours Lowers potassium by: 0.5 – 1 mmol/L
Insulin/Glucose	Glucose 10% infused at rate of 5 mL/kg/hr PLUS Insulin (Actrapid) 0.1 unit/kg/hr	Start glucose infusion first Monitor BGL every 15 minutes Continue infusion until potassium is within range Onset of action: 10 – 20 minutes Maximum effect: 30 – 60 minutes Duration of effect: 4 – 6 hours Lowers potassium by: 0.5 – 1.2 mmol/L
Sodium bicarbonate	1 mmol/kg intravenous (via large vein) Infuse over 5 minutes Do not mix with other drugs	Use only in significant metabolic acidosis Monitor pH to avoid alkalosis Onset of action: within 1 hour Duration of effect: up to 2 hours Effect small and inconsistent
Furosemide	1 mg/kg intravenous Infuse over 10 minutes	Use as adjuvant therapy after consultation with renal or PICU Onset of action: within 1 – 2 hours

Step 4 – Promote the elimination of potassium from the body

Sodium polystyrene sulfonate (Resonium A®)	0.25 gram/kg orally or rectally Max 15g Can be repeated up to four times/day	Contraindicated in recent abdominal surgery, perforation, ileus and hypernatraemia Onset of action: within 1 – 2 hours Duration of effect: 4 – 6 hours
Dialysis		Indicated in life-threatening hyperkalaemia or when pharmacological therapies fail Early liaison with renal and/or PICU

Moderate hyperkalaemia: potassium 6.1 – 7 mmol/L without ECG changes

Step 1 – Prioritise patient care

- Notify senior medical staff
- Manage in a resuscitation area
- Instigate full cardiac monitoring
- Cease any potassium containing feeds/fluids
- Cease any medications that increase serum potassium or reduce potassium excretion
- Obtain IV access
- Consider early discussion with renal and/or PICU

Step 2 – Lower the serum potassium level

Salbutamol	Less than 6 years – 2.5 mg nebulised Greater than 6 years – 5 mg nebulised Repeat 1-2hrly as required	Onset of action: 5 – 30 minutes Maximum effect: 90 minutes Duration of effect: 3 hours Lowers potassium by: 0.5 – 1 mmol/L
Insulin/Glucose	Glucose 10% infused at rate of 5 mL/kg/hr PLUS Insulin (Actrapid) 0.1 unit/kg/hr	Start glucose infusion first Monitor BGL every 15 minutes Continue infusion until potassium is within range Onset of action: 10 – 20 minutes Maximum effect: 30 – 60 minutes Duration of effect: 4 – 6 hours Lowers potassium by: 0.5 – 1.2 mmol/L
Sodium bicarbonate	1 mmol/kg intravenous (large vein) Infuse over 5 minutes Do not mix with other drugs	Use only in significant metabolic acidosis Monitor pH to avoid alkalosis Onset of action: within 1 hour Duration of effect: up to 2 hours Effect small and inconsistent
Furosemide	1 mg/kg intravenous Infuse over 10 minutes	Use as adjuvant therapy after consultation with renal or PICU Onset of action: within 1 – 2 hours

Step 3 - Promote the elimination of potassium from the body

Sodium polystyrene sulfonate (Resonium A®)	0.25 gram/kg orally or rectally Max 15 g Can be repeated up to four times/day	Contraindicated in recent abdominal surgery, perforation, ileus and hypernatraemia Onset of action: within 1 – 2 hours Duration of effect: 4 – 6 hours

Mild hyperkalaemia: potassium 5.5 – 6.0 mmol/L, no ECG changes and asymptomatic patient

Step 1 – Prioritise patient care

- Cease any potassium containing feeds/fluids
- Cease any medications that increase serum potassium or reduce potassium excretion

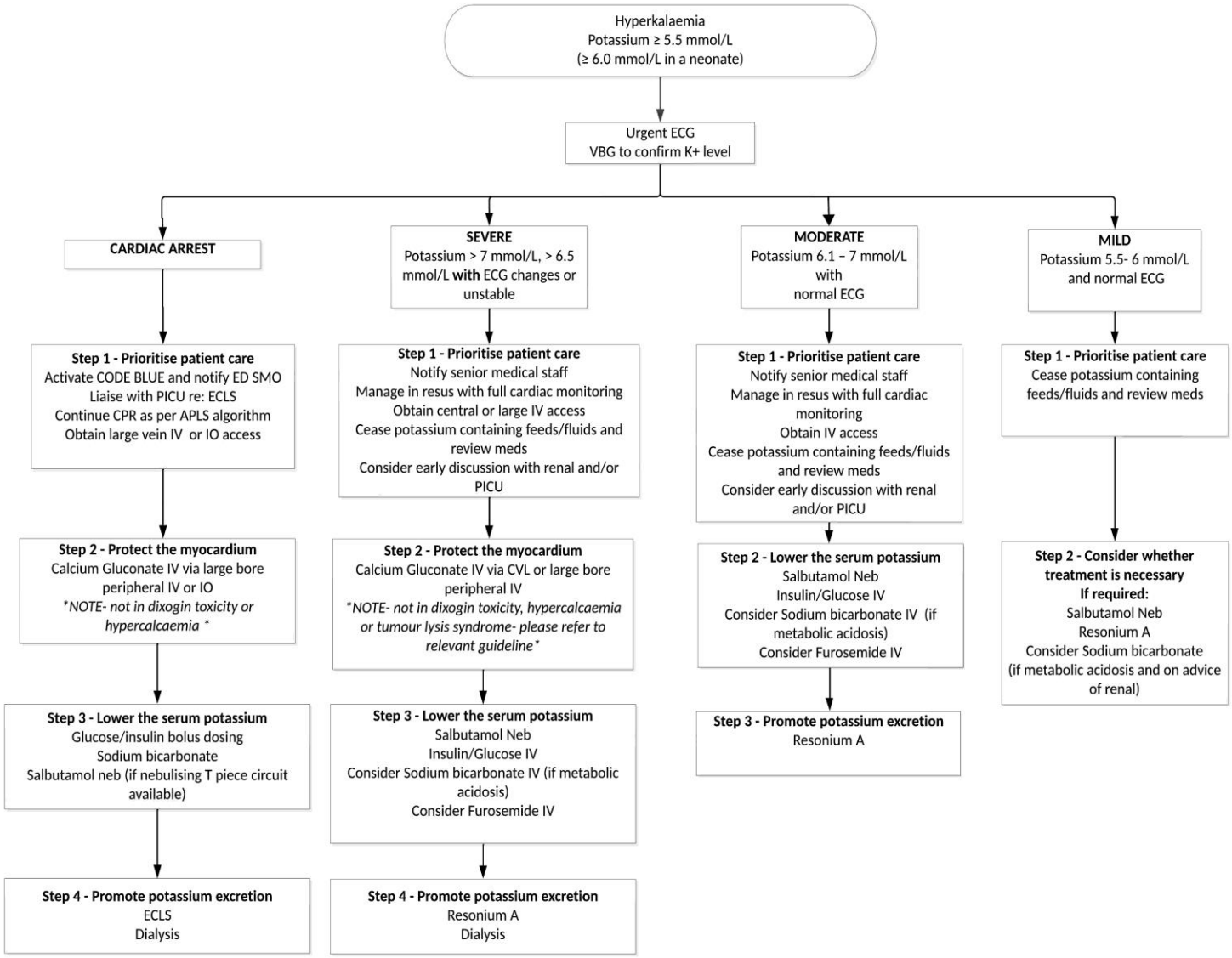
Step 2 - Consider whether treatment is necessary

If required:

Salbutamol	Less than 6 years – 2.5 mg nebulised Greater than 6 years – 5 mg nebulised Repeat 1-2hourly as required	Onset of action: 5 – 30 minutes Maximum effect: 90 minutes Duration of effect: 3 hours Lowers potassium by: 0.5 – 1 mmol/L
Sodium polystyrene sulfonate (Resonium A®)	0.25 gram/kg orally or rectally Max 15 g Can be repeated up to four times/day	Contraindicated in recent abdominal surgery, perforation, ileus and hypernatraemia Onset of action: within 1 – 2 hours Duration of effect: 4 – 6 hours
Sodium bicarbonate	1 mmol/kg intravenous (large vein) Infuse over 5 minutes Do not mix with any other drugs	Use only in significant metabolic acidosis (after consultation with renal) Monitor pH to avoid alkalosis Onset of action: within 1 hour Duration of effect: up to 2 hours Effect small and inconsistent

ALERT

Patients with marked tissue breakdown (rhabdomyolysis, crush injury, tumour lysis syndrome) may require aggressive therapy to remove potassium despite only mild hyperkalaemia.



Hyperkalaemia: Emergency Management in Children flowchart

Consultation

Key stakeholders who reviewed this version:

- Senior Medical Officer Renal
- Senior Medical Officer PICU
- Senior Medical Officers Emergency
- CREDD

Definition of terms

Term	Definition
Hyperkalaemia	Serum potassium greater than 5.5mmol/L (greater than 6.0mmol/L in a neonate). Further classified by severity: Mild: 5.5 – 6 mmol/L Moderate: 6.1 – 7 mmol/L Severe: greater than 7 mmol/L or greater than 6.5 mmol/L with ECG changes
Pseudohyperkalaemia	Falsely elevated serum potassium level greater than 5.5mmol/L

References and suggested reading

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Guideline revision and approval history

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1.0 22/09/2020	Senior Medical Officer, Emergency Department	Divisional Director, Critical Care	Executive Director Clinical Services QCH
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Accreditation references	NSQHS Standards (1-8): 1 Clinical Governance, 4 Medication Safety,8 Recognising and Responding to Acute Deterioration