

## 1. Introduction and Who Guideline applies to

Hyperkalaemia is defined as a serum potassium ( $K^+$ ) concentration of  $> 5.5 \text{ mmol/L}$ . Even lesser elevations increase all-cause mortality: Patients with  $K^+ 5.1 - 5.5$  have twice the risk of dying in hospital than those with  $K^+ 3.5 - 5.0$ . [1]

Raised  $K^+$  is seen in up to 10% of hospitalised adults. Those with CKD are at particular risk, with the incidence of hyperkalaemia rising from 2 to 42% as GFR falls from 60 to 20  $\text{mL min}^{-1}$ . [2]

This document provides practical guidance on managing acute hyperkalaemia based on the 2023 UK Kidney Association guideline [3] and the 2019 NICE technology assessment of sodium zirconium cyclosilicate (SZC). [4] It applies to all adult inpatients, and covers clinical staff working within, the Emergency and Specialist Medicine (ESM) Clinical Management Group (CMG) but may also be used in other CMGs where adult inpatients with acute hyperkalaemia are managed.

Caveats:

- Information about pseudohyperkalaemia and its avoidable causes is kept intentionally brief, as in-depth discussion is readily accessible elsewhere. [5]
- The guideline does not apply to managing hyperkalaemia in diabetic ketoacidosis (DKA).
- An aggressive diuretic regimen combining a loop diuretic, a thiazide, and acetazolamide - the 'nephron bomb' described in the Internet Book of Critical Care (IBCC) - can markedly increase urinary  $K^+$  excretion. Provided the patient is not hypotensive, hypovolaemic, or anuric, it may help avoid dialysis when routine treatments including a loop diuretic alone have failed. The technique requires some expertise and is therefore not included in our treatment algorithm.

## 2. Guideline Standards and Procedures

- 2.1 Patients should initially be managed according to [Appendix A](#) and [Appendix B](#), also available as a single double-sided proforma from the 'ED on-demand print kiosk'.
- 2.2 'Shifting treatments'—namely nebulised salbutamol and IV insulin-glucose infusion (with IV sodium bicarbonate where indicated) —often need repeating to reduce  $K^+$  to  $<6.0 \text{ mmol/L}$ .
- 2.3 Repeated shifting treatments increase the risk of hypoglycaemia. If capillary blood glucose (CBG) is  $<7 \text{ mmol/L}$  before a new round, give an additional 25g glucose (250 mL of 10% IV over 5 hours).
- 2.4 Shifting treatments lower serum  $K^+$  only temporarily. Levels typically rebound within 2–6h unless  $K^+$  is removed from the body via diuresis (responding to fluid resuscitation or loop diuretics),  $K^+$  binders or, ultimately, renal replacement therapy (RRT).
- 2.5 For patients on regular dialysis, arrange Renal Unit transfer as soon as stable. Emergency haemofiltration in critical care is a suboptimal alternative and should be avoided.
- 2.6 Criteria for safe inter-hospital transfer to the Renal Unit are outlined in [Appendix A](#), Box 4.
- 2.7 In patients not on dialysis, repeated shifting treatments combined with a  $K^+$  binder (sodium zirconium cyclosilicate) often avoid the need for emergency RRT.
- 2.8 Cation-exchange resins (e.g. calcium resonium) should no longer be routinely used for acute hyperkalaemia in hospital. [3]
- 2.9 Medications—especially RAAS inhibitors, NSAIDs, and trimethoprim—commonly contribute to hyperkalaemia. A thorough medicines review ([Appendix C](#)) is therefore essential to reduce recurrence.
- 2.10 This applies also to patients with mild hyperkalaemia not requiring emergency treatment, as even they face an elevated mortality risk.
- 2.11 In patients with diabetes, kidney, or cardiovascular disease at high risk of AKI who are able to self-manage, consider providing 'sick day' guidance on temporary cessation of certain medications (RAASi, diuretics, metformin, NSAIDs and SGLT2i) during acute dehydrating illness (e.g. diarrhoea, vomiting). For an example, see the patient leaflet produced by the London Kidney Network. [7]
- 2.12 Recommended interventions to prevent hyperkalaemia are listed in [Appendix D](#).

### **3. Education and Training**

No additional skills are required to follow this guideline.

### **4. Monitoring Compliance**

What will be measured to monitor compliance	Compliance monitoring	Monitoring Lead	Frequency	Reporting arrangements
Proportion of haemodialysis pts presenting to ED with $K^+ \geq 6.0$ who have CVVH in ICU rather than dialysis at the renal unit	Audit	Richard Baines	Once every three years	ESM Q&S board
Appropriate Szc prescribing practice	Audit	Rishi Gupta	Once every three years	ESM Q&S board

### **5. Supporting References**

1. Singer A, Thode HC and Peacock WF. [A retrospective study of emergency department potassium disturbances: severity, treatment, and outcomes](#). Clin Exp Emerg Med 2017;4:73–79. Accessed 10Feb25.
2. LottC, TruhlářA, AlfonzoA et al. [European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances](#). Resuscitation. 2021;161:152-219. Accessed 10Feb25.
3. AlfonzoA, HarrisonA, BainesR et al. [Clinical Practice Guidelines - Treatment of Acute Hyperkalaemia in Adults](#). UK Kidney Association Oct 2023. Accessed 10Feb25.
4. NICE (2019) Sodium zirconium cyclosilicate for treating hyperkalaemia. [TA599](#). London: National Institute for Health and Care Excellence. Accessed 10Feb25.
5. Wills MR. Pseudohyperkalemia [Internet]. Lyngby, Denmark: Radiometer Medical ApS; [date unknown]. Available from: <https://acute care testing.org/en/articles/pseudohyperkalemia>.
6. Weingart SD. IBCC chapter & cast – Hyperkalemia [Internet]. EMCrit Project. Available from: <https://emcrit.org/ibcc/hyperkalemia/> Accessed 18May25.
7. London Kidney Network. Sick day rules: guidance for people with kidney disease [Internet]. London: NHS London Kidney Network; 2024. Available from: <https://londonkidneynetwork.nhs.uk/wp-content/uploads/2025/01/LKN-Sick-Day-RulesDec24-final-v2.2.pdf> Accessed 10Feb25.

### **6. Key Words**

Hyperkalaemia, potassium, pseudohyperkalaemia, emergency, treatment, bicarbonate, sodium zirconium cyclosilicate, Szc, glucose, insulin, salbutamol, renal, failure, acute kidney injury, dialysis, haemofiltration, AKI, ITU, ICU, intensive care, ECG, electrocardiogram, resuscitation, cardiac arrest, CPR, mortality, death

CONTACT AND REVIEW DETAILS	
<b>Guideline Lead (Name and Title)</b> Martin Wiese, Emergency Physician	<b>Executive Lead</b> Andrew Furlong, Medical Director
<b>Details of Changes made during review:</b> <ul style="list-style-type: none"><li>Extensive formatting changes – including presentation of the initial management guidance as a proforma</li><li>Content adjusted to changes in UK guidance, fully compliant with UKKA 2023 guideline</li><li>Cation-exchange resins (e.g. calcium resonium) removed from in-hospital hyperkalaemia care</li><li>References to Nervecentre hyperkalaemia order sets and dose sentences made throughout</li><li>Explicit information regarding repeated rounds of 'shifting treatments' added</li><li>Detailed information regarding options for fluid resuscitation added</li><li>Safety criteria for transfer to the Renal Unit added</li><li>Information about causes (including medicines) and interventions to help prevent recurrence added</li></ul>	

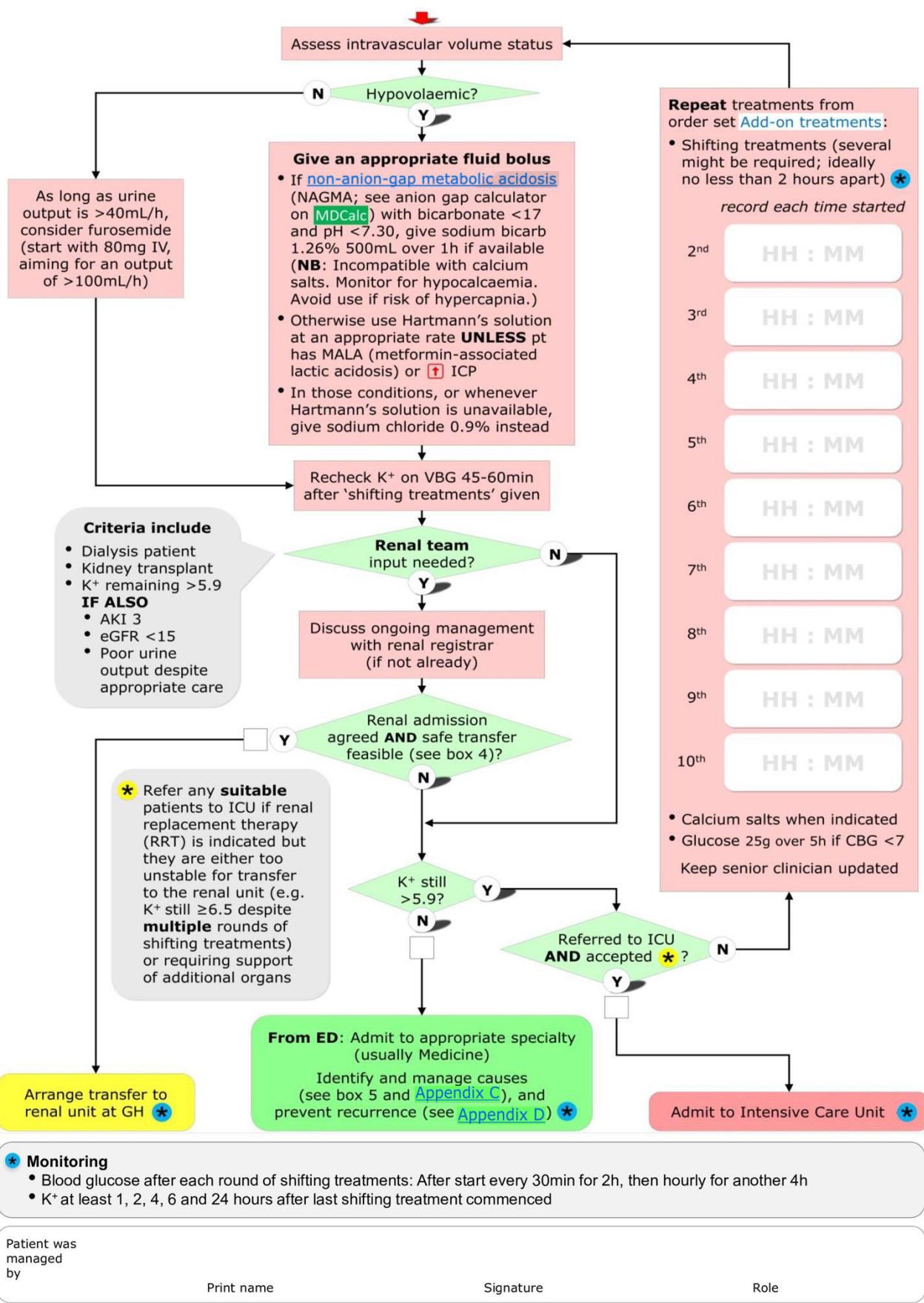
## Appendix A. Hyperkalaemia management proforma - page one.

<p>Developed by Martin Wiese · Version 76</p> <p><b>Emergency and Specialist Medicine</b></p> <p><b>Acute hyperkalaemia in adults</b></p> <p><b>Use in the ED and inpatients areas for patients with <math>K^+ 5.5</math> or above</b></p> <p><b>DO NOT use in</b></p> <ul style="list-style-type: none"> <li>• Diabetic ketoacidosis; follow DKA guideline (<a href="#">B66/2011</a>)</li> <li>• Dialysis patients with elevated <math>K^+</math> levels but <b>NO</b> hyperkalaemic ECG changes immediately prior to a dialysis session</li> <li>• Pts sent to ED from primary care (use guideline <a href="#">C41/2020</a> instead)</li> </ul> <p><b>Disclaimer:</b> This is a clinical template; clinicians should always use judgment when managing individual patients</p>	<p>Re-approved by UHL PGC on 13Nov2015 · Review due: Nov 2030 · Trust Ref: B28/2015</p> <p><b>Patient details</b></p> <p>Full name</p> <p>DoB</p> <p>Unit number</p> <p>(use sticker if available)</p>	<p><b>① Hyperkalaemic ECG changes?</b></p> <p><input checked="" type="checkbox"/> <b>Yes</b> - one or more of the below</p> <ul style="list-style-type: none"> <li>Peaked T waves (narrow base, high amplitude, sharp pointy apex and usually symmetrical)</li> <li>Absent or flattened P waves</li> <li>Wide QRS (<math>&gt;0.12</math>sec)</li> <li>Ventricular tachycardia (VT)</li> <li>Merging S and T ('sine') waves</li> <li>Bradycardia (sinus / AV block)</li> <li>Pseudo-STEMI</li> <li>Brugada phenocopy</li> </ul> <p><input type="checkbox"/> <b>No</b> - none of the above</p> <p><b>② Shifting treatments</b></p> <p>administer both at the same time</p> <p><b>Salbutamol nebulised solution</b></p> <p>Usual dose is 20mg (5mg x4 back-to-back) Reduce to 10mg (5mg x2 back-to-back) if coronary artery disease, tachyarrhythmia or open angle glaucoma</p> <p><b>Insulin-glucose IV infusion</b></p> <p>Add Actrapid 10units to 50mL glucose 50mL (=25G), add mix to 100mL of 0.9% sodium chloride and infuse over 15min via pump</p> <p><b>NB:</b> These treatments are only temporising. <math>K^+</math> level will rise again within 2-6h unless <math>K^+</math> is removed from the body by diuresis (in response to fluid resuscitation or loop diuretics), <math>K^+</math> binders or, if those efforts are ineffective, haemodialysis or haemofiltration.</p> <p><b>③ Removing <math>K^+</math> from body</b></p> <p><b>Sodium zirconium cyclosilicate (S2C)</b></p> <p>Prescribe S2C (brand name Lokelma) 10G in 45mL of water TDS for three days (but stop once <math>K^+</math> is 5.0 or less; usually after 24-48h) Powder will not dissolve. Stir just before giving cup to patient; the liquid should be taken while still cloudy.</p> <p>If patient is taking 'azole' antifungals, anti-HIV medicines or tyrosine kinase inhibitors, administer S2C two hours before or after to avoid reducing pH-dependent bioavailability</p> <p>Hypokalaemia, gastro-intestinal issues and oedema are and potential risks</p> <p>S2C may be radiopaque; state 'patient is taking sodium zirconium cyclosilicate' in request if abdominal imaging is needed</p> <p><b>④ Is renal unit transfer safe?</b></p> <p><input checked="" type="checkbox"/> <b>Yes</b> - as ALL of the below</p> <ul style="list-style-type: none"> <li>Hyperkalaemic ECG changes resolved</li> <li><math>K^+ &lt;6.5</math></li> <li><math>pH &gt;7.2</math></li> <li>Bicarb &gt;12</li> <li>Lactate &lt;4</li> <li><math>SpO_2</math> in range</li> <li><math>FIO_2</math> 35% or less</li> <li>MAP &gt;65</li> <li>Heart rate &lt;131</li> <li>GCS &gt;12</li> </ul> <p><input checked="" type="checkbox"/> <b>No</b> - as not all of the above</p> <p><b>NB:</b> Renal consultant may agree to waive some of the above on a case-by-case basis</p> <p><b>⑤ Hyperkalaemia causes</b></p> <p>tick any that might apply in your patient</p> <ul style="list-style-type: none"> <li><input type="checkbox"/> <b>Medicines</b> (go to <a href="#">Appendix C</a> for a comprehensive list)</li> <li><input type="checkbox"/> Continuing (not pausing) the medicines below during acute dehydrating illness 'sick days' <b>RAASi</b> (i.e. ACE inhibitors, ARBs, MRAs), diuretics, metformin, NSAIDs or <b>SGLT2i</b></li> <li><input type="checkbox"/> Renal impairment (AKI / CKD / CCF / diabetes)</li> <li><input type="checkbox"/> CKD worsened by constipation, acidosis, poor diabetic control or taking <b>trimethoprim</b></li> <li><input type="checkbox"/> Urinary tract obstruction</li> <li><input type="checkbox"/> Adrenal insufficiency (Addison's, Type 4 RTA)</li> <li><input type="checkbox"/> Massive tissue injury (e.g. crush injury, rhabdomyolysis or severe burns)</li> <li><input type="checkbox"/> Tumour lysis syndrome</li> <li><input type="checkbox"/> Sickle cell disease</li> <li><input type="checkbox"/> Haemolysis</li> <li><input type="checkbox"/> <b>Transfusion</b> - especially if massive, using a pressure device or older / irradiated RBCs; young children are at particular risk</li> </ul>
--	--	--

The flowchart details the management of acute hyperkalaemia, starting with a decision point for cardiac arrest with  $K^+ \geq 6.5$ . If yes, it leads to resuscitation with calcium chloride and urgent blood gas analysis. If no, it moves to repeat sampling and ECG monitoring. The process then branches into managing ECG changes (if present) or shifting potassium into cells (if  $K^+ > 6.4$  and no ECG changes). It includes steps for glucose administration if  $CBG < 7$  and monitoring potassium levels. The flowchart concludes with a section on mild hyperkalaemia and a summary of causes.

## Appendix B. Hyperkalaemia management proforma - page two.

### ⑥ Emergency hyperkalaemia treatment continued



Martin Wiese . Version 76 . Re-approved by UHL PGC on 13Nov25 . Review due: Nov 2030 . Trust Ref: B28/2015

## Appendix C. Medicines that can cause raised K<sup>+</sup> and suggested prescribing strategy.

- *Commonly implicated medicines are highlighted in BLUE*
- *Consider printing this page for your patient's records (ticking the relevant medicines)*

### Drugs that affect aldosterone secretion / related effects

- ACE inhibitors**  
(inhibit conversion of Angiotensin I to Angiotensin II)
- Angiotensin receptor blockers (ARBs)**  
(inhibit activation of Angiotensin IR by Angiotensin II)
- NSAIDs and COX-2 inhibitors**  
(inhibit renin release)
- Renin inhibitors (e.g. aliskiren)
- Calcineurin inhibitors (e.g. ciclosporin or tacrolimus)  
(also inhibit Na<sup>+</sup>/K<sup>+</sup>-ATPase necessary for K<sup>+</sup> secretion)
- Heparins including LMWH (reduce aldosterone production)
- Antifungals (e.g. ketoconazole, fluconazole and itraconazole)  
(suppress aldosterone synthesis)

### Drugs that block aldosterone binding to mineralocorticoid receptor (MRA)

- Spironolactone**, eplerenone
- Finerenone (a non-steroidal MRA)

### Drugs that inhibit activity of epithelial sodium channel

- Potassium sparing diuretics** (e.g. amiloride and triamterene)
- Trimethoprim and co-trimoxazole**
- Pentamidine

### Drugs that alter transmembrane potassium movement

- Beta blockers** (atenolol, metoprolol, propranolol)
- Digoxin at toxic levels** (inhibits Na<sup>+</sup>/K<sup>+</sup>-ATPase )
- Intravenous cationic amino acids
- Hyperosmolar solutions (e.g. mannitol or high-strength glucose)
- Suxamethonium, especially in burns, major trauma and infection
- Octreotide (suppresses insulin secretion)
- Metformin (through MALA; metformin-associated lactic acidosis)

### Potassium containing agents

- K<sup>+</sup> supplements** (e.g. Sando-K<sup>®</sup> and Kay-Cee L Liquid<sup>®</sup>)
- Salt substitutes (e.g. LoSalt)
- Herbal remedies  
(e.g. alfalfa, dandelion, horsetail, milkweed and nettle)
- Laxatives (e.g. Movicol<sup>®</sup>, Laxido<sup>®</sup>, Klean-Prep<sup>®</sup> and Fybogel<sup>®</sup>)

### Suggested prescribing strategy

- Medicines listed under **sick day guidance** above should be **PAUSED**, with a plan to **RESTART** them when recovered. For more info see 'Think Kidneys' [guidance](#).
- **STOP** any other implicated medicines if it is safe to do so, or **REPLACE** them with suitable alternatives if indications remain
- Seek advice from renal team if a medicine can be neither be stopped nor replaced

## Appendix D. Hyperkalaemia prevention.

	<b>Primary</b> ... to avoid an initial episode of hyperkalaemia	<b>Secondary</b> ... to avoid recurrence
<b>Non-dialysis patients</b>	<p><b>Regular blood monitoring for patients at risk</b> e.g. those with CKD, heart failure or diabetes as well as any patients taking RAASi medications</p> <p><b>Address modifiable factors</b></p> <ul style="list-style-type: none"><li>Avoid drug combinations that potentiate hyperkalaemia (e.g. trimethoprim in a patient taking an ACE inhibitor)</li><li>Correct acidosis (using a cause-specific approach)</li><li>Avoid and treat constipation</li><li>Optimise diabetic control</li><li>Dietary modifications where indicated: Seek specialist dietary advice for those with CKD 4 - 5</li></ul> <p><b>Anticipate risk of hyperkalaemia in acute illness</b></p> <ul style="list-style-type: none"><li>Consider who is at risk at time of hospital admission</li><li>Consider need to withhold drugs that potentiate hyperkalaemia during admission</li><li><b>Sick day guidance:</b> Advise those with diabetes, kidney or cardiovascular disease to pause <b>RAASi medications (i.e. ACEi, ARBs and MRAs), diuretics, metformin, SGLT2i, NSAID and COX-2 inhibitors</b> during any acute dehydrating illness (e.g. D&amp;V) until recovered</li></ul>	<p><b>Same as for primary prevention AND</b></p> <ul style="list-style-type: none"><li>If a K<sup>+</sup>-binder is given during this admission, monitor for recurrence of hyperkalaemia when binder is discontinued</li><li>If patient has no heart failure and no CKD 3b – 5, <b>STOP</b> RAASi drugs</li><li>For patients with persistent K<sup>+</sup> ≥ 6.0 and heart failure or CKD 3b – 5 who benefit from RAASi medications, consider a K<sup>+</sup>-binder (SZC or patiromer). This may allow patient to <b>RESTART</b> RAASi medications at optimal doses after recovery and thus help avoid adverse clinical outcomes.</li><li>Consider diuretic in those with heart failure or CKD, particularly if volume overloaded</li></ul> <p><b>• Sick day guidance:</b> Check that the patient fully understands the advice given to them</p>
<b>Dialysis patients</b>	<p><b>Regular blood monitoring</b></p> <p><b>Address modifiable factors</b></p> <ul style="list-style-type: none"><li>Avoid prolonged fasting. If nil oral intake, consider a 10% glucose infusion at 42mL/h (= 100G in 24h) unless diabetic. If diabetic, use a VRIII with substrate instead.</li><li>Avoid and treat constipation</li><li>Optimise diabetic control</li><li>Dietary modifications (seek specialist dietitian input)</li></ul> <p><b>Address dialysis-related factors</b></p> <ul style="list-style-type: none"><li>Maintain good dialysis access</li><li>Optimise adequacy</li><li>Minimise re-circulation</li></ul> <p><b>K<sup>+</sup>-binders</b> Consider as bridge if dialysis delayed (e.g. access issues)</p>	<p><b>Same as for primary prevention AND</b></p> <ul style="list-style-type: none"><li>Increase frequency of blood monitoring (vigilance if high likelihood of recurrence)</li><li>K<sup>+</sup>-binders – potential role for chronic hyperkalaemia if other strategies fail</li></ul>