1. Introduction and Who Guideline applies to

Hyperkalaemia is defined as a serum potassium (K^+) concentration of **> 5.5mmol/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 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 mmol/L.

2.3 Repeated shifting treatments increase the risk of hypoglycaemia. If capillary blood glucose (CBG) is <7 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.
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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://acutecaretesting.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

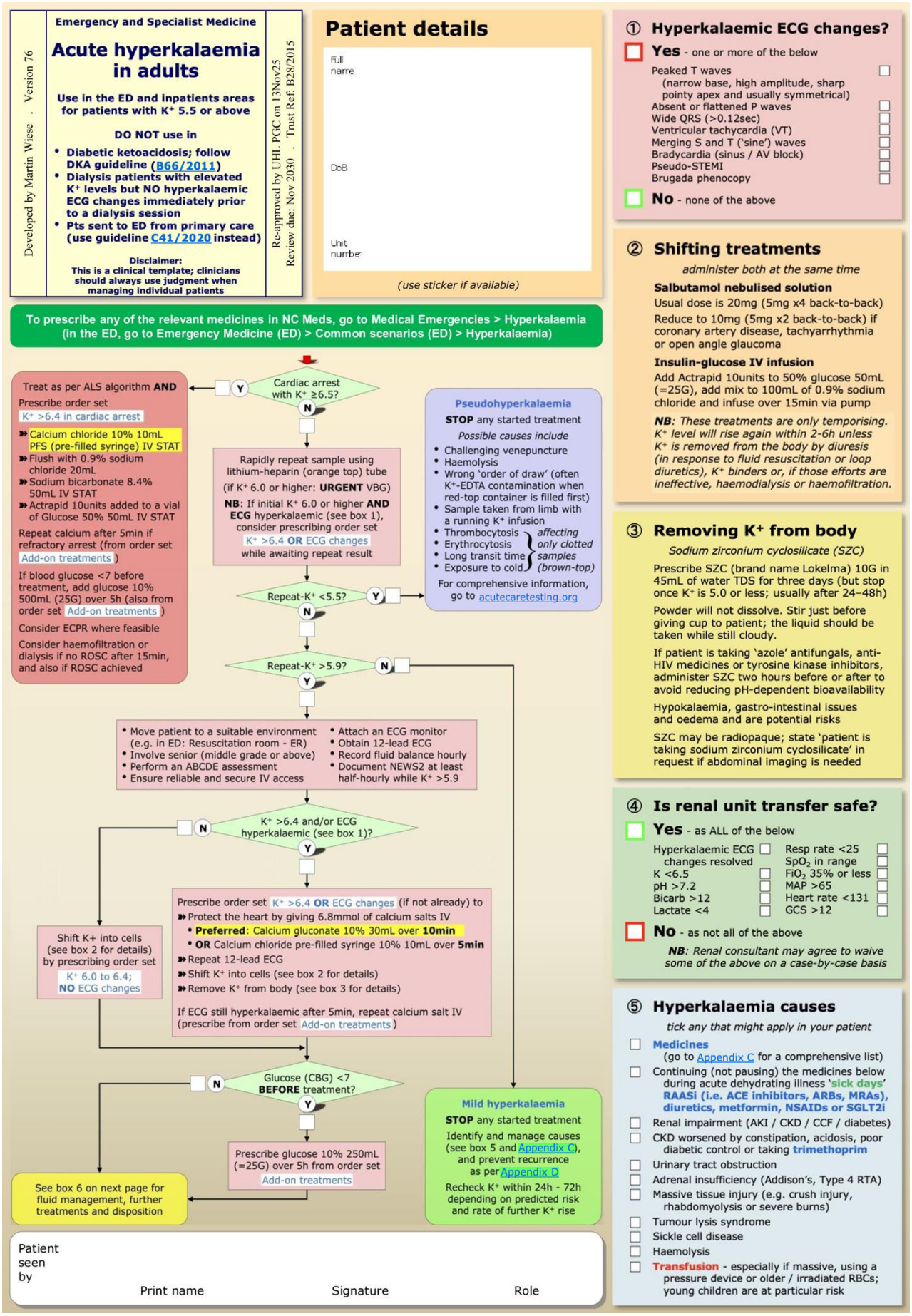
Hyperkalaemia, potassium, pseudohyperkalaemia, emergency, treatment, bicarbonate, sodium zirconium cyclosilicate, SZC, glucose, insulin, salbutamol, renal, failure, acute kidney injury, dialysis, haemofilitration, AKI, ITU, ICU, intensive care, ECG, electrocardiogram, resuscitation, cardiac arrest, CPR,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

CONTACT AND REVIEW DETAILS	
Guideline Lead (Name and Title) Martin Wiese, Emergency Physician	**Executive Lead** Andrew Furlong, Medical Director

Details of Changes made during review:

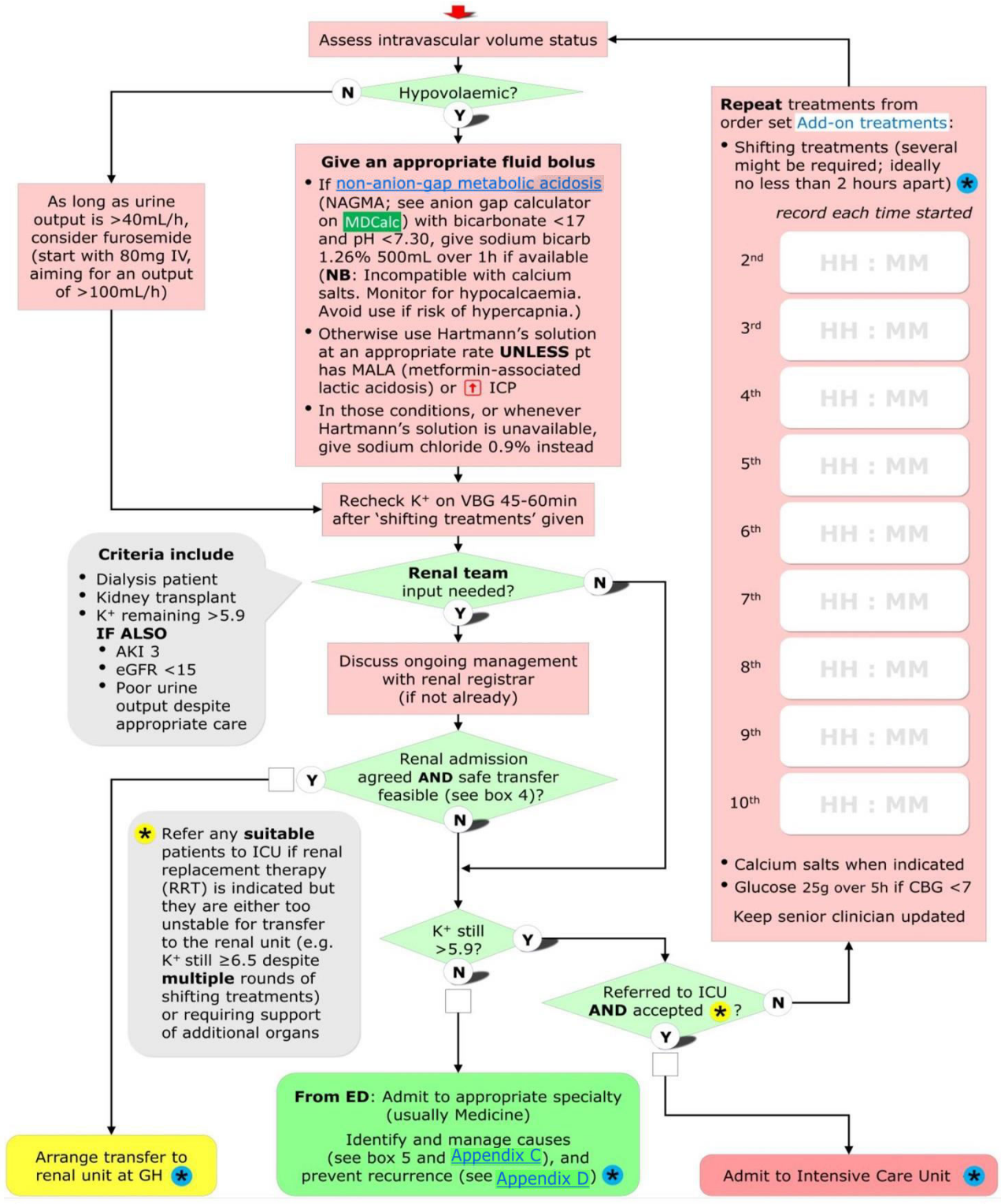
- Extensive formatting changes – including presentation of the initial management guidance as a proforma
- Content adjusted to changes in UK guidance, fully compliant with UKKA 2023 guideline
- Cation-exchange resins (e.g. calcium resonium) removed from in-hospital hyperkalaemia care
- References to Nervecentre hyperkalaemia order sets and dose sentences made throughout
- Explicit information regarding repeated rounds of ‘shifting treatments’ added
- Detailed information regarding options for fluid resuscitation added
- Safety criteria for transfer to the Renal Unit added
- Information about causes (including medicines) and interventions to help prevent recurrence added

Appendix A. Hyperkalaemia management proforma - page one.



Appendix B. Hyperkalaemia management proforma - page two.

⑥ Emergency hyperkalaemia treatment continued



*** Monitoring**

- Blood glucose after each round of shifting treatments: After start every 30min for 2h, then hourly for another 4h
- K+ at least 1, 2, 4, 6 and 24 hours after last shifting treatment commenced

Patient was managed by

Print name | Signature | Role

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

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.

	Primary *... to avoid an initial episode of hyperkalaemia*	**Secondary** *... to avoid recurrence*
Non-dialysis patients	**Regular blood monitoring for patients at risk** e.g. those with CKD, heart failure or diabetes as well as any patients taking RAASi medications **Address modifiable factors** • Avoid drug combinations that potentiate hyperkalaemia (e.g. trimethoprim in a patient taking an ACE inhibitor) • Correct acidosis (using a cause-specific approach) • Avoid and treat constipation • Optimise diabetic control • Dietary modifications where indicated: Seek specialist dietary advice for those with CKD 4 - 5 **Anticipate risk of hyperkalaemia in acute illness** • Consider who is at risk at time of hospital admission • Consider need to withhold drugs that potentiate hyperkalaemia during admission • **Sick day guidance**: Advise those with diabetes, kidney or cardiovascular disease to pause **RAASi medications (i.e. ACEi, ARBs and MRAs), diuretics, metformin, SGLT2i, NSAID and COX-2 inhibitors** during any acute dehydrating illness (e.g. D&V) until recovered	**Same as for primary prevention AND** • If a K^+-binder is given during this admission, monitor for recurrence of hyperkalaemia when binder is discontinued • If patient has no heart failure and no CKD 3b – 5, **STOP** RAASi drugs • For patients with persistent $K^+ \geq 6.0$ and heart failure or CKD 3b – 5 who benefit from RAASi medications, consider a K^+-binder (SZC or patiromer). This may allow patient to **RESTART** RAASi medications at optimal doses after recovery and thus help avoid adverse clinical outcomes. • Consider diuretic in those with heart failure or CKD, particularly if volume overloaded • **Sick day guidance**: Check that the patient fully understands the advice given to them
Dialysis patients	**Regular blood monitoring** **Address modifiable factors** • 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. • Avoid and treat constipation • Optimise diabetic control • Dietary modifications (seek specialist dietician input) **Address dialysis-related factors** • Maintain good dialysis access • Optimise adequacy • Minimise re-circulation **K^+-binders** Consider as bridge if dialysis delayed (e.g. access issues)	**Same as for primary prevention AND** • Increase frequency of blood monitoring (vigilance if high likelihood of recurrence) • K^+-binders – potential role for chronic hyperkalaemia if other strategies fail