Hyperkalemia

Protect · shift · remove · then find out why it happened

K ≥ 6.5
or any ECG change

TREAT NOW IF K ≥ 6.5, OR AT ANY LEVEL WITH ECG CHANGES OR RAPID RISE

Get a 12-lead and put the patient on a monitor. The classic sequence is **peaked T waves → flat P and long PR → wide QRS → sine wave → arrest**, but **the ECG is insensitive and around half of dangerous hyperkalemia shows no classic change at all**, so treat the number and the trend rather than the tracing. A **bradycardic or wide-complex rhythm in a dialysis patient is hyperkalemia until proven otherwise**.

THE THREE STEPS *protect the myocardium, shift potassium inward, then actually remove it*

Step	Agent and dose	Why, and what to watch
1 · PROTECT *onset minutes, lasts 30–60 min*	**Calcium gluconate 10%, 10 mL IV over 2–3 min.** Repeat if the ECG has not improved in 5–10 min. **Calcium chloride 10 mL** gives 3× the elemental calcium but needs central access.	Raises the threshold potential and **stabilizes the myocardium without lowering the potassium at all**. It buys minutes, so it must always be followed by shift and removal. Peripheral calcium chloride causes tissue necrosis if it extravasates. **Never run calcium and bicarbonate through the same line**, they precipitate.
2 · SHIFT *onset 15–30 min, lasts 2–6 h*	**Insulin 10 units IV with 25 g dextrose.** Consider **5 units** if eGFR is low. **Albuterol 10–20 mg nebulized** (4–8× the bronchodilator dose). **Bicarbonate only if genuinely acidotic.**	Drops K by roughly **0.6–1.2** mEq/L, then it comes back. **Hypoglycemia is the main harm, in 20–30% of patients**, and 5 units halves severe events in renal impairment while lowering K a little less. **Check glucose at 1 h and every 1–2 h for 6 h.** Up to a third of patients do not respond to albuterol at all.
3 · REMOVE *the only step that changes the balance*	**Hemodialysis** is definitive, especially in AKI or oliguria. **Loop diuretic** if the patient still makes urine. **Oral binder** (SZC or patiromer). Stop the culprit drugs and all potassium.	Shifting is temporary, so **K rebounds within 2–6 h unless it leaves the body**. Call nephrology early rather than after the third rebound. Binders act too slowly for the acute emergency but prevent the next one.

FIND THE CAUSE *almost always reduced excretion, a drug, or cells spilling their contents*

REDUCED EXCRETION

AKI and advanced CKD are the commonest causes. Then **adrenal insufficiency**, **type 4 RTA** (see below), and **effective volume depletion**, where poor distal sodium delivery leaves nothing to exchange potassium for.

DRUGS

ACE inhibitors and ARBs, **spironolactone and eplerenone**, amiloride, **NSAIDs**, **trimethoprim** (blocks ENaC exactly as amiloride does), **heparin** (suppresses aldosterone synthesis), **calcineurin inhibitors**, non-selective beta-blockers, digoxin toxicity, succinylcholine, pentamidine, and potassium supplements or salt substitutes.

CELL RELEASE AND SPURIOUS

Rhabdomyolysis, tumor lysis, hemolysis, burns, crush injury, acidosis. Then rule out **pseudohyperkalemia**: a hemolyzed sample, a clenched fist under the tourniquet, a delayed specimen, or marked **leukocytosis or thrombocytosis**, where potassium leaks out during clotting. **Recheck on fresh plasma if the result does not fit the patient.**

TYPE 4 RTA *the cause people forget: mild kidney disease with unexplained, persistent hyperkalemia*

	Type 4 renal tubular acidosis (hyporeninemic hypoaldosteronism)
The lesion	**Too little aldosterone, or a distal tubule that cannot respond to it.** Aldosterone normally drives sodium reabsorption in exchange for potassium and hydrogen, so losing it impairs excretion of **both**. That is why the picture is hyperkalemia **and** acidosis together.
The picture	**Normal anion gap (hyperchloremic) metabolic acidosis with hyperkalemia**, in a patient whose creatinine is only mildly to moderately raised. **Urine pH is usually below 5.5**, which separates it from type 1 RTA, where acidification fails and the urine stays inappropriately alkaline. The hyperkalemia is out of proportion to the degree of kidney impairment, and that mismatch is the clue.
Who gets it	**Diabetic nephropathy is the classic setting**, then chronic interstitial disease, obstructive uropathy, HIV, sickle cell, and lupus. Drug causes overlap with the list above: **ACE inhibitors and ARBs, spironolactone, NSAIDs, trimethoprim, heparin, calcineurin inhibitors**.
Treatment	**Stop or reduce the offending drug first**, since that alone often fixes it. Then a **low-potassium diet**, a **loop or thiazide diuretic** to increase distal sodium delivery, and **sodium bicarbonate** if the acidosis persists, which also shifts potassium. **Fludrocortisone 0.1 mg daily** is a last resort, because the mineralocorticoid load worsens hypertension, edema, and heart failure in exactly the population that has this diagnosis.

ONGOING CONTROL *the acute fix lasts hours; this is what stops the readmission*

Option	Dose	Where it fits
Sodium zirconium cyclosilicate	**10 g PO three times daily for up to 48 h**, then **5–10 g daily**	Onset within about an hour, so it is the binder of choice when you want potassium gone rather than hidden. **Carries a sodium load**, so watch for edema in heart failure and cirrhosis.
Patiromer	**8.4 g PO daily**, titrated weekly	Onset around **7 h**, so it is a maintenance drug, not a rescue. **Binds magnesium as well**, so check magnesium, and **separate it from other oral drugs by 3 h**.
Sodium polystyrene sulfonate	Largely superseded	Slow, poorly evidenced, and associated with **colonic necrosis**, particularly with sorbitol and in postoperative or ileus patients.
Drugs and diet	Review at every visit	**Do not reflexively stop the ACE inhibitor, ARB, or MRA in heart failure or CKD**, where they carry a mortality benefit. Dose-reduce, add a binder, and correct acidosis instead. **Ban salt substitutes**, which are potassium chloride.

MONITOR AND DISPOSITION *the rebound is the part that catches people out*

AFTER TREATMENT

Cardiac monitor until K < 6.0 and the ECG has normalized. Recheck potassium at 1 h and again at 2–4 h, because insulin and albuterol wear off together and the level climbs back. Recheck **glucose** on the same schedule. **Do not discharge on the strength of a single post-treatment potassium** if nothing has been removed from the body.

WHEN TO CALL RENAL EARLY

Refractory or rapidly rising potassium, oliguric AKI, dialysis dependence, or hyperkalemia driven by ongoing tissue breakdown such as rhabdomyolysis or tumor lysis, where potassium arrives faster than you can shift it. **Shifting only postpones the dialysis, so make the call at the start.**

PEARLS & PITFALLS

- **Calcium protects but does not lower potassium.** Giving it and stopping there is the classic error; it must always be followed by shift and removal.
- **Insulin and albuterol only move potassium into cells.** Unless it leaves the body, the level rebounds in 2–6 h.
- **Recheck glucose after insulin**, since hypoglycemia is common and late.
- **A normal ECG does not exclude danger.** Sensitivity is poor, so treat the number and the trend.
- **Unexplained hyperkalemia in a diabetic with mild CKD is type 4 RTA** until proven otherwise.
- **Check the sample before the guidelines.** Hemolysis explains a lot of this.

References: UK Kidney Association Treatment of Acute Hyperkalemia in Adults · KDIGO and standard critical care references · trials of insulin dosing in renal impairment · patiromer and sodium zirconium cyclosilicate prescribing information.

Educational aid; verify doses against local protocol, renal function, and patient factors.