Management of Acute and Chronic Hyperkalemia

Hyperkalemia is generally defined as a serum potassium level greater than 5.5 mEq (mmol)/L, but may vary by lab.[16] Hyperkalemia can be asymptomatic, or cause nonspecific symptoms such as muscle pain or weakness, nausea, paresthesia, or palpitations.[16] Moderate or severe hyperkalemia can cause potentially life-threatening arrhythmias.[16] Common causes of hyperkalemia include kidney failure, chronic kidney disease, heart failure, and certain medications (e.g., ACEIs, ARBs, potassium supplements, potassium-sparing diuretics, NSAIDs, trimethoprim-sulfamethoxazole).[7,14,23,25] Hyperkalemia may be the result of one or more possible mechanisms including increased potassium intake, potassium shifts from intracellular to extracellular space, or reduced potassium excretion.[25] The chart below reviews the management of acute and chronic hyperkalemia.

Medication/Method	Suggested Use/Pertinent Information

ACUTE/severe hyperkalemia: (K^+ ≥6.5 mEq [mmol]/L **with or without** EKG changes),[2,33] or EKG changes with any elevated potassium level.[25]
• Requires urgent correction[25] and should be managed in an inpatient setting.[1]
• Management strategy is often independent of underlying cause.[2]
• Goals of therapy:[16]
  ○ protect the heart against the effects of potassium (e.g., arrhythmias)
  ○ shift extracellular potassium into cells
  ○ increase potassium elimination
  ○ prevent recurrence (see chronic hyperkalemia section, below)	

Calcium	• Calcium GLUCONATE is usually preferred over calcium CHLORIDE.[2] Calcium CHLORIDE has about **three** times higher calcium content and is associated with more adverse events (e.g., bradycardia, more tissue damage with extravasation).[2] Calcium CHLORIDE is usually reserved for use during a code.[2]
	Calcium GLUCONATE:
	• **How it works**: Does NOT reduce potassium level.[16] Membrane potential stabilization normalizes EKG changes.[16]
	• **ADULT dose**: **usually** 1 gm given IV piggy back[2]
	  ○ Can give IV push (e.g., 1 gm over 5 minutes).[4] Rapid administration increases risk of bradycardia, hypotension, arrhythmias, and cardiac arrest.[4]
	  ○ Do not exceed maximum rate of 200 mg/minute in adults.[4]
	  ○ May repeat once 5 minutes after first dose if ineffective or EKG changes recur.[25]
	  ○ For **cardiac arrest** associated with known or suspected hyperkalemia, 1.5 to 3 g IV or IO over 2 to 5 minutes should be given.[31] Calcium chloride could also be used, but dosing is different.[31]
	• **Onset**: within 1 to 3 minutes[16]
	• **Duration of effect**: 15 minutes to 1 hour[25]
	• **Adverse effects**: local irritation, hypercalcemia, hypotension, bradycardia[4]

Medication/Method	Suggested Use/Pertinent Information
ACUTE/severe hyperkalemia, continued	

Insulin (regular)	**Regular insulin**
	• **How it works**: Shifts potassium from extracellular to intracellular space.[25]
	  o Most reliable of the medications used to shift potassium intracellularly.[24]
	• **ADULT dose**: 5 to 10 units (or 0.1 units/kg, max 10 units) with dextrose 25 grams to help minimize hypoglycemia.[24,33]
	  o The 10 unit dose has the best evidence of efficacy.[33]
	  o Also consider adding dextrose 10% at 50 mL/hr for five hours after the insulin/dextrose dose to reduce hypoglycemia risk, especially if glucose is ≤125 mg/dL (<7 mmol/L).[33]
	  o Check glucose often (e.g., at least hourly for six hours hours).[33]
	• **Onset**: within 15 to 30 minutes[25]
	• **Duration of effect**: 4 to 6 hours[16]
	• **Adverse effects**: hypoglycemia and hypokalemia[25]

Beta-agonists	**Albuterol (salbutamol [Canada])**
	• **How it works**: Shifts potassium from extracellular to intracellular space.[25]
	  o May be less effective in patients receiving a nonselective beta-blocker (e.g., propranolol, sotalol).[2,6]
	  o Inconsistent effect; consider using with insulin/dextrose.[25]
	• **ADULT dose**: 10 to 20 mg via nebulizer[5,24,30]
	• **Onset**: within 30 to 60 minutes[16,25,30]
	• **Duration of effect**: 2 to 4 hours[16,25]
	• **Adverse effects**: At these doses, tachycardia, tremor, vasoconstriction, and hyperglycemia may be of concern.[5,24,30]

Sodium bicarbonate	• **How it works**: May slightly shift potassium from extracellular to intracellular space or may increase potassium elimination, especially when combined with diuretics.[2,5]
	  o Not for routine use.[33]
	  o May work best in acidosis (pH <7.2).[25]
	• **ADULT dose**: 50 mEq (mmol) = 50 mL of 8.4% sodium bicarbonate over 5 minutes.[5] Repeat in 30 minutes as needed.[25]
	• **Onset**: variable; 30 minutes to 4 hours[25]
	• **Duration of effect**: ~2 hours[16]
	• **Adverse effects**: hypocalcemia (monitor ionized calcium), metabolic alkalosis, hypernatremia, fluid overload,[24] worsening hypertension or heart failure[16]

Medication/Method	Suggested Use/Pertinent Information
ACUTE/severe hyperkalemia, continued	
Loop diuretics	• **How they work**: Increase urinary potassium excretion.[5] o May be ineffectiveness in severe renal failure.[24] Adults who do not produce at least 200 mL of urine within 2 hours of a dose of furosemide 1 to 1.5 mg/kg should be considered nonresponders, and alternate treatment should be started.[24] • **ADULT dose**: furosemide 40 mg IV (or 1 to 1.5 mg/kg in acute kidney insufficiency) or equivalent[16,24] • **Onset**: 5 to 30 minutes[27] • **Duration of effect**: 2 to 6 hours[27] • **Adverse effects**: low electrolytes (calcium, phosphorus, potassium, magnesium, sodium), metabolic alkalosis, dehydration[4,24]
Potassium binders Sodium polystyrene sulfonate (generics, *Kayexalate* [Canada], *Solystat* [Canada]) Calcium polystyrene sulfonate (*Resonium Calcium* [Canada]) Patiromer (*Veltassa*) Sodium zirconium cyclosilicate (*Lokelma*) *Continued…*	• **How they work**: Exchange sodium (sodium polystyrene sulfonate), sodium and hydrogen (sodium zirconium cyclosilicate), or calcium (calcium polystyrene sulfonate, patiromer) for potassium in the GI tract to reduce absorption and increase elimination.[4,9] o **Not for monotherapy** in life-threatening hyperkalemia.[16,22] o A third-step option in acute treatment, after protecting the heart (i.e., with calcium gluconate) and shifting potassium into the cells (e.g., with insulin plus dextrose).[33] Work more slowly than loop diuretics. o All seem to provide comparable potassium reduction within 24 hours with a single dose (sodium polystyrene sulfonate 15 g, sodium zirconium cyclosilicate 10 g, patiromer 8.4 g).[35-37] • **ADULT dose**: oral administration more reliably lowers potassium levels compared to rectal administration[9,28] o **Sodium polystyrene sulfonate**: 15 grams one to four times daily (orally) or 30 to 50 grams every 6 hours (Canada: once or twice daily at 6-hour intervals) (rectally)[8,15] o **Calcium polystyrene sulfonate**: 15 grams three to four times daily (orally) or 30 grams (rectally)[9] o **Patiromer**: 8.4 mg once daily. Dose can be titrated weekly by 8.4 mg to a max of 25.2 grams orally.[10,29] o **Sodium zirconium cyclosilicate**: 10 grams three times daily for up to 48 hours, then 10 grams once daily (Canada: 5 mg once daily).[12,20] Maintenance dose titrated to response (U.S., ≥ weekly). Range 5 grams every other day to 15 grams once daily (Canada: 10 mg once daily).[12,20] Dialysis: 5 to 15 mg once daily on non-dialysis days.[12,20] • **Onset**: 1 to 6 hours (**sodium zirconium cyclosilicate**), 2 to 6 hours (oral **sodium polystyrene sulfonate**), or 4 to 7 hours (**patiromer**)[22,26,33] • **Duration of effect**: 4 to 6 hours (**oral sodium polystyrene sulfonate**),[16] 24 to 48 hours (calcium polystyrene sulfonate),[9] 4 to 12 hours (**sodium zirconium cyclosilicate**),[26] or 12 to 48 hours (**patiromer**)[16,26]

Medication/Method	Suggested Use/Pertinent Information
ACUTE/severe hyperkalemia, continued	

Potassium binders, continued	• **Drug interactions**:
	  ○ **Sodium and calcium polystyrene sulfonate**: Separate from oral meds by at least 3 hours (before and after) to avoid potential interactions.[8,9,13,15]
	  ▪ Consider separating oral meds by 6 hours (before and after) in patients with gastroparesis.[8.9,13,15]
	  ○ **Patiromer**: Separate from oral meds by at least 3 hours (before and after) to avoid potential interactions (exceptions are listed in the product labeling).[10,29]
	  ○ **Sodium zirconium cyclosilicate**: Separate from oral meds (Canada: drugs with pH-dependent bioavailability [e.g., atorvastatin, dabigatran, azoles, protease inhibitors]) by at least 2 hours (before and after) to avoid potential interactions.[12,20]
	• **Adverse effects**: intestinal necrosis (usually when mixed with sorbitol) (**sodium/calcium polystyrene sulfonate**);[8,9,15] constipation, hypomagnesemia (**sodium/calcium polystyrene sulfonate, patiromer**);[8-10,15,29] **bad taste** (sodium/calcium polystyrene sulfonate),[9,22] hypocalcemia (**sodium polystyrene sulfonate**);[8,15] edema (**sodium polystyrene sulfonate**, **sodium zirconium cyclosilicate**);[8,12,15,20] **hyper**calcemia (**calcium polystyrene sulfonate**)[9]
	  ○ **Sodium zirconium cyclosilicate** does not appear to cause significant electrolyte abnormalities (e.g., hypomagnesemia, hyponatremia, hypocalcemia).[12,20,21]

Dialysis or other renal replacement therapy	• **How it works**: Increases potassium elimination from the body.[25]
	  ○ For patients with hyperkalemia and acute kidney injury, or for patients who are refractory to medical therapy[24]
	  ○ Hemodialysis lowers potassium levels more effectively than peritoneal dialysis.[25]
	• **Onset**: within minutes[16]
	• **Duration of effect**: until the end of dialysis session and possibly longer[16]
	  ○ Be aware that pre-dialysis treatment with drugs that shift potassium intracellularly (e.g., insulin, beta-agonist) will reduce potassium removal during dialysis.[24]
	  ○ Watch for rebound in potassium level after intermittent dialysis session (continuous modalities pose lower risk of rebound).[24]

Hypertonic saline	• **How it works**: Does NOT lower potassium levels, but protects the heart and reduces potential for arrhythmias.[16] ○ **Only useful in patients with concomitant hyponatremia** (rarely used)[16] • **ADULT dose**: 50 to 250 mL of 3% to 5% saline IV (central line preferred).[4,16] See our chart, *Hyponatremia and Hypernatremia*, for more about the safe administration of hypertonic saline. • **Onset**: 5 to 10 minutes[16] • **Duration of effect**: up to 2 hours[16] • **Adverse effects**: May lead to volume overload, hypertension, heart failure.[4]

Medication/Method	Suggested Use/Pertinent Information

CHRONIC/mild to moderate hyperkalemia: (K+ >5 mEq [mmol]/L)[1]
• Can be safely managed in an outpatient setting.[1]
• Management strategy often determined by underlying cause.[2]
• Goals of therapy:[16]
  o increase potassium elimination from the body
  o prevent recurrence	

Limit dietary potassium intake	• Used to prevent recurrence of hyperkalemia.
	• Limit intake from all sources to 40 to 60 mEq (mmol)/day (e.g., food, salt substitutes, supplements).[1]
	• Poor patient adherence to low-potassium diets may be due to:[11]
	  o constipation or other gastrointestinal issues associated with these diets
	  o lack of awareness of potassium content in foods and salt substitutes

Diuretics	• Consider especially for patients with another indication for a diuretic (e.g., hypertension, heart failure). • Lowers potassium levels by increasing potassium elimination.[16] • Loops are preferred in patients with an estimated glomerular filtration rate (eGFR) <40 mL/min/1.73 m^2.[16] • Combination products with thiazide plus ACEIs or ARBs are available. • Use with caution in patients with gout, diabetes, or patients that are volume depleted.[4] • Effectiveness declines as renal function declines.[16]
SGLT2 inhibitors	• Associated with lower potassium, but not studied in a trial in which potassium reduction was the primary outcome.[3] • Consider when otherwise indicated (e.g., diabetes, heart failure, chronic kidney disease).[3]
Potassium binders	• See details in "Acute" section, above. • Sodium polystyrene sulfonate data from randomized trials is very limited.[22] Although sodium/calcium polystyrene sulfonate haven't been compared head-to head, the newer agents (patiromer and sodium zirconium cyclosilicate) seem to cause less severe GI adverse effects[8-10,12,15,20,29] and be better tolerated. • For patients with heart failure or renal failure, consider patiromer to avoid sodium load.[22]

Address contributing medications	• Several medications have the potential to increase potassium levels:[14,17-19,25,32,34]
	  o ACEIs
	  o ARBs
	  o beta-blockers (nonselective)
	  o calcineurin inhibitors (e.g., tacrolimus, cyclosporine)
	  o dietary supplements (e.g., dandelion, noni juice, Siberian ginseng, milkweed, Hawthorn berries, nettle).
	  o NSAIDs
	  o potassium-sparing diuretics (e.g., amiloride, spironolactone, triamterene)
	  o trimethoprim-sulfamethoxazole

Medication/Method	Suggested Use/Pertinent Information
CHRONIC/mild to moderate hyperkalemia, continued	
Contributing meds, continued	• Look for alternative antibiotic options instead of trimethoprim-sulfamethoxazole in patients with an elevated baseline potassium level or risk factors for hyperkalemia (e.g., chronic kidney disease, heart failure, certain medications).[14,18] o Monitor potassium if concomitant use of trimethoprim-sulfamethoxazole with a medication that elevates serum potassium (e.g., ACEI, ARB, spironolactone) is unavoidable.[4] • When making decisions regarding dose reduction of ACEI, ARB, spironolactone, or eplerenone in heart failure or post-MI, see our charts, *Target Doses of Medications for Heart Failure* and *Target Dose of Post-MI Medications*. • Some clinicians give scheduled potassium binder doses to avoid dosage reductions or discontinuation of therapy for evidence-based regimens (e.g., ACEIs or ARBs in patients with chronic kidney disease, heart failure, post-MI).[22] Patiromer and sodium zirconium cyclosilicate have been studied for this purpose.[23]

Abbreviations: ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; EKG = electrocardiogram; IO = intraosseous; IV = intravenously; GI = gastrointestinal; K^+ = potassium; MI = myocardial infarction.

Users of this resource are cautioned to use their own professional judgment and consult any other necessary or appropriate sources prior to making clinical judgments based on the content of this document. Our editors have researched the information with input from experts, government agencies, and national organizations. Information and internet links in this article were current as of the date of publication.

Levels of Evidence

In accordance with our goal of providing Evidence-Based information, we are citing the **LEVEL OF EVIDENCE** for the clinical recommendations we publish.

Level	Definition	Study Quality
A	Good-quality patient-oriented evidence.*	1. High-quality randomized controlled trial (RCT) 2. Systematic review (SR)/Meta-analysis of RCTs with consistent findings 3. All-or-none study
B	Inconsistent or limited-quality patient-oriented evidence.*	1. Lower-quality RCT 2. SR/Meta-analysis with low-quality clinical trials or of studies with inconsistent findings 3. Cohort study 4. Case control study
C	Consensus; usual practice; expert opinion; disease-oriented evidence (e.g., physiologic or surrogate endpoints); case series for studies of diagnosis, treatment, prevention, or screening.	

***Outcomes that matter to patients** (e.g., morbidity, mortality, symptom improvement, quality of life).

[Adapted from Ebell MH, Siwek J, Weiss BD, et al. Strength of Recommendation Taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. Am Fam Physician 2004;69:548-56. https://www.aafp.org/pubs/afp/issues/2004/0201/p548.html.]

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