

Hyperkalemia

High potassium (Hyperkalemia) — the heart's electrical safety margin is vanishing.

THE ONE-LINE TRUTH

Too much potassium in the blood shrinks the voltage gap heart muscle cells need to beat reliably.

The chain — how it unfolds

1. Rising blood potassium shrinks the voltage gap across the heart muscle cell membrane. — why: Normal resting potential is about -90 millivolts inside; threshold to fire is -70 millivolts — a safe 20-millivolt gap. High blood potassium makes the inside less negative (depolarizes it toward -80 or -75 millivolts), cutting the safety margin in half.
2. The smaller gap makes the heart cell fire unpredictably and sluggishly. — why: With only a 5- or 10-millivolt gap, tiny drifts in voltage can trigger a beat early or skip a beat entirely. At the same time, some of the fast sodium channels that create the sharp upstroke become stuck in an inactive state because the membrane never repolarizes fully, so the heartbeat loses speed and strength.
3. The T wave becomes tall and peaked first. — why: The T wave represents ventricular repolarization (the heart muscle resetting). When the membrane is already partway depolarized, repolarization happens faster and the T wave gets narrow and tall — this is often the first EKG clue, appearing around 6.0-6.5 mEq/L.
4. The P wave flattens and the PR interval stretches. — why: As potassium climbs above 6.5 mEq/L, the atria (top chambers) depolarize weakly because their thin muscle has fewer sodium channels to spare. The P wave shrinks or disappears. Conduction through the AV node (the relay station between atria and ventricles) slows, so the PR interval — the time from atrial start to ventricular start — gets longer.
5. The QRS complex widens into a thick, slurred shape. — why: The ventricles (bottom chambers) rely on the His-Purkinje system (fast highways of conducting fibers) to spread the signal quickly. High potassium slows conduction in these fibers, so the QRS — normally a sharp spike under 0.10 seconds — stretches to 0.12 seconds or more and loses its crisp peaks.

6. The EKG turns into a sine wave — a smooth, rolling curve with no clear P, QRS, or T. — why: When potassium rises above 8–9 mEq/L, depolarization and repolarization blur together. The membrane voltage drifts slowly up and down instead of snapping on and off, so the EKG looks like a gentle wave instead of sharp beats. This is pre-arrest.
7. The heart stops completely (asystole) or quivers uselessly (ventricular fibrillation). — why: With almost no voltage gap left, the cells cannot coordinate. Some freeze in a depolarized state (asystole — flat line), others fire randomly in tiny chaotic loops (ventricular fibrillation). Either way, the heart pumps no blood.
8. This entire cascade can happen in minutes if potassium spikes suddenly. — why: The heart muscle membrane responds to blood potassium in real time — there is no buffering step. A sudden release from cell lysis (crush injury, tumor lysis) or a rapid IV potassium infusion can push the level from 5.0 to 8.0 mEq/L in under an hour, and EKG changes follow within minutes.

Spot it — what you see, and why

- Often no symptoms at all until the level is dangerously high: Potassium quietly does its electrical work inside cells, so when blood levels creep up slowly, the body often doesn't ring alarm bells until the heart's rhythm is in trouble.
- Muscle weakness (especially in the legs and arms): Too much potassium outside muscle cells changes the voltage balance, making it harder for muscles to squeeze and relax properly — like a light switch stuck between on and off.
- Tingling or numbness (paresthesias), especially around the mouth or in the hands and feet: High potassium disturbs the electrical signals nerves use to talk to each other, so you can feel pins and needles or strange buzzing sensations even though nothing is touching your skin.
- Heart palpitations (feeling your heart skip, race, or pound): The heart's rhythm depends on potassium flowing in and out of heart muscle cells in a smooth pattern. When potassium in the blood is too high, the heart's electrical system misfires and beats irregularly.
- Nausea or vomiting: High potassium can irritate the stomach and slow gut movement, triggering waves of nausea or the urge to throw up.
- Slow heart rate (bradycardia): Excess potassium slows down the sinoatrial node (the heart's natural pacemaker in the right atrium) and blocks electrical signals from traveling smoothly through the atrioventricular node (the relay station between the top and bottom chambers), so the heart beats fewer times per minute — like a car engine sputtering because the spark plugs misfire.

Red flags — act now

- EKG changes (peaked T waves, wide QRS, flat P waves, or any new rhythm problem) — even if the patient feels fine
- Muscle weakness so severe the patient cannot stand or move arms and legs normally
- Heart rate below 50 or above 120, or any irregular, skipped beats
- Potassium level 6.5 mEq/L or higher (immediate treatment needed)
- Sudden chest pain, shortness of breath, or feeling faint (may signal a dangerous heart rhythm about to start)

Treat it — in this order

1. Calcium gluconate 1–2 g IV (or calcium chloride 0.5–1 g IV) over 2–3 minutes → Calcium raises the threshold voltage that heart cells need to reach before they fire. Normally, the resting voltage is around -90 millivolts and the threshold is around -70 millivolts. High potassium brings the resting voltage up closer to threshold — dangerous. Calcium pushes the threshold higher, widening the safety gap again and stabilizing the heart membrane within 1–3 minutes. This protects the heart from arrhythmias (like ventricular fibrillation or asystole) while you work on lowering potassium.
2. Regular insulin 10 units IV with dextrose 25 g (one ampule of D50) IV push → Insulin activates sodium-potassium pumps on muscle and fat cell membranes. These pumps pull potassium from blood into cells in exchange for sodium. This lowers blood potassium by 0.5–1.2 mEq/L within 15–30 minutes and lasts 4–6 hours. Dextrose (sugar) prevents low blood sugar (hypoglycemia) (low blood sugar, below 70 mg/dL) from the insulin — without it, the patient could pass out or seize.
3. Albuterol 10–20 mg nebulized (or 0.5 mg IV if available) → Albuterol activates beta-2 receptors on muscle cell membranes, which turn on sodium-potassium pumps and pull potassium into cells. This lowers blood potassium by 0.5–1.0 mEq/L within 30 minutes. The effect adds to insulin — using both together is more powerful than either alone.
4. Sodium bicarbonate 50–100 mEq IV over 5–10 minutes (only if metabolic acidosis (acid building up in the blood) is present, pH below 7.2) → Bicarbonate neutralizes acid in the blood. When blood pH rises, hydrogen ions (acid) move out of cells and potassium moves back in to keep the electrical charge balanced across the membrane. This lowers blood potassium by 0.5–1.0 mEq/L over 30–60 minutes. Works best when too much acid in the blood (acidosis) is the main driver of high potassium (hyperkalemia) — for example, in diabetic ketoacidosis (the acid crisis of missing insulin) or kidney failure with severe too much acid in the blood.

5. Loop diuretic (furosemide 40–80 mg IV, or bumetanide 1–2 mg IV) if the kidneys are still making urine → Loop diuretics block the sodium-potassium-chloride transporter in the thick ascending limb of the loop of Henle (a part of the kidney tubule). This stops sodium from being reabsorbed, which increases urine flow and forces the distal tubule (the next segment) to excrete more potassium into urine. This removes potassium from the body and lowers blood potassium over 2–4 hours.

Your top priorities at the bedside

1. Place the patient on continuous cardiac monitoring and obtain a 12-lead EKG immediately.
— why: High potassium (Hyperkalemia) stops the heart before it causes other symptoms; the EKG shows peaked T waves, widened QRS complexes (the big spike on the rhythm strip), or sine waves (a pattern right before the heart stops) so we know how urgent treatment is.
2. Establish large-bore IV access and prepare calcium gluconate 1 to 2 grams for immediate IV push if EKG changes are present. — why: Calcium stabilizes the heart muscle cell membrane (outer layer of the cell) within minutes and prevents fatal arrhythmias like ventricular fibrillation (chaotic deadly heart rhythm); it does not lower potassium but buys time for other treatments to work.
3. Administer insulin 10 units IV with dextrose 25 grams IV (one ampule of D50) as ordered.
— why: Insulin pushes potassium from the blood back into cells by activating sodium-potassium pumps (tiny gates in cell membranes); dextrose prevents low blood sugar (hypoglycemia) from the insulin; potassium drops within 15 to 30 minutes.