

Severe Hyperkalemia with ECG Changes

Severe high potassium (hyperkalemia) with ECG changes: protect the heart with calcium first, shift potassium into cells second, then remove it from the body third.

THE ONE-LINE TRUTH

Potassium above 6.5 with heart-rhythm changes on the electrocardiogram (ECG) — this is a true emergency that can stop the heart in minutes.

The chain — how it unfolds

1. Kidneys fail to excrete potassium, or medicines block excretion, or cells dump potassium into the blood faster than kidneys can clear it. — why: Whether it's acute kidney injury, chronic kidney disease, ACE inhibitor, ARB, MRA, potassium-sparing diuretic, rhabdomyolysis, tumor lysis, or red blood cell destruction (hemolysis), the end result is the same: blood potassium climbs above 6.5 mEq/L. That high level is the match that lights the rest of the cascade.
2. Blood potassium floods the space outside every heart cell and muscle cell, flattening the voltage gradient across each cell membrane. — why: Normally, potassium is 30 times more concentrated inside the cell than outside. When outside potassium doubles or triples, the gradient shrinks, the cell's resting voltage drifts closer to the firing threshold, and the reset (repolarization) phase takes much longer because the cell has to pump against a smaller gradient.
3. The ECG shows the heart's electrical chaos in real time: first peaked T waves, then PR prolongation, then QRS widening, then sine wave, then ventricular fibrillation or asystole. — why: Peaked T waves (tall, narrow, symmetric T waves in the chest leads) appear first because repolarization is faster when the gradient is only mildly flattened. As potassium climbs higher, conduction through the atria and AV node slows (PR interval stretches), then conduction through the ventricles slows (QRS widens beyond 120 milliseconds), then the P wave (atrial contraction) disappears and the QRS blurs into the T wave, making a smooth sine wave. Finally, the ventricles quiver chaotically (ventricular fibrillation) or stop entirely (asystole), and the heart arrests.

4. Skeletal muscles weaken and then become paralyzed, starting in the legs and moving up toward the chest. — why: The same voltage flattening hits voluntary muscle fibers. Nerve signals can trigger a muscle fiber to fire once, but the fiber can't reset, so it becomes refractory and limp. Respiratory muscles (the diaphragm and intercostals, the muscles between the ribs) can fail last, causing respiratory arrest (breathing stops).
5. The heart arrests (ventricular fibrillation or asystole), and blood flow stops. — why: Once the heart's conduction system is fully poisoned, there is no organized electrical wave to trigger contraction. The ventricles either quiver uselessly or sit still. Without immediate CPR and treatment, brain death follows in 4–6 minutes.
6. Treatment must protect the heart membrane first (IV calcium), shift potassium into cells second (insulin with glucose, beta-2 agonists like albuterol, sodium bicarbonate if acidotic), and remove potassium from the body third (diuretics if kidneys work, dialysis if they don't, or potassium binders like patiromer or sodium zirconium cyclosilicate). — why: Calcium (given as calcium gluconate or calcium chloride through an IV) does not lower potassium, but it raises the threshold voltage that triggers a heart cell to fire, buying 30–60 minutes of protection while you shift and remove potassium. Insulin (with glucose to prevent low blood sugar) and beta-2 agonists activate the sodium-potassium pumps in muscle and liver cells, pulling potassium out of the blood and into cells within 15–30 minutes. Dialysis (running the blood through a filter machine) physically removes potassium in 2–4 hours and is the only option when kidneys are completely shut down. None of this happens on its own — the body cannot reverse severe high potassium (hyperkalemia) without help, so every second of delay brings the patient closer to arrest.
7. Trace it backwards: the wide QRS and sine wave point to high potassium poisoning the heart, the high potassium points to failed kidney excretion or cell rupture, and that points to the upstream break — AKI, CKD, RAAS blocker, rhabdo, tumor lysis, or red blood cell destruction (hemolysis). Find and fix the upstream cause while you protect the heart and clear the potassium, or the cycle will repeat. — why: If you only treat the potassium once and don't stop the source (for example, continuing an ACE inhibitor in a patient with stage 4 CKD, or not giving fluids and dialysis to crush-injury rhabdo), potassium will climb right back up within hours and the patient will arrest again. The full fix requires protecting the heart NOW, shifting and removing potassium FAST, and addressing the root cause ALWAYS.

Spot it — what you see, and why

- Peaked T waves on ECG (tall, narrow, triangle-shaped T waves): When potassium rises above normal in the blood, it changes how fast electricity moves across heart-muscle cells. The cells reset (repolarize) faster than usual, which makes the T wave on the ECG shoot up into a tall, narrow peak—like a tent pole instead of a gentle hill.

- Widened QRS complex on ECG (the tall spike stretches out wider than normal): Very high potassium slows the electrical signal that tells the ventricles (the heart's main pumping chambers) to squeeze. That delay stretches the QRS spike wider on the ECG—a sign the heart's wiring is dangerously sluggish.
- Flattened or absent P waves on ECG: Severe high potassium (hyperkalemia) can paralyze the atria (the heart's top chambers), so they stop making a clear electrical wave. The P wave—the little bump before each heartbeat—shrinks or disappears entirely because the atria are barely working.
- Muscle weakness (feels heavy or hard to move arms and legs): High potassium changes the electrical balance across muscle-cell membranes. At first muscles get twitchy, but when potassium climbs even higher they become weak or even paralyzed because the cells can't reset properly to fire again.
- Nausea or vomiting: The stomach and gut also rely on smooth electrical signals to squeeze and move food along. High potassium scrambles those signals, slowing the gut and triggering nausea or vomiting.
- Tingling or numbness (prickling feeling, usually in fingers, toes, or around the mouth): Nerves use a careful balance of potassium and sodium to send signals. When potassium is too high, nerve cells misfire or send fuzzy signals, which the brain reads as tingling, numbness, or pins-and-needles.

Red flags — act now

- Peaked T waves, widened QRS, or absent P waves on ECG—the heart's electrical system is breaking down and cardiac arrest can happen within minutes.
- New muscle paralysis or inability to move legs—very high potassium can shut down skeletal muscles completely, and the same process threatens the diaphragm (the breathing muscle).
- Sudden slow heart rate (bradycardia) or irregular rhythm—the heart may stop or fall into ventricular fibrillation (a deadly quivering that cannot pump blood) at any moment.
- Patient on dialysis missed a session, or has acute kidney injury with rising creatinine—the main escape route for potassium is blocked, so levels can skyrocket quickly.
- Watch for rebound high potassium (hyperkalemia) after initial treatment—insulin and albuterol only push potassium temporarily into cells; it drifts back out over hours, so potassium must be removed from the body with dialysis or binding resins to prevent a second spike.

Treat it — in this order

1. IV calcium gluconate 1-2 grams (10-20 mL of 10% solution) over 2-5 minutes, repeat every 5-10 minutes until ECG improves → Calcium raises the electrical threshold of heart cells—makes them harder to fire randomly—so the heart won't go into a deadly rhythm even though potassium is still high. Buys you 30-60 minutes of safety. The ECG should improve (QRS narrows, sine wave resolves) within minutes.
2. IV regular insulin 10 units plus 50 grams dextrose (one amp of D50), or insulin alone if blood glucose >250 mg/dL → Insulin turns on pumps (sodium-potassium ATPase) in muscle and liver cells that suck potassium from blood into cells. Lowers serum potassium by 0.5-1.2 mEq/L in 15-30 minutes. Dextrose prevents dangerous low blood sugar (hypoglycemia) from the insulin.
3. Nebulized albuterol 10-20 mg (four 5 mg vials back-to-back) over 10 minutes → Albuterol is a beta-2 agonist—it tells muscle cells to pull potassium inside. Lowers serum potassium by 0.5-1.0 mEq/L in 30-90 minutes. Works alongside insulin; they help each other.
4. IV sodium bicarbonate 50-150 mEq (1-3 amps) over 15-30 minutes, ONLY if blood pH <7.2 (severe acidemia) → Raising blood pH (making it less acidic) pulls hydrogen ions out of cells; potassium moves in to replace them, lowering blood potassium. Works in 30-60 minutes.
5. IV loop diuretic (furosemide 40-80 mg) if kidneys make urine → Furosemide forces the kidneys to dump potassium into urine. Removes potassium from the body (not just shifts it). Works in 1-2 hours if kidney function is decent.

Your top priorities at the bedside

1. Place patient on continuous cardiac monitor and get a 12-lead electrocardiogram immediately, then repeat ECG every 30 to 60 minutes until potassium is safe. — why: High potassium blocks the heart's electrical signals. Peaked tall T-waves come first, then QRS widens, then P-wave disappears, then the heart rhythm becomes a sine wave (looks like a smooth wave) right before the heart stops. The ECG tells us how close the patient is to cardiac arrest (heart stopping) and whether our treatments are working.
2. Give intravenous calcium gluconate 1 gram over 2 to 5 minutes through a large vein, and prepare a second dose to give in 5 minutes if the ECG does not improve. — why: Calcium does not lower potassium, but it protects the heart muscle cell membrane from the electrical effects of high potassium. This buys time (about 30 to 60 minutes) while other treatments move potassium out of the blood. Calcium works in 1 to 3 minutes.

3. Verify the potassium result is real by checking if the blood sample was hemolyzed (red blood cells broke open in the tube) and redraw if needed, but do NOT wait for the new result to treat if the ECG shows changes. — why: Red blood cell destruction (Hemolysis) (破裂 of red cells during blood draw) releases potassium from inside cells into the tube, giving a falsely high result (pseudohyperkalemia). A hemolyzed sample looks pink or red. Always treat the ECG, not just the number, because the ECG shows what potassium is doing to the heart right now.