

Severe Hyperkalemia with ECG Changes

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

Potassium (K^+) is a charged mineral particle that lives mostly inside cells, with only a tiny amount in the blood. That careful inside-versus-outside balance creates the electrical charge that lets every heart cell and muscle cell fire, contract, and then reset for the next beat.

BODY'S JOB

Heart muscle cells and skeletal muscle cells work like rechargeable batteries: potassium inside the cell and sodium outside create a voltage difference across the cell membrane (the cell's outer wrapper). When a signal arrives, sodium rushes in and potassium leaks out, flipping the charge and making the cell fire. Then pumps in the membrane (called sodium-potassium ATPase pumps) work like conveyor belts to push potassium back inside and sodium back outside, resetting the battery so the cell can fire again. In the heart, this cycle happens in perfect rhythm about 60–100 times every minute, keeping the squeeze-and-fill pattern smooth and orderly. The kidneys are the body's main potassium-removal system: they filter blood, sense how much potassium is there, and dump any extra into urine through channels in the collecting duct (the final tube in each kidney unit).

Aldosterone (a hormone made by the adrenal glands, two small glands sitting on top of the kidneys) tells the kidney to hold onto sodium and let go of potassium, fine-tuning the balance every day.

1. Potassium enters the body from food (bananas, potatoes, beans, salt substitutes).
2. The small intestine absorbs it into the bloodstream.

3. Insulin (the blood-sugar hormone from the pancreas) and beta-2 receptors (landing spots on cells triggered by adrenaline) actively push potassium from blood into muscle cells and liver cells, keeping blood levels steady.
4. The kidneys filter all the blood every few minutes, and the collecting duct in each nephron (the kidney's tiny filter unit) decides how much potassium to dump into urine based on aldosterone's signal.
5. Normal blood potassium stays between 3.5 and 5.0 milliequivalents per liter (mEq/L), and normal inside-cell potassium is about 30 times higher — that steep gradient is what creates the cell's voltage.
6. The heart's electrical system (the sinoatrial node, a tiny patch of pacemaker cells in the right atrium) fires a signal that spreads in an orderly wave through the atria (the top two chambers), pauses at the AV node (the gatekeeper between top and bottom chambers), then spreads through the ventricles (the bottom two pumping chambers) along special fast highways called the bundle branches and Purkinje fibers, making the whole heart squeeze in sync.

What breaks

One upstream break — too much potassium sitting in the blood instead of inside cells or being dumped into urine — explains all the weird findings together. The break happens when the kidneys can't remove potassium, when medicines block potassium removal, or when a flood of potassium spills out of dying cells faster than the body can clear it.

CLINICAL TERM

Severe high potassium (hyperkalemia) (blood potassium above 6.5 mEq/L) with ECG changes.

How the body compensates

The body has almost no built-in way to compensate for severe high potassium (hyperkalemia). Vital signs (blood pressure, heart rate, oxygen level) can look completely normal until the heart suddenly arrests. That is the dangerous trap: the patient may feel weak but look stable, and then the rhythm degenerates to cardiac arrest in seconds. Your job is to see the ECG warning signs and treat before the crash, because once the heart stops, the window to save the patient shrinks fast.

1. The adrenal glands try to make more aldosterone to tell the kidneys to dump potassium, but if the kidneys are already broken or blocked by medicines, aldosterone's signal has nowhere to land — compensation fails.

2. Insulin and beta-2 receptors try to shift potassium from blood into cells, but this is a temporary buffer, not a removal system. The total body potassium is still too high, so the blood level will creep back up in hours unless you physically remove potassium.
3. The heart's conduction system has no backup plan. As membrane voltage flattens, cells become refractory (unable to fire again), and the rhythm falls apart step by step: peaked T → wide QRS → sine wave → ventricular fibrillation or asystole. There is no compensatory rhythm that rescues this — the heart simply stops.
4. Blood pressure and oxygen saturation can stay normal even as the ECG deteriorates, because the heart is still squeezing (for now) and the lungs are fine. This makes the patient look stable when they are actually seconds from arrest.
5. Muscle weakness is a clue, but many patients don't notice it or don't report it until paralysis is severe. You cannot wait for symptoms — the ECG is the early-warning system.
6. Watch for the crash point before it happens: the transition from peaked T waves to widened QRS is the red line. Once QRS widens, cardiac arrest is imminent. Every minute without treatment doubles the risk.

Domino effect

1. Kidneys fail to excrete potassium, or medicines block excretion, or cells dump potassium into the blood faster than kidneys can clear it. — 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. — 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. — 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. — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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.
- Slow or irregular heartbeat (bradycardia or arrhythmia): Excess potassium disrupts the heart's natural pacemaker (the SA node) and the wiring (the AV node and bundle branches) that keeps each beat regular. The heart may beat too slowly, skip beats, or fall into chaotic rhythms that can't pump blood effectively.
- Lab confirmation: serum potassium ≥ 6.5 mEq/L (often ≥ 7.0 mEq/L) with ECG changes: The kidneys usually keep blood potassium tightly controlled between 3.5 and 5.0 mEq/L. When potassium climbs to 6.5 or higher—because the kidneys are failing, certain medicines block potassium excretion, or cells dump potassium into the blood—and the ECG shows peaked T waves or QRS widening, that combination defines severe high potassium (hyperkalemia) with immediate cardiac risk.
- Pseudohyperkalemia ruled out (sample was not hemolyzed or clotted): Red blood cells are packed with potassium. If a blood sample sits too long, gets shaken too hard, or clots in the tube, red cells burst and spill their potassium into the serum, making the lab result look falsely high. True severe high potassium (hyperkalemia) requires a clean, non-hemolyzed sample and clinical signs (especially ECG changes) that match.

Focused exam

- **Peaked T waves on ECG:** The earliest heart warning sign. High potassium makes heart cells twitchy—they fire too easily—so the T wave (the moment the bottom chambers reset) shoots up tall and narrow like a tent or mountain peak. This is your first red flag.
- **Prolonged PR interval (>200 ms):** The electrical signal from the top chambers to the bottom chambers is traveling too slowly. High potassium gums up the wiring. This happens right after peaked T waves and means danger is climbing.
- **Wide QRS complex (>120 ms):** The bottom chambers (ventricles) are taking too long to squeeze. Electricity is crawling through the heart muscle instead of zipping across. This is a late sign—the heart is struggling hard now.
- **Sine-wave pattern on ECG:** The rhythm looks like a smooth rolling wave instead of sharp peaks and valleys. The heart's electrical system is nearly dead. Cardiac arrest is minutes away—this is the most dangerous ECG pattern in high potassium (hyperkalemia).
- **Muscle weakness or flaccid paralysis:** High potassium makes all muscle cells—not just the heart—less able to contract. Arms and legs feel heavy or won't move at all. Same chemical problem, different tissue.
- **A slow heart rate (Bradycardia) or irregular pulse:** High potassium slows or scrambles the heart's natural pacemaker (the sinoatrial node in the top right chamber). The pulse drops below 60 or skips beats. Part of the same electrical breakdown.

Diagnostics · every test answers a question

- **12-lead electrocardiogram (ECG)** → Does the heart rhythm show electrical toxicity from high potassium (hyperkalemia)?
- **Serum potassium (K^+)** → How high is the potassium in the blood?
- **Basic metabolic panel (BMP) or comprehensive metabolic panel (CMP)** → Are the kidneys working? Is there too much acid in the blood (acidosis) or other electrolyte problems?
- **Arterial blood gas (ABG)** → Is the blood acidic, and does that explain why potassium is high?
- **Creatine kinase (CK) and urine myoglobin** → Is muscle breakdown (rhabdomyolysis) dumping potassium into the blood?
- **Urine potassium and transtubular potassium gradient (TTKG)** if cause unclear → Are the kidneys trying to get rid of potassium, or are they holding onto it?
- **Repeat serum potassium 1-2 hours after treatment** → Did the emergency treatment lower potassium, or is it climbing again?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.
- Potassium binders: patiromer 8.4 g or sodium zirconium cyclosilicate (SZC) 10 g by mouth, OR sodium polystyrene sulfonate (Kayexalate) 30 g by mouth or rectum → These medicines sit in the gut and trap potassium so it leaves in stool instead of being absorbed into blood. Actually removes potassium from the body. Patiromer and SZC work in 1-7 hours; Kayexalate takes 2-24 hours.
- Emergency hemodialysis → Dialysis pulls potassium directly out of the blood across a filter membrane. Lowers potassium by 1-2 mEq/L per hour. The only reliable fix if kidneys are dead, if potassium won't budge with medicine, or if the patient is in kidney failure.

Medications

Calcium gluconate (or calcium chloride if a central IV line is in place) · Membrane stabilizer

Calcium does not lower the potassium number at all. Instead, it wraps a protective shield around the heart muscle cells so they can still squeeze and relax normally even while potassium is dangerously high. Think of it like putting rubber bumpers on a sensitive switch so

it doesn't fire by accident. This buys time—usually 30 to 60 minutes—for the next medicines to actually move potassium out of the blood.

1. High potassium makes the resting voltage inside heart cells less negative (closer to the threshold for firing), which can freeze the heart or trigger dangerous rhythms.
2. Calcium raises the threshold voltage needed to trigger a heartbeat, moving it farther away from the resting level.
3. This restores the normal gap between resting and firing, so the heart's electrical system works reliably again.
4. The effect starts in 1 to 3 minutes and lasts about 30 to 60 minutes, so it must be followed by treatments that actually remove potassium.

SAFETY WATCH

Give it slowly—1 gram over 2 to 5 minutes—to avoid a sudden spike in blood calcium that can cause the heart to stop. · If the patient is taking digoxin (a heart pill), calcium can make digoxin toxicity worse; use extra caution and give it more slowly or use a smaller dose. · Calcium chloride is three times stronger than calcium gluconate but can burn and destroy tissue if it leaks outside the vein, so it should only go through a central line (a deep IV in the chest or neck). · Watch the heart monitor continuously; if the dangerous rhythm (peaked T waves, wide QRS) does not improve within 5 to 10 minutes, give another dose. · Calcium does not replace the other steps—you must still shift potassium into cells and remove it from the body.

Regular insulin (10 units IV) plus dextrose 50% (one ampule, 25 grams, unless blood sugar is already above 250 mg/dL) · Cellular potassium-shift agent

Insulin opens tiny doors (called sodium-potassium pumps) on the surface of muscle and liver cells, pushing potassium from the blood back inside the cells where it belongs. Think of it like turning on a vacuum that sucks potassium out of the bloodstream and tucks it away safely in storage. The dextrose (sugar) keeps blood sugar from dropping dangerously low while insulin does its job. This lowers blood potassium by about 0.5 to 1.2 mEq/L within 15 to 30 minutes, and the effect lasts 4 to 6 hours.

1. Insulin binds to insulin receptors on muscle and liver cell surfaces.
2. This activates sodium-potassium pumps (ATPase enzymes) embedded in the cell membrane.
3. Each pump uses energy (ATP) to push three sodium ions out of the cell and pull two potassium ions in.
4. Potassium moves from the blood into the cells, lowering the dangerous level in the blood.
5. The effect peaks in 15 to 30 minutes and lasts 4 to 6 hours, so you must follow up with treatments that actually remove potassium from the body (diuretics, binders, or dialysis).

SAFETY WATCH

Always check blood sugar before and every hour for 4 to 6 hours after giving insulin, because low blood sugar (hypoglycemia) is the biggest risk and can cause confusion, seizures, or loss of consciousness. · If blood sugar is already above 250 mg/dL, skip the dextrose but still give the insulin. · If blood sugar is low to start with, give extra dextrose (sometimes two ampules) to prevent it from dropping further. · Potassium will drift back up after a few hours, so plan the next steps (binders, diuretics, or dialysis) before insulin wears off.

Albuterol (10 to 20 mg by nebulizer, inhaled over 10 minutes) · Beta-2 adrenergic agonist (cellular potassium-shift agent)

Albuterol flips on the same cellular pumps that insulin activates—the sodium-potassium pumps—but through a different switch (beta-2 receptors instead of insulin receptors). It pushes potassium from the blood into muscle cells. Think of it like a second vacuum running alongside the insulin vacuum, pulling even more potassium out of the bloodstream. It lowers potassium by about 0.5 to 1.0 mEq/L within 30 minutes, and the effect lasts 2 to 4 hours. It works faster when given in high doses through a nebulizer (the mist machine used for asthma).

1. Albuterol binds to beta-2 receptors on muscle cells.
2. This triggers a cascade inside the cell that activates sodium-potassium pumps.
3. The pumps push potassium from the blood into the cells, lowering the blood level.
4. The effect starts in about 30 minutes and lasts 2 to 4 hours.

SAFETY WATCH

High doses can make the heart race (tachycardia) or cause chest discomfort, especially in people with existing heart disease. · It can trigger shaking (tremor) and anxiety because it revs up the nervous system. · About 20 to 40% of patients do not respond well to albuterol (the potassium does not drop), so always combine it with insulin for better results. · Use caution in patients with fast or irregular heart rhythms to start with.

Sodium bicarbonate (50 to 100 mEq IV over 5 minutes, or as an infusion) · Alkalinizing agent (cellular potassium-shift agent in too much acid in the blood (acidosis))

When the blood is too acidic (low pH, high acid load), cells trade hydrogen ions (acid) for potassium to keep the blood pH from dropping dangerously. This pushes extra potassium out of cells and into the blood. Sodium bicarbonate is a base (the opposite of acid) that neutralizes the extra acid in the blood. Once the blood pH rises, cells stop dumping potassium and may even pull some back in. Think of it like turning off a faucet that was flooding potassium into the

bloodstream. This only works when too much acid in the blood (acidosis) (too much acid) is part of the problem—if the blood pH is normal or high, bicarbonate will not help and may be harmful.

1. In too much acid in the blood (acidosis), cells try to buffer the blood by taking up hydrogen ions (acid) and releasing potassium in exchange.
2. This raises blood potassium even if total body potassium is normal.
3. Sodium bicarbonate neutralizes the extra acid (raises pH), so cells no longer need to trade potassium for hydrogen.
4. Potassium shifts back into cells, lowering the blood level by about 0.5 to 1.0 mEq/L over 1 to 2 hours.

SAFETY WATCH

Only use bicarbonate if the blood is truly acidic (pH below about 7.2 or bicarbonate level very low); giving it when pH is normal or high can cause dangerous shifts in calcium and oxygen delivery. · It can cause fluid overload (too much volume in the blood) because it contains a lot of sodium. · It can paradoxically worsen intracellular too much acid in the blood (acidosis) in some cases (by generating CO₂ that crosses into cells), so it is not a first-line agent unless too much acid in the blood is severe. · Measure blood pH or bicarbonate level before giving it; do not guess.

Furosemide (40 to 80 mg IV, or higher doses if needed) · Loop diuretic (potassium removal via urine)

Furosemide is a water pill that forces the kidneys to flush extra water, sodium, and potassium out of the body through urine. It works on a part of the kidney's plumbing (the loop of Henle) that normally reabsorbs these chemicals back into the blood. By blocking that reabsorption, furosemide makes the kidneys dump potassium into the urine and out of the body. Think of it like opening a drain valve to let the flood out. This only works if the kidneys are still making urine—if they have shut down (acute kidney injury or end-stage kidney disease), furosemide will not help.

1. Furosemide blocks the NKCC2 transporter in the loop of Henle.
2. Sodium, potassium, and chloride stay in the urine instead of being pulled back into the blood.
3. The kidneys flush them out, lowering blood potassium by about 0.5 to 1.0 mEq/L over several hours.
4. This only works if the kidneys are functioning and making urine.

SAFETY WATCH

Can cause dangerously low potassium (hypokalemia) once the crisis is over, so monitor levels and replace potassium later if needed. · Can cause dehydration and low blood pressure if too much fluid is lost. · Can worsen kidney function if the patient is already volume-depleted (dehydrated). · Can cause low magnesium and calcium over time, which can trigger abnormal heart rhythms. · Use carefully in patients with severe kidney disease; if they are not making urine, switch to dialysis instead.

Patiromer or sodium zirconium cyclosilicate (SZC) · Potassium binders (gastrointestinal potassium removal)

These are powders that you swallow (or give through a rectal tube in an emergency). They act like tiny sponges in the gut, soaking up potassium from food, digestive juices, and blood that seeps into the intestines, then carrying it out in the stool. Think of it like a magnet that grabs potassium and won't let go until it leaves the body. They lower potassium by about 0.5 to 1.0 mEq/L over 4 to 7 hours (SZC is faster, patiromer slower). Because they work slowly, they are NOT the first choice in a true emergency with dangerous heart rhythms—they are used alongside or after the faster treatments.

1. The binder powder mixes with fluid and food in the gut.
2. Potassium ions stick to the binder particles (patiromer trades calcium for potassium; SZC trades sodium and hydrogen for potassium).
3. The binder-potassium complex moves through the intestines and leaves the body in stool.
4. Blood potassium drops as the gut pulls potassium out of the bloodstream to replace what the binder removed.
5. SZC works faster (4 to 6 hours) than patiromer (7 to 24 hours).

SAFETY WATCH

Can cause constipation or, rarely, gut blockage, so make sure the patient can have bowel movements. · Patiromer can lower magnesium over time; monitor and replace if needed. · SZC can cause mild swelling (edema) because it releases sodium as it binds potassium. · Do not give with other oral medications at the same time—binders can trap them and make them useless; separate doses by at least 3 hours. · Rectal use (retention enema) is off-label and less effective, but may be tried if the patient cannot swallow and dialysis is not immediately available.

Hemodialysis · Renal replacement therapy (direct potassium removal)

Hemodialysis is an artificial kidney machine. It pulls blood out of the body, runs it through a filter (dialyzer) with tiny pores, and washes potassium, acid, and other waste products out of

the blood into a cleansing fluid (dialysate), then returns the clean blood. Think of it like running dirty water through a coffee filter that traps the grit. Dialysis removes potassium faster and more completely than any medication—it can drop potassium by 1.0 to 1.5 mEq/L per hour. It is the definitive treatment when kidneys have failed, when medications are not working, or when potassium is so high that waiting is too dangerous.

1. Blood is pumped from the patient through tubing into the dialyzer.
2. Inside the dialyzer, blood flows on one side of a semi-permeable membrane and dialysate (cleansing fluid with low or zero potassium) flows on the other side.
3. Potassium moves from the blood (high concentration) into the dialysate (low concentration) by diffusion.
4. The potassium-loaded dialysate is discarded, and the cleaned blood is returned to the patient.
5. This continues for 3 to 4 hours, removing large amounts of potassium and also correcting acid-base balance and fluid overload.

SAFETY WATCH

Requires a dialysis catheter, fistula, or graft; placing a catheter in an emergency carries risks of bleeding, infection, and lung collapse (pneumothorax). · Can cause sudden drops in blood pressure, muscle cramps, and rarely heart rhythm problems during the session. · Must be done in a hospital with trained staff and equipment. · Potassium can rebound (rise again) after dialysis as it leaks back out of cells, so check levels a few hours after treatment and plan long-term management.

Red flags

- 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.

Nursing priorities

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. — 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. — 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. — 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.
4. Administer intravenous regular insulin 10 units plus 50 grams of dextrose (one ampule of D50) over 15 to 30 minutes, but skip the dextrose if fingerstick glucose is above 250 mg/dL. — Insulin pushes potassium from the blood into body cells by activating sodium-potassium pumps in cell membranes. This lowers blood potassium by 0.5 to 1.0 mEq/L in 15 to 30 minutes and lasts 4 to 6 hours. Dextrose prevents low blood sugar (hypoglycemia) from the insulin. Check glucose every 1 hour for 6 hours because low blood sugar can happen late (rebound low blood sugar).
5. Give nebulized albuterol 10 to 20 milligrams (use 4 to 8 times the asthma dose, which is 2.5 mg) by continuous nebulizer over 30 minutes. — Albuterol (a beta-2 agonist, the same medicine used for asthma) activates cell membrane pumps that pull potassium into cells. It lowers potassium by 0.5 to 1.0 mEq/L in 30 minutes. The high dose may cause a fast heart rate (tachycardia) (fast heart rate) and tremor (shaking), but these are safer than high potassium.
6. Check arterial blood gas or venous pH, and if pH is below 7.2 (acidemia, meaning the blood is too acidic), give intravenous sodium bicarbonate 50 to 100 milliequivalents over 5 to 10 minutes. — Acidemia keeps potassium outside cells. When blood pH is low (acidic), hydrogen ions (H⁺) move into cells and push potassium out into the blood. Bicarbonate raises the pH, which lets potassium move back into cells. This only works if the patient has metabolic acidosis (acid building up in the blood) (low bicarbonate level).

7. Eliminate potassium from the body by giving intravenous furosemide (Lasix) 40 to 80 milligrams if the patient makes urine, OR give potassium binders like patiromer (Veltassa) or sodium zirconium cyclosilicate (Lokelma) by mouth or rectum if kidneys do not work. — Calcium, insulin, and albuterol only shift potassium into cells temporarily—potassium will come back out (rebound high potassium (hyperkalemia)) in 4 to 6 hours. We must remove potassium from the body. Furosemide (a loop diuretic) makes kidneys dump potassium into urine. Binders stick to potassium in the gut so it leaves in stool. Old sodium polystyrene sulfonate (Kayexalate) can cause bowel death (necrosis) so newer binders are safer.
8. Notify the physician immediately if potassium stays above 6.5 after one hour of treatment, if ECG keeps getting worse, if urine output is under 30 mL per hour for 2 hours, or if the patient has chest pain, severe weakness, or confusion—because emergency hemodialysis (dialysis machine) may be needed. — Dialysis removes potassium from blood in 1 to 3 hours and is the only reliable option when kidneys cannot remove potassium or when medicines fail. Indications for emergency dialysis include potassium above 6.5 despite treatment, worsening ECG changes, no urine output (anuria), severe too much acid in the blood (acidosis), or life-threatening arrhythmia.

Sources

- American Heart Association (AHA) ACLS guidelines
- National Kidney Foundation KDOQI guidelines
- UpToDate clinical reference (Nephrology)
- Journal of the American Society of Nephrology (JASN)

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