

Severe hypokalemia

Severe low potassium (hypokalemia): replace potassium fast through a central line, check and fix magnesium at the same time, hunt down the cause (gut loss, kidney loss, or shift into cells), and keep the patient on a heart monitor the whole time.

THE ONE-LINE TRUTH

Potassium below 2.5 mEq/L or any level with life-threatening signs like dangerous heartbeats, muscle paralysis, or breathing trouble.

The chain — how it unfolds

1. Potassium drops below 2.5 mEq/L or symptoms appear — why: Potassium is the charged particle that sets the resting electrical tone of every nerve and muscle cell. When blood potassium falls, the inside of the cell becomes too negative compared to the outside (hyperpolarized), making it harder for cells to reach the threshold needed to fire a signal.
2. Skeletal muscles weaken or go paralyzed (flaccid paralysis) — why: Muscle cells cannot generate the electrical spark needed to contract. Weakness starts in the legs and moves up. In severe cases, muscles go completely limp—you cannot lift your arms, stand, or even hold your head up. This is flaccid paralysis, and it can hit the diaphragm (the breathing muscle), causing respiratory failure.
3. Gut smooth muscle stops moving (ileus) — why: The smooth muscle that squeezes food through the intestines also depends on potassium to fire. When potassium is too low, the gut goes quiet—no rumbling, no movement. The belly swells, you feel bloated and nauseated, and bowel sounds disappear. This is called a stalled gut (ileus) (paralyzed gut).
4. Heart repolarization slows, ECG shows U waves and long QT — why: The heart's electrical cycle has a recharge phase (repolarization) that depends on potassium flowing out of heart cells. When potassium is low, repolarization takes longer. On the ECG, the T wave flattens, a new hump called a U wave appears after it, and the QT interval (the time from heartbeat start to recharge finish) stretches out. A QT over 500 milliseconds is dangerous.

5. Ventricular arrhythmias: PVCs, torsades de pointes, ventricular fibrillation — why: The long QT creates an electrical window where a stray spark can trigger a chaotic rhythm. Premature ventricular contractions (PVCs) start, then the rhythm can spiral into torsades de pointes—a twisting, rapid heartbeat that cannot pump blood. If untreated, torsades degenerates into ventricular fibrillation (total electrical chaos) and the heart stops pumping at all.
6. Low magnesium blocks potassium replacement and worsens arrhythmias — why: Magnesium is the gatekeeper that keeps potassium inside cells. If magnesium is low (common with diuretics, diarrhea, or alcohol use), giving potassium does not work—it pees right back out. Low magnesium (Hypomagnesemia) also directly prolongs the QT interval and makes torsades more likely. You must check and replace magnesium at the same time as potassium, or the whole cascade continues.
7. Respiratory muscles fail, leading to pauses in breathing (apnea) and death — why: The diaphragm and intercostal muscles (the muscles between your ribs) are skeletal muscles that need potassium to contract. When they weaken or stop, you cannot take a breath. Respiratory arrest is the final common pathway of untreated severe low potassium (hypokalemia).
8. Replace potassium (10–20 mEq/h through central line), fix magnesium (1–2 g IV), stop the leak, monitor continuously—and the cascade reverses — why: Severe low potassium (hypokalemia) is one broken thing with many downstream effects. Restore potassium, correct magnesium, and treat the cause (stop the diuretic, give fluids for vomiting, hold insulin if overshooting), and the domino chain stops—muscles regain strength, the gut wakes up, the ECG normalizes, arrhythmias disappear, and the patient survives. Speed matters: the faster you recognize and replace, the less time the heart and lungs spend in the danger zone.

Spot it — what you see, and why

- Muscle weakness (especially in the legs and arms): Potassium is needed to make muscles contract. When potassium drops too low, the electrical signals muscles use to squeeze don't fire properly, so muscles feel weak and heavy.
- Muscle cramps or twitching: Low potassium makes muscle cells jumpy and unstable — they fire randomly or lock up in painful cramps because the balance of electrical charge inside and outside the cell is off.
- Constipation or bloating (gut slows down): The smooth muscle in the intestines needs potassium to squeeze and push food along. When potassium is too low, the gut moves slowly, so food sits longer and causes bloating or constipation.

- Heart palpitations (feeling your heart skip, flutter, or race): The heart's rhythm depends on potassium flowing in and out of heart cells to create each beat. Too little potassium disrupts that rhythm, causing extra beats, pauses, or a fluttery feeling.
- Abnormal EKG (flattened T waves, U waves, prolonged QT interval): Potassium controls the repolarization phase — when heart cells reset after each beat. Low potassium makes that reset slower and weaker, which shows up as flat or upside-down T waves, extra U waves, and a longer QT stretch on the heart tracing.
- Feeling very tired or weak all over: Every cell uses potassium to control energy production and nerve signals. When levels drop, cells can't work efficiently, so the whole body feels drained and sluggish.

Red flags — act now

- Chest pain, severe palpitations, or fainting (risk of dangerous heart rhythm — ventricular a fast heart rate (tachycardia) or torsades de pointes)
- Severe muscle weakness or paralysis (risk of respiratory failure if breathing muscles stop working)
- Potassium below 2.5 mEq/L (very high risk of life-threatening arrhythmia)
- New confusion or altered mental status (may signal severe metabolic derangement)
- Shortness of breath or difficulty breathing (may mean diaphragm weakness or heart failure)

Treat it — in this order

1. IV potassium chloride (KCl) 10–40 mEq/hour via central line with continuous cardiac monitoring in the ICU → Potassium moves back into the blood and restores the electrical charge difference across heart and nerve cell membranes. This stops the heart from going into dangerous rhythms like torsades de pointes and lets muscles and nerves start working again. Guideline max rate is 10 mEq/h via peripheral IV, but 20–40 mEq/h via central line is used in life-threatening cases.
2. IV magnesium sulfate 1–2 grams over 15–30 minutes, then magnesium replacement to keep level above 2 mg/dL → Magnesium allows the sodium-potassium pump (the cell's battery charger) to work and helps the kidneys hold onto potassium. Without magnesium, potassium replacement stalls and the heart rhythm stays dangerous. Fixing magnesium makes the potassium you give actually stick inside cells.
3. Continuous telemetry (heart monitor) until serum potassium rises above 3.0 mEq/L and stays stable → Catches dangerous rhythm changes (long QT, U waves, premature ventricular beats, torsades de pointes) the moment they start, so the team knows if the potassium infusion is working fast enough or if the patient needs emergency defibrillation.

4. Identify and stop the cause: hold diuretics, treat vomiting or diarrhea, work up hyperaldosteronism, or correct metabolic alkalosis (the blood turning too alkaline) → Stops the leak. If you only pour potassium in without plugging the hole (the broken step upstream), levels fall again within hours. Stopping a loop diuretic, giving antiemetics for vomiting, or treating primary aldosteronism fixes the root problem.
5. Oral potassium chloride (KCl) 40–100 mEq per day in divided doses once the gut is working and the patient can swallow (no nausea or a stalled gut (ileus)) → Continues potassium replacement by mouth when the intestines are moving again, avoiding repeated IV sticks and making it possible to send the patient home safely.

Your top priorities at the bedside

1. Place patient on continuous telemetry (heart monitor) and watch for T-wave flattening, U-waves, ST depression, or arrhythmias on the monitor screen — why: Potassium controls the electrical recharge of heart cells; when potassium drops below 2.5 mEq/L, the heart cannot repolarize (reset) properly, causing ventricular fibrillation or torsades de pointes (twisting-ribbon deadly arrhythmia) that can stop the heart in seconds
2. Start IV potassium chloride through a central line at 10 to 20 mEq per hour, never faster than 20 mEq per hour, and never give IV push — why: Peripheral veins (small arm veins) burn and can scar from concentrated potassium; fast IV potassium causes the heart to stop in diastole (relaxed phase); central lines dilute potassium in large blood volume quickly so it does not hit the heart all at once
3. Check serum magnesium level and give IV magnesium sulfate 1 to 2 grams over 15 minutes if magnesium is low, then start continuous infusion — why: Magnesium helps cells hold onto potassium; the kidney cannot reabsorb potassium when magnesium is low, so replacing potassium alone will not work; 40 percent of patients with severe low potassium (hypokalemia) also have low magnesium (hypomagnesemia) (low magnesium)