

Hypokalemia

Low potassium (Hypokalemia) means your potassium level is too low, either because you lost it (kidneys or gut) or it shifted into your cells. The heart is the organ most in danger.

THE ONE-LINE TRUTH

Not enough potassium in the blood — the heart and muscles are at risk.

The chain — how it unfolds

1. Blood potassium drops below 3.5 mEq/L. — why: Losses through kidneys or gut, or a sudden shift into cells, lower the potassium concentration in the blood (the extracellular fluid).
2. The resting membrane potential becomes more negative (hyperpolarized). — why: With less potassium outside the cell, the voltage difference across the membrane gets bigger — the inside becomes even more negative compared to the outside, like over-charging the battery.
3. Cells become harder to fire (higher threshold) and slower to reset. — why: A bigger voltage gap means it takes a stronger signal to flip the cell from rest to active (depolarization is delayed), and after firing, the cell takes longer to pump potassium back out and reset (repolarization is prolonged).
4. Skeletal muscles weaken; smooth muscle in the gut slows or stops. — why: Muscle cells that are slow to fire and reset cannot generate strong, sustained contractions — you feel weak, your legs feel heavy, and your gut stops squeezing (causing constipation, bloating, or a stalled gut (ileus)). Severe cases can trigger rhabdomyolysis (muscle breaks down) because the cells become so energy-starved and leaky.
5. The heart's repolarization (reset phase) drags out. — why: Cardiac myocytes need potassium flowing out to bring the voltage back down after each beat; low potassium slows this, so repolarization takes longer — this shows up as a long QT interval on the EKG.
6. T waves flatten or invert; U waves appear. — why: The T wave represents ventricular repolarization; when it is slow and weak, the T wave shrinks and flattens. U waves (a small extra bump after the T wave) appear because of after-depolarizations — the cells try to fire again too soon, creating a visible ripple in the electrical tracing.

7. The QT interval lengthens (prolonged QT). — why: The QT measures the total time from ventricular contraction to full reset; low potassium stretches both depolarization and repolarization, so the QT interval grows longer (QTc > 500 ms is high risk).
8. Torsades de pointes (polymorphic VT) or ventricular fibrillation can suddenly start. — why: The long QT and early after-depolarizations create the perfect setup for a re-entry loop — an electrical signal spins around the ventricles instead of moving forward, triggering a chaotic, twisting rhythm (torsades) that can degenerate into ventricular fibrillation and cardiac arrest. This is the life-threatening crash point.

Spot it — what you see, and why

- Muscle weakness, especially in the legs and arms: Potassium keeps muscle cells ready to fire. When potassium in the blood drops, muscle cells become too electrically quiet (hyperpolarized), so they struggle to contract and feel weak.
- Muscle cramps and twitches: Low potassium throws off the balance of electrical signals that tell muscles when to squeeze and relax, causing them to cramp or twitch on their own.
- Peeing a lot and feeling very thirsty: Low potassium can stop the kidneys from responding to ADH (the hormone that tells the kidneys to save water), so the kidneys let too much water leave as urine — this is called nephrogenic diabetes insipidus.
- Constipation and bloating: The muscles that push food through the intestines need potassium to squeeze properly. When potassium is low, the gut slows down (ileus), and stool moves sluggishly.
- Abnormal heart rhythm seen on ECG monitor (flattened T waves, U waves, ST depression): Potassium controls the electrical recharge (repolarization) of heart muscle after each beat. Low potassium makes the heart's electrical pattern look different and can set the stage for dangerous rhythms.
- Feeling your heart skip, race, or flutter (palpitations): The heart's rhythm depends on potassium flowing in and out of heart cells. When potassium is too low, the heart can misfire and create extra beats or chaotic rhythms like atrial fibrillation (an irregular, quivering heartbeat) or ventricular a fast heart rate (tachycardia).

Red flags — act now

- Potassium below 2.5 mEq/L — high risk of life-threatening heart rhythms
- New irregular or very fast heartbeat, chest pain, or feeling faint
- Sudden severe muscle weakness or paralysis, especially trouble breathing
- Confusion, extreme fatigue, or passing out
- Taking digoxin (a heart medicine) — even mild low potassium can cause dangerous toxicity

Treat it — in this order

1. Check and replace magnesium — give 2 grams IV magnesium sulfate over 15 minutes if severe, or 400–800 mg oral magnesium oxide daily if mild → Magnesium sits on the pump (the sodium-potassium ATPase) that keeps potassium inside cells. Without magnesium, the pump leaks and potassium spills out no matter how much you give. Replacing magnesium plugs the leak so potassium can stay in.
2. Stop or reduce the cause — hold loop or thiazide diuretics, treat vomiting or diarrhea, stop beta-agonist inhalers if overused, or treat high aldosterone → Shuts off the drain so potassium stops leaving. Without this step, low potassium (hypokalemia) will come right back after you replace it.
3. Give potassium chloride — oral 40–100 mEq daily in divided doses if mild (K 3.0–3.5) and asymptomatic; IV 10 mEq/hour via peripheral line or up to 20 mEq/hour via central line if severe (K below 2.5) or symptomatic (weakness, arrhythmia, a stalled gut (ileus)) → Replaces the missing potassium so nerve and muscle cells can recharge to their normal resting voltage. This stops weakness, fixes gut movement, and stabilizes the heart rhythm.
4. Use a potassium-sparing diuretic (spironolactone 25–100 mg daily or eplerenone 25–50 mg daily) if high aldosterone is the cause, or amiloride 5–10 mg daily if the kidney is losing potassium for another reason → Blocks aldosterone's signal to the distal tubule in the kidney, so the kidney stops dumping potassium into the urine and holds onto it instead. This prevents chronic potassium loss.
5. Put the patient on continuous heart monitoring (telemetry) and repeat EKGs every 4–6 hours until potassium is above 3.0 mEq/L → Catches dangerous rhythm changes (like torsades de pointes or ventricular fibrillation) before they kill the patient. Lets you see when the heart is getting safer as potassium rises.

Your top priorities at the bedside

1. Place patient on continuous telemetry monitor and watch for peaked T-waves becoming flat, U-waves appearing, ST depression, or wide QRS complex — why: Low potassium makes heart muscle cells repolarize (reset after each beat) too slowly which shows as EKG changes; arrhythmias including ventricular fibrillation can happen suddenly especially when potassium drops below 2.5 and cause cardiac arrest
2. Start IV potassium chloride replacement through a pump at no more than 10 milliequivalents per hour through a peripheral arm vein, or 20 milliequivalents per hour if using a central line — why: Potassium given faster than 10 milliequivalents per hour into a small vein causes severe burning pain and vein inflammation; too-fast infusion into any vein can cause sudden high potassium in the heart during that minute and trigger fatal arrhythmia even though total body potassium is low

3. Check magnesium level and give magnesium sulfate IV or magnesium oxide by mouth if magnesium is low — why: Magnesium (a mineral) controls the pump that moves potassium into cells; when magnesium is low the kidneys waste potassium no matter how much you give, so you cannot fix potassium until magnesium is fixed first