

Hypokalemia

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

THE STORY IN ONE SENTENCE

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

“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.”

Normal anatomy — the body's job

Potassium (symbol K^+) is the most common positively charged particle inside your cells. A pump called the sodium-potassium ATPase sits in every cell membrane and constantly trades sodium out for potassium in, using energy. This creates a voltage difference across the cell wall — think of it like charging a battery. That voltage is called the resting membrane potential, and it is what lets nerve cells send signals and muscle cells squeeze on command.

BODY'S JOB

The sodium-potassium pump keeps potassium concentrated inside cells (about 140 mmol/L inside versus 3.5–5.0 mmol/L in the blood). This steep difference makes cells electrically 'loaded' and ready to fire. When a nerve or muscle cell needs to act, tiny gates open, ions rush across, and the voltage flips — that is the signal. Afterward, the pump resets everything so the cell can fire again. Your heart, skeletal muscles, smooth muscles in your gut, and every nerve depend on this cycle happening fast and reliably.

1. The sodium-potassium pump pushes 3 sodium ions out and pulls 2 potassium ions in with every cycle, using one ATP (energy molecule).
2. This keeps the inside of the cell negative (about -90 mV in a muscle cell) compared to the outside — like a charged battery.
3. When a signal arrives, sodium gates open and sodium rushes in, flipping the voltage positive (depolarization) — the cell fires.
4. Right after, potassium gates open and potassium flows out, bringing the voltage back down (repolarization) — the cell resets.
5. The pump then restores the original balance so the cell is ready to fire again.

6. Normal blood potassium (3.5–5.0 mEq/L) keeps this cycle smooth and quick; if blood potassium drops, the cycle slows and cells become unstable.

What breaks

Low potassium (Hypokalemia) happens when you lose potassium from your body or when potassium suddenly shifts out of your blood and hides inside your cells (even though your total body potassium might be low either way). Once blood potassium drops, the voltage difference across cell membranes gets bigger — cells become over-excitabile at first, then they fatigue and stop working right. Muscles (including your heart) and nerves are hit hardest because they depend on that voltage to fire and reset quickly.

CLINICAL TERM

Serum potassium < 3.5 mEq/L. Hyperpolarization of the resting membrane potential increases the threshold for depolarization, leading to muscle weakness, cardiac conduction delays, and arrhythmia risk.

How the body compensates

The body tries to protect vital functions by shifting potassium between compartments and adjusting kidney handling, but these fixes are limited. Blood pressure and heart rate may stay normal for a while even as potassium drops dangerously low — this 'stable' window is deceptive and hides how close the patient is to a life-threatening arrhythmia. You must recognize the trajectory, not just the snapshot.

1. The kidneys try to hold onto potassium by reabsorbing more in the collecting ducts (aldosterone effect), but if the cause is diuretics or high aldosterone itself, this compensation fails.
2. Cells release a small amount of potassium into the blood to buffer the drop, but total body stores keep falling if losses continue.
3. The heart tries to maintain output, but the long QT and unstable repolarization set up re-entry circuits — the electrical signal can loop back and trigger torsades de pointes without warning.
4. Muscles try to keep working, but as potassium drops below 3.0 mEq/L, weakness becomes obvious; below 2.5 mEq/L, respiratory muscles can fail.
5. The gut's smooth muscle slows (hypokalemia blunts muscle contraction), causing constipation, bloating, and eventually a stalled gut (ileus) (the bowel stops moving entirely).

6. The crash point is **torsades de pointes** (a polymorphic ventricular a fast heart rate (tachycardia)) or **ventricular fibrillation** — triggered by the prolonged QT and early after-depolarizations. This is especially dangerous if the patient is also on a QT-prolonging drug (antipsychotics, certain antibiotics, antiarrhythmics), has low magnesium, or has structural heart disease.
7. Low potassium (Hypokalemia) makes **digoxin toxicity** much more likely — low potassium lets digoxin bind more tightly to the sodium-potassium pump, so even therapeutic digoxin levels can become toxic (causing nausea, vision changes, heart blocks, or ventricular arrhythmias).
8. Trend over time: a potassium of 3.2 mEq/L that is dropping (3.8 yesterday, 3.2 today) is more dangerous than a stable 3.2; always ask *which direction are we going?*

Domino effect

1. Blood potassium drops below 3.5 mEq/L. — 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). — 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. — 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. — 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. — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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).
- Complete muscle paralysis, especially if potassium drops very low suddenly: Severe low potassium (hypokalemia) (below 2.5 mEq/L) can paralyze muscles entirely because the cells cannot generate the electrical spark needed to contract — this is most dangerous in the breathing muscles.
- Trouble breathing or shallow breaths: The diaphragm and chest muscles need potassium to work. When potassium falls very low, these muscles weaken and cannot pull air into the lungs properly, leading to respiratory failure.

Focused exam

- EKG shows flattened or inverted T waves and a small U wave after the T wave: Potassium controls how heart muscle cells reset after each beat. When potassium drops below 3.5 mEq/L, the cells take longer to recharge, so the T wave flattens and a little extra bump (the U wave) appears. This is your first electrical warning that potassium is too low.
- Weakness in the legs more than the arms, trouble standing up from a chair: Potassium sets the resting charge inside every muscle cell. When it drops, the charge falls and muscles cannot fire strongly. Big muscles in the legs weaken first because they need the most power.
- Belly is soft, bloated, and bowel sounds are quiet or absent: The smooth muscle in the gut wall needs potassium to squeeze and move food along. When potassium is low, the gut stops moving (this is called a stalled gut (ileus)). Food and gas pile up inside.
- Patient says they are peeing large amounts of pale urine even when they are not drinking much: Low potassium damages the cells in the kidney's collecting duct so they cannot respond to ADH (the hormone that tells the kidney to hold water). The kidney keeps dumping dilute urine even when the body needs to save water. This is called nephrogenic diabetes insipidus.
- Cramping, tingling, or aching in the arms and legs: When potassium first drops, nerves become jumpy and fire too easily, causing cramps and pins-and-needles sensations. As it falls further, muscles stop working altogether.
- Blood pressure drops when the patient stands, or the patient feels dizzy and faint: The heart is a muscle. When potassium is low, the heart contracts more weakly and cannot pump enough blood to the brain when the person stands up. This is orthostatic low blood pressure (hypotension).

Diagnostics · every test answers a question

- Serum potassium (part of a basic metabolic panel, BMP) → Is potassium actually low, and how severe is it?
- Serum magnesium → Is magnesium also low?
- Urine potassium or spot urine potassium-to-creatinine ratio → Is the kidney losing potassium, or is potassium leaving through the gut or shifting into cells?
- Arterial blood gas (ABG) or venous blood gas (VBG) → Is the blood too alkaline (high pH), and is that pushing potassium into cells?
- Aldosterone and renin levels (plasma aldosterone concentration and plasma renin activity) → Is the body making too much aldosterone, which tells the kidney to dump potassium?

- EKG (12-lead electrocardiogram) → Is the heart showing dangerous electrical changes from low potassium?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.
- Make sure the patient is eating enough calories and protein, especially after a period of not eating (starvation, anorexia, alcoholism, or refeeding) → Calories and insulin signal cells to pull potassium, magnesium, and phosphate back in from the blood. If you feed someone who has been starving without giving these minerals first, potassium crashes dangerously low (refeeding syndrome). Giving food with close lab monitoring prevents this crash.

Medications

Magnesium sulfate or magnesium oxide (if magnesium is low) · Magnesium supplement

Magnesium is needed to keep potassium inside cells. When magnesium is low, the tiny pumps in your cell walls that hold potassium in place stop working right, so potassium leaks out no matter how much you give. Replacing magnesium first fixes the pump so potassium can stay where it belongs.

1. Magnesium enters the bloodstream from the gut (oral) or directly (IV).
2. It travels to cells all over the body.
3. Magnesium activates the sodium-potassium ATPase pump in the cell wall.
4. The pump now works correctly and pulls potassium back into the cell.
5. Potassium levels in the blood rise and stay stable.

SAFETY WATCH

Too much magnesium can slow your heart, make you sleepy, or weaken your muscles. · Go slow if kidney function is poor—kidneys clear extra magnesium, so buildup is a risk. · Watch for diarrhea with oral magnesium; switch to IV if it happens.

Potassium chloride (KCl) - oral · Electrolyte replacement (oral potassium salt)

This is potassium in pill or liquid form. You swallow it, it dissolves in your stomach, and your gut absorbs it into your blood. Your kidneys and cells then move it where it is needed. Oral potassium is safe and works well when the shortage is mild to moderate and the person has no serious symptoms.

1. You swallow the potassium chloride pill or liquid.
2. It dissolves in your stomach and small intestine.
3. Potassium is absorbed through the gut wall into the blood.
4. Blood potassium level rises.
5. Cells pull potassium in through their pumps, restoring normal balance.

SAFETY WATCH

Pills can irritate or burn the esophagus (the tube to your stomach); take with food and plenty of water. · Too much potassium can stop the heart—never take more than prescribed. · Watch closely if kidney function is weak; kidneys must clear extra potassium or it builds up dangerously.

Potassium chloride (KCl) - intravenous (IV) · Electrolyte replacement (intravenous potassium salt)

This is potassium given directly into a vein through an IV line. It skips the gut and raises blood potassium quickly. Use IV potassium when the level is very low (under 2.5 mEq/L), the person has dangerous symptoms (muscle weakness, dangerous heart rhythms), or they cannot swallow.

1. Potassium chloride is mixed in IV fluid (saline or dextrose).
2. It flows through the IV catheter into a vein.
3. Potassium spreads quickly through the bloodstream.
4. The heart, muscles, and nerves immediately have more potassium to use.
5. Cells pull potassium in over the next hours, and the blood level stabilizes.

SAFETY WATCH

Never give faster than 10 mEq per hour through a regular arm or hand vein—it burns and can damage the vein. · Never exceed 20 mEq per hour even through a central line (a larger IV in the chest)—too fast can stop the heart. · Always monitor the heart rhythm on a cardiac monitor during IV potassium. · Recheck blood potassium every few hours to avoid giving too much. · If the patient feels burning or pain at the IV site, slow the rate or dilute the solution more.

Spirolactone · Potassium-sparing diuretic (aldosterone antagonist)

Spirolactone blocks aldosterone, a hormone made by the adrenal glands (two small glands on top of your kidneys). Aldosterone tells the kidney to dump potassium into the urine and hold onto sodium. By blocking aldosterone, spironolactone stops the kidney from wasting potassium, so levels stay up without needing constant replacement.

1. You swallow the spironolactone pill.
2. It is absorbed in the gut and travels to the kidneys.
3. Spirolactone binds to aldosterone receptors in the kidney tubule cells and blocks them.
4. Without aldosterone's signal, the kidney stops pumping potassium into the urine.
5. Potassium stays in the blood instead of being lost.
6. Sodium and water leave in the urine (mild diuretic effect), but potassium is saved.

SAFETY WATCH

Can raise potassium too high, especially if kidney function is poor or you are also taking ACE inhibitors, ARBs, or potassium supplements—monitor blood potassium regularly. · Can cause breast tenderness or enlargement in men (gynecomastia) because it also blocks some testosterone. · Can make you dizzy when you stand up (low blood pressure). · Avoid if potassium is already normal or high.

Eplerenone · Potassium-sparing diuretic (selective aldosterone antagonist)

Eplerenone works just like spironolactone—it blocks aldosterone so the kidney stops wasting potassium. It is more selective, meaning it targets aldosterone receptors more precisely and causes fewer hormone-related side effects (like breast tenderness). Use it when high aldosterone is driving potassium loss and you want to avoid spironolactone's side effects.

1. You swallow the eplerenone pill.
2. It is absorbed and travels to the kidneys.
3. Eplerenone binds tightly to aldosterone receptors in the kidney tubule and blocks them.
4. Aldosterone can no longer tell the kidney to dump potassium.
5. Potassium stays in the blood; sodium and water are lost in urine.
6. Blood potassium level rises slowly over days.

SAFETY WATCH

Can raise potassium dangerously high, especially with poor kidney function or other potassium-raising drugs—check blood potassium often. · Can lower blood pressure and cause dizziness. · Costs more than spironolactone. · Avoid if potassium is already normal or elevated.

Stop or reduce the medication causing potassium loss · Cause removal (not a drug—a treatment strategy)

Many common drugs force the kidney to throw away potassium. Loop diuretics (furosemide, bumetanide) and thiazide diuretics (hydrochlorothiazide) make you urinate more and dump potassium in the process. Beta-agonists (albuterol inhalers for asthma) push potassium from blood into cells, making the blood level look low. Stopping or lowering the dose of these drugs stops the potassium drain.

1. Identify the drug causing potassium loss (common culprits: furosemide, hydrochlorothiazide, albuterol).
2. Stop the drug or lower the dose if medically safe.
3. The kidney stops being forced to waste potassium (if a diuretic was the cause).

4. Or cells stop pulling potassium out of the blood (if a beta-agonist was the cause).
5. Potassium level stabilizes or rises naturally.

SAFETY WATCH

Do not stop a necessary medication without your doctor's guidance—heart failure or high blood pressure may worsen. · Stopping a diuretic suddenly can cause fluid buildup and swelling. · Always discuss alternatives with your care team.

Red flags

- 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

Nursing priorities

1. Place patient on continuous telemetry monitor and watch for peaked T-waves becoming flat, U-waves appearing, ST depression, or wide QRS complex — 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 — 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 — 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
4. Assess muscle strength by asking patient to squeeze your fingers, lift legs against resistance, and take a deep breath — Potassium is required for skeletal muscle contraction and for the diaphragm (breathing muscle) to work; weakness that reaches the diaphragm causes respiratory failure and need for a breathing tube

5. Check blood sugar and give insulin carefully if ordered, because insulin drives potassium from blood into cells and can drop the level more — Insulin activates the sodium-potassium pump on cell membranes that pulls potassium out of the bloodstream into cells; this can make lab potassium dangerously low even though total body potassium has not changed
6. Listen to bowel sounds in all four quadrants and ask about last bowel movement — Low potassium slows smooth muscle contraction in the intestines causing constipation or even a stalled gut (ileus) (intestines stop moving entirely); severe cases cause the colon to dilate and risk perforation
7. Hold or call provider about diuretic doses, and review medication list for other potassium-wasting drugs — Continuing the medicine that caused potassium loss means you are trying to fill a bucket with a hole in it; loop diuretics like furosemide and thiazides like hydrochlorothiazide waste 10 to 40 milliequivalents of potassium daily in urine
8. Recheck potassium level 2 to 4 hours after IV replacement starts and after each 40 milliequivalents given — Potassium moves from blood into cells over hours so the serum level does not show total body deficit; rechecking prevents over-replacement that causes high potassium (hyperkalemia) and lets you see if kidneys are still wasting potassium

Sources

- KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
- UpToDate: Hypokalemia — Clinical Features and Treatment
- American Heart Association (AHA) — Electrolyte Disorders and Arrhythmias
- National Kidney Foundation — Potassium and Chronic Kidney Disease

Last reviewed: 2026-07-07