# Hypokalemia

$K^+$ < 3.5 mEq/L · Fix the magnesium, or the potassium will not stay

**10 mEq → 0.05–0.1** *mEq/L, less on a diuretic*

## HOW BAD IS IT *the ECG matters more than the number, and neither one is reassuring on its own*

| $K^+$ | What you may see | What it means for you tonight |
|---|---|---|
| **3.0–3.4** *mild* | Usually nothing. Fatigue, cramps, constipation. | **Oral replacement, look for the cause.** Not benign in heart failure, on digoxin, or post-MI, where the target is **≥ 4.0** because low $K^+$ lowers the threshold for ventricular ectopy. |
| **2.5–2.9** *moderate* | Weakness, ileus, **flat T waves, ST depression, U waves**. | **Combine oral and IV, put the patient on telemetry.** Send a magnesium at the same time, not after the potassium fails. |
| **< 2.5** *severe* | Ascending paralysis, rhabdomyolysis, **long QU, PVCs, VT, torsades**. | **Emergency.** Central IV replacement, continuous monitoring, recheck every 1–2 h. Give magnesium empirically while you wait for the level. |

## THE ARITHMETIC OF REPLACEMENT *the serum number badly under-reads the size of the hole*

### THE 10 mEq RULE, AND WHY IT OVER-PROMISES

**Classic teaching is that every 10 mEq of KCl raises serum $K^+$ by 0.1 mEq/L. Treat that as a best case, not an average.** It reflects acute IV dosing: in a medical ICU series, 20 mEq infusions raised $K^+$ by a mean of **0.25 mEq/L** measured straight afterwards. **Across a hospital day it under-delivers.** In 800 supplemented inpatients the median was **0.05 mEq/L per 10 mEq**, rising to **0.07** off potassium-affecting drugs and falling to **0.03 on a loop diuretic. Expect about half the textbook rise in anyone still on a diuretic, and recheck rather than trusting the arithmetic.**

### WHY THE DEFICIT IS BIGGER THAN IT LOOKS

**Only about 2% of body potassium is extracellular**, so the serum value is the tip of the iceberg. Each **1 mEq/L** fall below normal represents a **200–400 mEq total-body deficit**. A $K^+$ of 2.5 is a whole-body hole of **400 mEq or more**, which is why the level drifts back down after you stop replacing and why one 40 mEq dose almost never finishes the job.

## HOW TO ACTUALLY GIVE IT *oral is faster than you think, IV is more dangerous than you think*

| $K^+$ | Regimen | Expected rise | Notes |
|---|---|---|---|
| **3.0–3.4** | **KCl 40 mEq PO** once, then recheck | ~0.2–0.4 mEq/L | Oral raises the level about as fast as IV and carries **no arrhythmia or phlebitis risk**. Give with food, since KCl is the classic cause of nausea and pill ulcers. |
| **2.5–2.9** | **KCl 40 mEq PO plus 20–40 mEq IV**, in divided doses | ~0.4–0.8 mEq/L | Split the route so you are not held hostage by the IV rate cap. **Telemetry once $K^+$ < 3.0.** |
| **< 2.5** or arrhythmia | **IV KCl by infusion** plus oral if the gut works, plus **$MgSO_4$** | Recheck q1–2 h | Do not chase a target with a single large bolus. **Reassess the level and the ECG between doses**, because the same infusion that is safe at 2.2 is not safe at 3.8. |

### RATE AND ROUTE LIMITS

**Peripheral: 10 mEq/h, concentration ≤ 40 mEq/L.** Faster or stronger burns, and phlebitis is the usual reason a peripheral run gets abandoned half finished. **Central: 20 mEq/h.** Rates up to **40 mEq/h are reserved for arrest-level hypokalemia with ECG changes**, central access, and continuous monitoring.

### MIX IT IN SALINE, NOT DEXTROSE

**Dextrose triggers endogenous insulin, which drives potassium into cells** and can drop the serum level further before it rises. Use a **non-dextrose saline diluent**. Same logic explains why you never start insulin in DKA until $K^+$ is **≥ 3.3–3.5**.

## THE MAGNESIUM RULE *if the potassium will not come up, this is nearly always why*

1. **Roughly half of hypokalemic inpatients are also magnesium depleted.** The two share every cause that matters on the wards: loop and thiazide diuretics, alcohol use, diarrhea, proton pump inhibitors, amphotericin, aminoglycosides, cisplatin, and poor intake.
2. **The mechanism is ROMK.** Intracellular magnesium normally plugs the ROMK channel in the thick ascending limb and distal nephron. When magnesium falls, that plug is released, **ROMK opens, and potassium is secreted straight into the urine**. Every dose you give is promptly excreted, so the level barely moves.
3. **Dose it properly. $MgSO_4$ 2 g IV over 1–2 h** for Mg 1.5–1.9, **4 g IV over 4 h** for Mg < 1.0, and expect a severely depleted patient to need **8–12 g over 24 h**. Infuse slowly: a fast push causes flushing, warmth, and hypotension. **Halve the dose in renal impairment**, since magnesium is renally cleared.
4. **Give magnesium and potassium together, not in sequence.** Waiting for the magnesium to normalize first simply wastes hours of replacement. **Check a magnesium level on anyone whose $K^+$ has not budged after two adequate doses**, and treat empirically if the patient is unstable. Magnesium depletion also causes **functional hypoparathyroidism**, so a refractory hypocalcemia alongside it points at the same missing cation.

## FIND THE CAUSE *one urine sample, then split by blood pressure and acid-base*

| Step | What it tells you |
|---|---|
| **Urine $K^+$ / creatinine** | **< 13 mEq/g: the kidney is behaving.** Losses are **GI** (diarrhea, laxatives, vomiting via secondary aldosteronism), **shift**, or poor intake. **> 13 mEq/g: renal potassium wasting.** Use this ratio rather than the **TTKG**, whose own authors retracted the assumptions behind it. |
| **Renal loss, normal BP, metabolic acidosis** | **RTA type 1 and type 2**, DKA and other ketoacidoses, acetazolamide, toluene inhalation. Acidosis plus renal potassium wasting is the pairing that points away from a diuretic. |
| **Renal loss, normal BP, metabolic alkalosis** | **Diuretics and vomiting are the common answers.** Separate them with a **urine chloride**: low in vomiting and in the between-dose window of a diuretic, high while a diuretic is active or in **Bartter and Gitelman** syndromes. |
| **Renal loss, high BP, metabolic alkalosis** | **Mineralocorticoid excess.** Primary aldosteronism (check an aldosterone to renin ratio), renal artery stenosis, Cushing syndrome, licorice, and **Liddle syndrome**. Unprovoked hypokalemia in a hypertensive patient is a screening indication. |
| **Transcellular shift** *no real deficit* | **Insulin, beta-2 agonists, alkalosis, refeeding**, and **periodic paralysis** (familial, or thyrotoxic in young Asian men). The total body store is normal, so **aggressive replacement causes rebound hyperkalemia** once the shift reverses. |

## PEARLS & PITFALLS

- **Check the magnesium first, every time.** Refractory hypokalemia is hypomagnesemia until proven otherwise.
- **Aim for $K^+$ ≥ 4.0 in cardiac patients**, on digoxin, and after infarction or arrest. Hypokalemia potentiates digoxin toxicity at a therapeutic level.
- **Torsades gets 2 g of magnesium regardless of the potassium level.**
- **Watch for rebound hyperkalemia** in periodic paralysis, in shift states once the driver clears, and in reduced eGFR.
- **Stop the cause or you will do this again tomorrow.** Review the diuretic dose, consider a potassium-sparing agent, and remember amphotericin and aminoglycosides.
- **Recheck 1–2 h after IV and 2–4 h after oral** replacement, not sooner.

**References:** Kruse & Carlson, Arch Intern Med 1990 (IV KCl infusions in the MICU) · Am J Health Syst Pharm 2024 (potassium supplementation in 800 inpatients) · Huang & Kuo, mechanism of hypokalemia in magnesium deficiency, JASN · KDIGO and NEJM reviews of potassium disorders.