PHARMACOLOGY | RENAL / CARDIOVASCULAR | NCLEX 2026 | NGN READY

Potassium-Sparing Diuretics

Spironolactone · Eplerenone · Triamterene — the three diuretics that **conserve potassium** while removing sodium and water. Their shared red flag is **HYPERKALEMIA**.

SOURCE NOTE: This topic was not present in the uploaded study notes for this project. Content drawn from standard NGN NCLEX 2026 references (Saunders Comprehensive Review, ATI Pharmacology Made Easy, Lippincott's Nursing Drug Guide) and current FDA prescribing information.

SECTION ONE

1 Definition & Overview

Potassium-sparing diuretics are *weak* diuretics that promote sodium and water excretion at the late distal tubule and collecting duct **without** wasting potassium.

They are almost always used **in combination** with a loop or thiazide diuretic (which waste K^+) to balance the picture — or as primary therapy in heart failure and hyperaldosteronism.

- **Two mechanisms:** aldosterone-receptor antagonists (spironolactone, eplerenone) vs. direct epithelial sodium channel blockers (triamterene, amiloride).
- **Why they matter:** They are mortality-reducing drugs in HFrEF — but they can cause **life-threatening hyperkalemia**, especially with ACE-Is, ARBs, NSAIDs, or salt substitutes.

QUICK FACTS

Where they work

Site: **Late distal convoluted tubule + collecting duct** — the very last stop before urine exits the nephron.

Effect: **Na^+ & H_2O out · K^+ stays in**

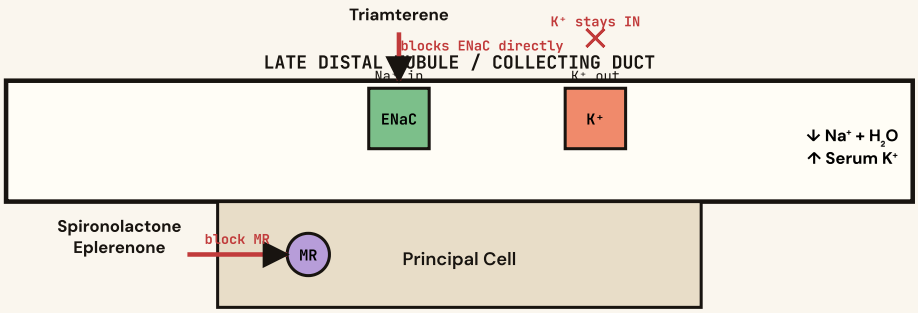
Potency: **Weak diuretic, strong K^+ effect**

Onset (oral): hours to days depending on agent

SECTION TWO

2 Mechanism of Action (Simplified)

How K⁺-Sparing Diuretics Work



1. **Aldosterone** normally tells the principal cell of the collecting duct to retain Na^+ and water, and dump K^+ into the urine.
2. **Spironolactone & Eplerenone** competitively block the **mineralocorticoid (aldosterone) receptor (MR)** inside the cell.
3. **Triamterene & Amiloride** take a different route: they directly block the **epithelial sodium channel (ENaC)** on the apical membrane — no aldosterone needed.
4. Either way, less Na^+ is reabsorbed → less driving force to push K^+ out into urine → **Na^+ & water leave the body, K^+ is conserved**.
5. Net result: mild diuresis, lowered BP, K^+ rises. **HYPERKALEMIA risk.**

SECTION THREE

3 Signs & Symptoms — Recognizing Hyperkalemia 🚨

Normal K⁺: 3.5 – 5.0 mEq/L. The most dangerous adverse effect of every drug in this class is rising potassium. Memorize these in order — they track tightly with rising serum levels.

EARLY / MILD · K⁺ 5.1 - 6.0

Subtle Neuromuscular Cues

- **Muscle weakness** (legs first, then trunk)
- Fatigue, lethargy
- Paresthesias — tingling around mouth, fingers, toes
- Nausea, abdominal cramping, diarrhea
- Anxiety / irritability

LATE / SEVERE · K⁺ > 6.0 🚨

Cardiac & Skeletal Failure

- **Peaked T waves** on ECG (first sign — K⁺ ~5.5–6.5)
- Widened QRS, prolonged PR, flat/absent P waves
- Sine wave → **V-fib / asystole**
- Bradycardia, irregular pulse
- Flaccid paralysis, ascending weakness
- Respiratory muscle failure

RED FLAG MNEMONIC — MURDER

Muscle weakness · **U**rine output decreased · **R**espiratory distress · **D**ecreased cardiac contractility · **E**CG changes (peaked T) · **R**eflexes diminished/areflexia

SECTION FOUR

4 Priority Nursing Interventions — ABC Framework

A · AIRWAY / CARDIAC

Heart First

- Continuous **cardiac monitoring** if $K^+ \geq 6$ or symptomatic
- **Obtain 12-lead ECG** — peaked T = emergency
- Hold drug & **notify HCP** for $K^+ > 5.5$
- Prepare for emergency Tx: IV calcium gluconate (stabilize), insulin + D50, kayexalate/patiromer, dialysis

B · BASELINE & LABS

Assess Before Each Dose

- Check **K^+, BUN, creatinine, eGFR** at baseline + 1 wk + monthly
- **Daily weight** (same scale, same time, same clothes)
- Strict **I&O** — diuresis is mild but real
- Vital signs — **BP and HR** (hold for SBP < 90 or HR < 60 per protocol)

C · CAUTION / SAFETY

Drug Interactions to Catch

- **ACE-I / ARB** + K-sparer = hyperkalemia risk ↑↑
- **NSAIDs** reduce renal flow → ↑ K^+
- **Heparin, trimethoprim, tacrolimus** ↑ K^+
- Watch **renal impairment** — hold if eGFR < 30
- **Fall risk** — orthostatic BP, especially elderly

SECTION FIVE

5 Drug-by-Drug Comparison

	Spironolactone Aldactone · CaroSpir	Eplerenone Inspra	Triamterene Dyrenium · in Dyazide/Maxzide
CLASS	Aldosterone-receptor antagonist (non-selective)	Selective aldosterone-receptor antagonist	Direct ENaC blocker — *not* aldosterone-dependent
INDICATIONS	HFrEF, resistant HTN, primary hyperaldosteronism, ascites in cirrhosis, off-label for PCOS hirsutism & acne	HF post-MI w/ LV dysfunction (EF ≤ 40%), HTN	Edema (with HCTZ), mild HTN, prevents K^+ loss from thiazides/loops
ROUTE	PO (tablet, suspension)	PO	PO
ONSET	24–48 hr (delayed — full effect 2–3 days)	~ 1.5 hr (fastest of the three)	2–4 hr
PEAK	2–3 days for full diuretic effect	1.5 hr (plasma); 4 wk for BP effect	6–8 hr
DURATION	2–3 days (long active metabolites)	~ 24 hr	7–9 hr
TYPICAL DOSE	HF: 12.5–25 mg/day · HTN: 25–100 mg/day	HF: start 25 mg/day → 50 mg · HTN: 50–100 mg/day	100 mg BID (max 300 mg/day)
TAKE WITH FOOD?	**YES** — improves absorption + ↓ GI upset	With or without food	**YES** — ↓ GI upset
COMMON SE	**Gynecomastia, menstrual irregularities**, ↓ libido, dizziness, GI upset, drowsiness	Dizziness, fatigue, headache, ↑ triglycerides (much less gynecomastia)	Nausea/vomiting, dizziness, photosensitivity, **blue-green urine** (harmless)
SERIOUS AE 🚨	**Hyperkalemia**, metabolic acidosis, AKI, gynecomastia (irreversible if > 6 mo)	**Hyperkalemia**, angioedema (rare), AKI	**Hyperkalemia**, **megaloblastic anemia** (folate antagonism), **kidney stones** (drug crystals), nephrotoxicity
CONTRAINDICATIONS	K^+ > 5.0, Addison's disease, anuria, severe renal impairment	K^+ > 5.5, CrCl < 30, type 2 DM w/ microalbuminuria, severe hepatic disease, strong CYP3A4 inhibitors	K^+ > 5.5, anuria, severe hepatic/renal disease, history of nephrolithiasis
PATIENT TEACHING HOOK	"**The breast-tender pill**" — report breast changes, take with food, AM dose	"**Cleaner spironolactone**" — fewer hormonal effects, watch CYP3A4 (no grapefruit, no ketoconazole)	"**Blue pee, drink three (liters)**" — hydrate to prevent stones; expect color change

SECTION SIX

6 NCLEX Test Traps ⚠

✕ WRONG THINKING	✓ RIGHT THINKING
"Spironolactone is a diuretic — the patient on it should eat lots of bananas and orange juice to replace the potassium they're losing."	K^+-sparing diuretics **conserve** potassium. **AVOID** high-K^+ foods (bananas, oranges, potatoes, spinach, tomatoes) and salt substitutes (KCl-based).
"Patient started spironolactone yesterday and BP is unchanged — the drug must not be working, call the HCP for a dose increase."	Spironolactone has a **delayed onset (24–48 hr)** with full effect at **2–3 days**. One day is too early to judge response. Continue monitoring.
"Patient on spironolactone and lisinopril for HF — both lower BP, this is great combination therapy."	Both drugs **raise potassium**. ACE-I + K-sparer = serious hyperkalemia risk. Check K^+ baseline, day 3, day 7, then monthly. Hold and notify HCP for $K^+ > 5.5$.
"Patient on triamterene has blue-green urine — must be a urinary tract infection or liver problem."	Blue-green/blue urine is a **harmless, expected side effect** of triamterene (the drug fluoresces). Reassure and educate — no intervention needed.
"K^+ is 6.8 and the patient has peaked T waves. Give IV potassium-binder (kayexalate) first — that lowers potassium fastest."	For **cardiac instability**: give **IV calcium gluconate FIRST** to stabilize the myocardium (works in minutes). Then shift K^+ in with insulin + D50 + albuterol, then eliminate with kayexalate/patiromer or dialysis.

SECTION SEVEN

7 Patient & Family Education

FOODS · DIET

What to Avoid

- **High-K^+ foods:** bananas, oranges, melons, dried fruit, potatoes, tomatoes, spinach, avocados, beans, nuts
- **Salt substitutes** — most are KCl (potassium chloride)
- **Low-sodium "lite" salt** — same issue
- Avoid **OTC NSAIDs** (ibuprofen, naproxen) — call HCP first
- **Grapefruit** — eplerenone only (CYP3A4)

WHEN TO CALL HCP

Report Immediately

- **Muscle weakness, cramps, or tingling** around mouth/fingers
- **Irregular or slow heartbeat**, palpitations
- Severe dizziness or syncope
- **Breast tenderness or enlargement** (spironolactone)
- **Flank pain, blood in urine** (triamterene — stones)
- Unusual fatigue or pallor (anemia w/ triamterene)

DAILY HABITS

Living With the Drug

- **Take in the morning** — diuresis can disturb sleep if PM-dosed
- Take with **food** (spironolactone, triamterene)
- **Weigh daily**, same time, report ≥ 2 lb in 24 hr or ≥ 5 lb in 1 wk
- Stand up **slowly** — orthostatic hypotension risk
- **Hydrate well** — especially on triamterene (stones)
- **Sun protection** — triamterene = photosensitivity

LAB FOLLOW-UP

What Will Be Checked

- **Basic metabolic panel** (K^+, Na^+, BUN, Cr) at baseline, **1 week, 1 month**, then every 3–6 months
- More often if dose changes, illness, or other K^+-raising drugs added
- Triamterene patients: **CBC** for anemia surveillance
- Keep a current med list — show every provider (incl. dentist)
- Don't stop suddenly in HF — can trigger fluid overload

SECTION EIGHT

8 Memory Boosters & Mnemonics

"The K Stays"

K-sparing diuretics → **K stays in the body** → think **HYPER**kalemia, not **HYPO**.

This single sentence flips the rule for every other diuretic class you'll learn.

"SET" the K-Sparers

S — Spironolactone
E — Eplerenone
T — Triamterene
(plus Amiloride — the bonus "A")

Gynecomastia → "Spiro"

Spironolactone = **Spiro**l of estrogen-like effects.

The non-selective MR blocker also binds androgen & progesterone receptors — that's why it causes **breast tenderness, gynecomastia, and menstrual changes**. Eplerenone is selective → far less of this.

"Blue Pee, Drink Three"

Triamterene turns urine **blue-green** (harmless, expected).

The "drink three" reminds you to push **3 L fluid/day** to prevent triamterene **kidney stones**.

MURDER for Hyperkalemia

Muscle weakness
Urine output decreased
Respiratory distress
Decreased cardiac contractility
ECG changes (peaked T)
Reflexes diminished

Treatment Order: "C BIG K Drop"

Calcium gluconate IV — stabilize heart (first!)
Beta-agonist (albuterol) — shift K^+ in
Insulin + D50 — shift K^+ in
Glucose & bicarb if acidotic
Kayexalate / patiromer — eliminate
Drop = dialysis if severe

SECTION NINE · NGN NEXT GEN

9 NCJMM Clinical Judgment Scenario

UNFOLDING CASE · DAY 8 OF THERAPY

Mr. R, 68 y/o male, HFrEF (EF 30%), CKD stage 3a, T2DM. Admitted to the med-surg unit 8 days ago for acute decompensation. Discharge meds were adjusted: lisinopril 20 mg PO daily, carvedilol 12.5 mg BID, furosemide 40 mg PO daily, and **spironolactone 25 mg PO daily** (new). He calls the clinic line today reporting "my legs feel heavy and I'm tired — and my heart feels funny."

BP	HR	RR	SpO_2	K^+	NA^+
108 / 64	52 irreg	18	95%	6.4	136

CR	GLU
1.9	142

ECG strip: peaked, narrow-based T waves in leads V2–V5; PR slightly prolonged.

1 RECOGNIZE CUES

Concerning cues to extract:

- Subjective: *heavy legs, fatigue, "heart feels funny"* — neuromuscular + cardiac
- Objective vitals: **HR 52 irregular**, BP soft at 108/64
- Labs: **K^+ 6.4 (high)**, **Cr 1.9 (rising)**
- ECG: **peaked T waves** + early PR prolongation
- Med profile: **ACE-I (lisinopril) + K-sparer (spironolactone) + CKD** — triple K^+ risk

2 ANALYZE CUES

Cues cluster around **symptomatic hyperkalemia from drug-induced K^+ retention**. Spironolactone (added 8 days ago) blocks aldosterone receptors → K^+ retained. Lisinopril (ACE-I) further reduces aldosterone → compounds K^+ rise. Baseline CKD reduces renal K^+ excretion. Cr rising from baseline suggests worsening renal function, amplifying every K^+ effect. Peaked T waves confirm **cardiac involvement** — this is no longer "mild" hyperkalemia.

3 PRIORITIZE HYPOTHESES

1. **(Priority) Symptomatic hyperkalemia with cardiac manifestations** — time-critical, life-threatening
2. Acute kidney injury overlay on CKD (rising Cr)
3. Bradyarrhythmia from K^+ effect ± carvedilol
4. Hypotension contributing to renal hypoperfusion

4 GENERATE SOLUTIONS

Immediate (life-safety):

- Send to ED / activate rapid response — do **not** manage outpatient
- Place on **continuous cardiac monitor**, 12-lead ECG
- IV access; anticipate **IV calcium gluconate** for membrane stabilization
- Anticipate **insulin + D50, albuterol neb, sodium bicarb** if acidotic, then **patiromer or sodium polystyrene sulfonate** for elimination

Medication adjustments (provider-directed):

- **Hold spironolactone** immediately
- Hold/decrease lisinopril pending K^+ trend
- Continue furosemide (may help shift K^+ out)
- Reassess all K^+-raising agents (no NSAIDs, no salt substitutes)

5 TAKE ACTION

1. Direct the patient to call **911** — symptomatic hyperkalemia with ECG changes is an emergency, not a clinic visit
2. On ED arrival: 12-lead ECG, cardiac monitor, IV access, stat BMP repeat
3. Administer **calcium gluconate 1 g IV** over 2–3 min per order
4. Insulin 10 units regular IV + D50W 25 g IV; albuterol 10 mg neb
5. Patiromer 25.2 g PO or sodium polystyrene sulfonate 15–30 g PO per order
6. Hold spironolactone & lisinopril; document held doses
7. Strict I&O, continuous telemetry, repeat K^+ in 1–2 hr
8. Educate patient and family on K^+ diet, salt substitutes, drug interactions before discharge

6 EVALUATE OUTCOMES

Expected (positive) outcomes within hours:

- K^+ trending down toward < 5.5 mEq/L on repeat labs
- ECG: T waves normalizing, PR & QRS within baseline
- HR returning to 60–80 bpm, rhythm regular
- Resolution of muscle weakness and "funny" cardiac sensation
- Cr stabilizing or trending down with hydration/medication adjustment

Reassess and escalate if: K^+ remains > 6, ECG changes persist or worsen (widened QRS, sine wave), hemodynamic instability — **dialysis** may be required.