Na⁺ K⁺ H₂O RAAS ⚖

NGN NCLEX 2026 · ENDOCRINE / FLUID & ELECTROLYTE

Hyperaldosteronism *vs* Hypoaldosteronism

Two opposite mineralocorticoid disorders that flip your sodium, potassium, acid–base, and blood pressure in mirror-image directions. Master one and the other becomes its perfect reverse.

`↑ ALDOSTERONE = CONN'S` `↓ ALDOSTERONE = ADDISON'S SPECTRUM` `RAAS AXIS` `K⁺ & NA⁺ BALANCE` `ACID-BASE`

📚 **Source transparency:** This reference was not drawn from Diane's uploaded personal notes. Content is synthesized from standard NCLEX references — **Saunders Comprehensive Review for NCLEX-RN**, **ATI RN Medical-Surgical / Pharmacology**, and **Lippincott**, aligned with the Endocrine Society clinical practice guidelines on primary aldosteronism and adrenal insufficiency. Always verify against current institutional protocol.

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Definition / Overview

What aldosterone does — and what happens when there's too much or too little of it.

↑ Hyperaldosteronism

- `WHAT:` Excess aldosterone secretion. **Primary** = adrenal adenoma (Conn's syndrome). **Secondary** = over-active RAAS (renal artery stenosis, HF, cirrhosis).
- `EFFECT:` Body holds onto sodium & water, dumps potassium & hydrogen.
- `WHY IT MATTERS:` Classic cause of resistant hypertension + unexplained hypokalemia.

↓ Hypoaldosteronism

- `WHAT:` Deficient aldosterone. Often part of **adrenal insufficiency / Addison's disease**; also seen with certain meds (ACE inhibitors, heparin, NSAIDs).
- `EFFECT:` Body loses sodium & water, retains potassium & hydrogen.
- `WHY IT MATTERS:` Causes hypotension, dehydration, and dangerous hyperkalemia.

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Pathophysiology (Simplified)

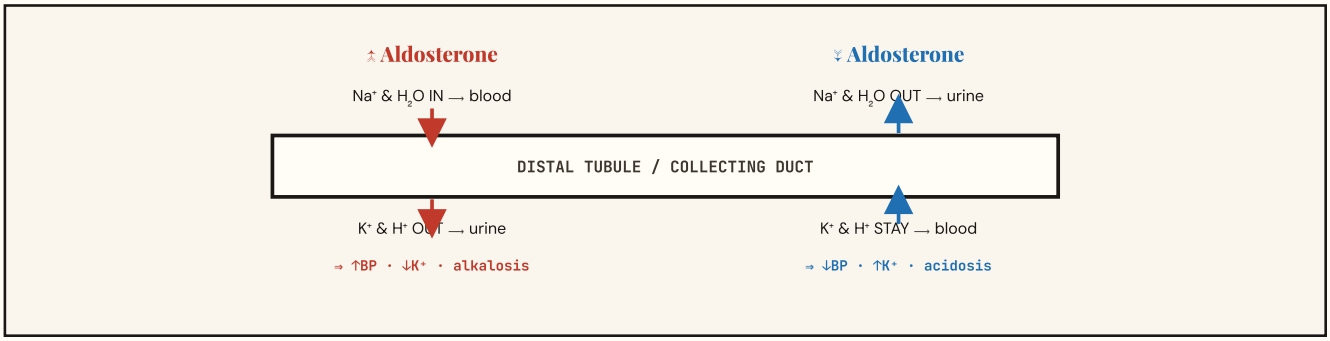
Aldosterone's job: **reabsorb Na^+ & H_2O · excrete K^+ & H^+.** Trace the chain reaction in each direction.

↑ TOO MUCH ALDOSTERONE

- ↑ **Aldosterone** at the kidney tubule
- ↑ **Na^+ & H_2O reabsorption** → hypervolemia → **HTN**
- ↑ **K^+ excretion** → **HYPOkalemia**
- ↑ **H^+ excretion** → **metabolic ALKALOSIS**

↓ TOO LITTLE ALDOSTERONE

- ↓ **Aldosterone** at the kidney tubule
- ↓ **Na^+ & H_2O reabsorption** → hypovolemia → **HYPOtension**
- ↓ **K^+ excretion** → **HYPERkalemia**
- ↓ **H^+ excretion** → **metabolic ACIDOSIS**



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Signs & Symptoms

Mirror images. Same axis, opposite poles.

↑ Hyperaldosteronism

- **BP:** **Hypertension** (often resistant), headache
- **HYPOKALEMIA:** muscle weakness, cramps, fatigue, paresthesias, polyuria & polydipsia (nephrogenic DI–like)
- **CARDIAC:** dysrhythmias, flattened T waves, **U waves**, ST depression
- **NEURO:** tetany, numbness/tingling (alkalosis lowers ionized Ca^{2+})
- **LABS:** ↑Na^+ (mild), **↓K^+**, ↑aldosterone, ↓renin (primary), metabolic alkalosis

↓ Hypoaldosteronism

- **BP:** **Hypotension**, orthostatic dizziness, dehydration, **salt craving**
- **HYPERKALEMIA:** muscle weakness, fatigue, abdominal cramping
- **CARDIAC:** dysrhythmias, **peaked T waves**, widened QRS → risk of arrest
- **VOLUME:** weight loss, tachycardia, poor turgor
- **LABS:** **↓Na^+**, **↑K^+**, ↓aldosterone, metabolic acidosis

RED FLAGS: HYPER → severe hypokalemia (K^+ < 2.5) with U waves = lethal dysrhythmia risk. | **HYPO →** hyperkalemia (peaked T waves / widening QRS) = impending cardiac arrest; sudden hypotension may signal **Addisonian (adrenal) crisis → shock**. Both are priority cardiac-monitoring situations.

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Priority Nursing Interventions

Filtered through **A · B · C → Safety → NCLEX prioritization.** Potassium and the cardiac monitor drive most "first action" answers here.

A/B Airway & Cardiac

- Continuous **ECG monitoring** — both disorders cause lethal dysrhythmias
- Treat the K^+ first: it is the most immediate life threat
- HYPO Watch for shock/crisis → ABCs, O_2, IV access

C Circulation / Fluids

- HYPER Monitor BP, daily weights, I&O; restrict Na^+
- HYPO Replace volume with **0.9% NaCl**; monitor for hypotension
- Trend electrolytes & ABGs every order/protocol

S Safety / Meds

- Fall precautions (weakness, orthostasis)
- Administer prescribed K^+ correction safely (never IV push KCl)
- Educate on med adherence & crisis prevention

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Pharmacology

The aldosterone **antagonist** (spironolactone) treats the excess; the aldosterone **replacement** (fludrocortisone) treats the deficit. Don't mix them up.

DRUG / CLASS	MECHANISM (SIMPLE)	KEY SIDE EFFECTS	NURSING CONSIDERATIONS
Spironolactone HYPER K⁺-sparing diuretic / aldosterone antagonist	Blocks aldosterone → excretes Na^+ & H_2O, **keeps K^+**. Lowers BP, corrects hypokalemia.	**Hyperkalemia**, gynecomastia, menstrual changes, dizziness	Monitor K^+ & BP; avoid salt substitutes (K^+-based) & other K^+ sources; teach signs of hyperkalemia.
Eplerenone HYPER Selective aldosterone antagonist	Same as spironolactone, more selective → fewer hormonal side effects.	Hyperkalemia, dizziness, fatigue	Same K^+ monitoring; useful when gynecomastia is a problem.
Antihypertensives HYPER	Adjuncts to control BP in primary/secondary disease.	Hypotension, varies by class	Monitor BP; surgery (adrenalectomy) is definitive Tx for an adenoma.
Fludrocortisone HYPO Synthetic mineralocorticoid	**Replaces aldosterone** → reabsorb Na^+ & H_2O, excrete K^+. Raises BP, lowers K^+.	Fluid overload, **HTN**, edema, **hypokalemia**, weight gain	Monitor BP, weight, edema, K^+; do **not** stop abruptly; teach signs of over/under-replacement.
Hydrocortisone HYPO Glucocorticoid (in Addison's)	Replaces cortisol; needed with fludrocortisone in full adrenal insufficiency.	Hyperglycemia, infection risk, GI upset	Stress-dosing for illness/surgery; never stop suddenly (crisis risk).

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NCLEX Test Traps

The exam loves to flip a single word. Watch these wrong-vs-right pairs.

✗ **Trap:** "Hyperaldosteronism causes hyperkalemia."

✓ **Right:** HYPER-aldo → **HYPO-kalemia** (K^+ is dumped). The prefixes are opposite for sodium-vs-potassium.

✗ **Trap:** "Give spironolactone for low aldosterone."

✓ **Right:** Spironolactone **blocks** aldosterone → used for HYPER. **Fludrocortisone** replaces it → used for HYPO.

✗ **Trap:** "Teach the patient on spironolactone to use a salt substitute."

✓ **Right:** Salt substitutes are **potassium-based** → cause hyperkalemia. **Avoid** them on K^+-sparing diuretics.

✗ **Trap:** "First action for peaked T waves is to recheck BP."

✓ **Right:** Peaked T waves = **hyperkalemia** (HYPO-aldo). Priority is the **cardiac threat** — monitor/treat K^+, not the BP cuff.

✗ **Trap:** "Hyperaldosteronism shows metabolic acidosis."

✓ **Right:** HYPER excretes H^+ → **alkalosis**. HYPO retains H^+ → **acidosis**. Acid follows the same path as potassium.

✗ **Trap:** "Stop fludrocortisone once BP normalizes."

✓ **Right:** Abrupt withdrawal risks **adrenal crisis**. It is replacement therapy — taper only under provider direction.

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Patient & Family Education

Teach to the direction of the imbalance — and to the medication's opposite risk.

↑ Hyperaldosteronism

- ✓ Take BP at home; report readings that stay high
- ✓ Limit dietary sodium; report swelling or sudden weight gain
- ✓ On spironolactone/eplerenone: **avoid** high-K^+ foods & salt substitutes; report muscle weakness or irregular heartbeat (hyperkalemia)
- ✓ Keep follow-up labs (K^+, aldosterone, renin)
- ✓ If surgery (adrenalectomy) is planned, understand pre/post care

↓ Hypoaldosteronism

- ✓ Do **not** skip or abruptly stop fludrocortisone/steroids
- ✓ Increase salt & fluids in heat, illness, or heavy sweating
- ✓ Report dizziness, fainting, persistent vomiting/diarrhea (crisis risk)
- ✓ Wear a **medical-alert bracelet**; carry emergency steroid info
- ✓ Know "sick-day rules" — illness/surgery may need a higher dose

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Memory Boosters

Lock in the direction with these patterns.

THE CORE RULE

Aldosterone = "Salt & water ON, potassium OFF"

More aldosterone → more Na^+/H_2O retained, more K^+ lost. Less aldosterone → reverse it all. Acid (H^+) always travels **with potassium**.

DIRECTION CHECK

HYPER = HI pressure, LOW K

HYPER-aldo: think **HI**gh BP + Conn's. **HYPO-aldo:** low BP + **HI**gh K^+ (Addison's spectrum). Sodium tracks the prefix; potassium does the opposite.

DRUG PAIRING

Spironolactone spares K^+

"Spiro-spares" potassium → it blocks aldosterone (treats HYPER, risk = hyperkalemia). **Fludro-FILLS** the missing aldosterone (treats HYPO).

ECG QUICK-TELL

U waves low · Peaked T high

U waves & flat T = **hypo**kalemia (HYPER-aldo). **Peaked, tented T** waves = **hyper**kalemia (HYPO-aldo). Read the T wave, name the K^+.

NCJMM Clinical Judgment Scenario

> *A 54-year-old client with resistant hypertension reports leg cramps and increased urination. Labs: Na^+ 146, **K^+ 2.7**, aldosterone elevated, renin suppressed. The cardiac monitor shows **U waves** and flattened T waves.*

RECOGNIZE CUES
Pattern = primary hyperaldosteronism
HTN + hypokalemia + ↑aldosterone/↓renin. U waves confirm low K^+.

ANALYZE / PRIORITIZE
K^+ 2.7 with ECG changes = top threat
Hypokalemia plus dysrhythmia risk outranks the BP itself right now.

TAKE ACTION
Cardiac monitor + safe K^+ replacement
Maintain ECG, replace K^+ per protocol (never IV push), start spironolactone as ordered.

GENERATE SOLUTIONS
Aldosterone antagonist + Na^+ restriction
Spironolactone/eplerenone to correct both BP and K^+; evaluate for adenoma/adrenalectomy.

EVALUATE
K^+ normalizing, ECG improving
T waves normalize, U waves resolve, BP trending down, cramps relieved.

SAFETY CHECK
Watch for the swing to hyperkalemia
On spironolactone, re-trend K^+ — over-correction can flip to hyperkalemia.

NGN NCLEX 2026 Visual Study Library — Endocrine / Fluid & Electrolyte module.
Content synthesized from Saunders, ATI, Lippincott & Endocrine Society guidelines. Verify against current institutional protocol.

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