

● NGN NCLEX 2026 • FLUID & ELECTROLYTE SERIES • CARDIAC PRIORITY

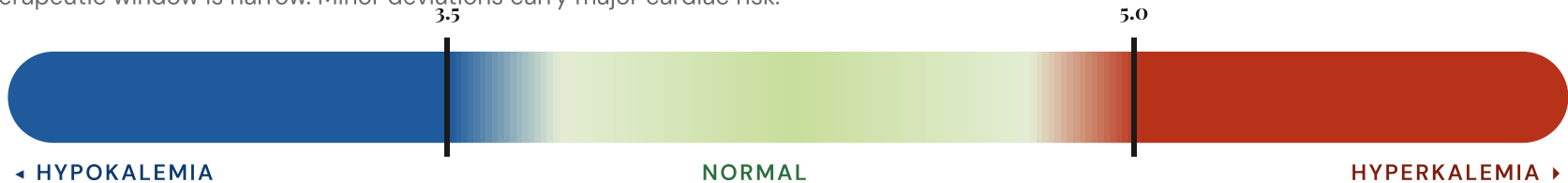
Hypokalemia versus Hyperkalemia

Potassium (K^+) is the most abundant *intracellular* cation — tiny serum shifts produce **lethal cardiac consequences**. This guide pairs pathophysiology, priority interventions, pharmacology, and NGN clinical judgment into one exam-ready reference.

📘 **System:** Fluid & Electrolyte / Cardiac ⚡ **Priority Level:** ABC — Cardiac 🧠 **NGN Framework:** NCJMM Integrated

Normal Serum Potassium: 3.5 – 5.0 mEq/L

The therapeutic window is narrow. Minor deviations carry major cardiac risk.



01 Definition & Overview

CORE CONCEPT

◀ LOW POTASSIUM

Hypokalemia

Serum K^+ < 3.5 mEq/L

Depletion of extracellular potassium disrupts nerve impulse conduction, muscle contraction, and cardiac repolarization. The heart becomes **hyperexcitable** — producing U waves, ectopy, and lethal dysrhythmias.

RED FLAG 🚩 Dangerously potentiates **digoxin toxicity**, even at therapeutic digoxin levels.

▶ HIGH POTASSIUM

Hyperkalemia

Serum K^+ > 5.0 mEq/L

Elevated extracellular potassium causes sustained depolarization of cardiac and muscle cells. Cells cannot re-polarize to fire again — the heart slows, widens, and ultimately **arrests**.

RED FLAG 🚩 Can progress to **ventricular fibrillation or asystole** — most lethal electrolyte imbalance.

02 Pathophysiology Simplified

MECHANISM

Hypokalemia → Hyperexcitable Cells

↓ Serum K⁺ (loss, shift, or low intake)



Resting membrane potential becomes **more negative** (hyperpolarized)



Delayed ventricular repolarization



ECG: **Flat T waves, U waves, ST depression**



Muscle weakness, paralytic ileus, ectopy, digoxin toxicity

Hyperkalemia → Cells Can't Re-fire

↑ Serum K⁺ (retention, shift out of cells, excess intake)



Resting membrane potential becomes **less negative** (partially depolarized)



Sodium channels inactivate — cells cannot re-depolarize



ECG: **Peaked T waves → wide QRS → loss of P wave**



Bradycardia → V-Fib → **Asystole**

ECG Rhythm Strip Comparison

Classic cardiac signatures of potassium imbalance.

Normal Sinus Rhythm



P → QRS → T | 60–100 bpm | PR < 0.20s

Hypokalemia



Flat T waves • U waves present • ST depression

Hyperkalemia



Tall, peaked T waves • Wide QRS • Loss of P wave

03 Causes & Risk Factors

RECOGNIZE CUES

What Causes Hypokalemia?

- **GI losses:** vomiting, diarrhea, NG suction, laxative abuse
- **Diuretics:** loop (furosemide), thiazide (HCTZ)
- **Steroids / Cushing's:** aldosterone excess
- **Alkalosis** (K^+ shifts into cells)
- **Insulin therapy / DKA treatment**
- **Inadequate intake** — NPO, anorexia, alcohol use disorder

🔑 MNEMONIC — "DITCH"

- D** Diuretics (loop/thiazide)
- I** Inadequate intake
- T** Too much insulin
- C** Cushing's / Corticosteroids
- H** Heavy fluid loss (vomit, diarrhea, NG)

What Causes Hyperkalemia?

- **Renal failure** (AKI or CKD) — #1 cause
- **K^+ -sparing diuretics:** spironolactone (Aldactone), triamterene
- **ACE inhibitors / ARBs**
- **Tissue breakdown:** burns, crush injury, rhabdomyolysis, hemolysis
- **Acidosis** (K^+ shifts out of cells)
- **Addison's disease** (aldosterone deficiency)
- **Salt substitutes** (contain KCl), excess K^+ supplements

🔑 MNEMONIC — "MACHINE"

- M** Medications (ACE, ARBs, NSAIDs)
- A** Acidosis (DKA)
- C** Cellular destruction (burns, crush)
- H** Hypoaldosteronism (Addison's)
- I** Intake (excess K^+ / salt substitutes)
- N** Nephrons failing (renal failure)
- E** Excretion impaired (K^+ -sparing diuretics)

04 Signs & Symptoms

ANALYZE CUES

HYPOKALEMIA — "EVERYTHING SLOWS & WEAKENS"

Clinical Manifestations

— CARDIOVASCULAR

- **Weak, thready pulse**; orthostatic hypotension
- Dysrhythmias: **PVCs, VT, bradycardia**
- **ECG: flat/inverted T, U waves, ST depression** 🚨
- ↑ sensitivity to digoxin → **digoxin toxicity**

— NEUROMUSCULAR

- Skeletal muscle weakness, leg cramps, fatigue
- **Hyporeflexia** (diminished DTRs)
- Paresthesias, lethargy, confusion

— GI & GU

- **Hypoactive bowel sounds** → **paralytic ileus**, constipation
- Nausea, vomiting, anorexia
- Polyuria, dilute urine, ↑ thirst

— RESPIRATORY

- **Shallow, weak respirations** 🚨 (diaphragm weakness → respiratory failure)

HYPERKALEMIA — "EVERYTHING TIGHTENS & THEN FAILS"

Clinical Manifestations

— CARDIOVASCULAR

- **Slow, irregular pulse**; hypotension
- Dysrhythmias: **bradycardia, V-fib, asystole** 🚨
- **ECG: tall peaked T waves, wide QRS, prolonged PR, loss of P**
- **Cardiac arrest** at $K^+ > 7.0$ mEq/L

— NEUROMUSCULAR

- **Early: twitching, cramps, hyperreflexia**
- **Late: muscle weakness** → **flaccid paralysis**
- Numbness/tingling of face, tongue, hands, feet

— GI & GU

- **Hyperactive bowel sounds**, diarrhea
- Nausea, abdominal cramping
- Oliguria (if renal failure is the cause)

— RESPIRATORY

- Profound muscle weakness → **respiratory failure** 🚨 (late sign)

05 Priority Nursing Interventions

TAKE ACTION

Hypokalemia — ABCs First

Replace K^+ · Protect the heart · Watch the diuretics

- 1 **Assess airway & respiratory status** — monitor rate, depth, and effort for shallow breathing.
- 2 **Place on continuous cardiac monitor;** obtain 12-lead ECG. Watch for U waves and ectopy.
- 3 **Hold digoxin** and notify HCP — verify digoxin level ($\uparrow$ risk of toxicity).
- 4 **Administer PO or IV K^+** as prescribed. PO preferred if alert, tolerating fluids.
- 5 **Verify urine output ≥ 30 mL/hr** BEFORE giving IV K^+ (to avoid causing hyperkalemia).
- 6 **Hold K^+ -wasting diuretics;** anticipate switch to K^+ -sparing (spironolactone).
- 7 **Monitor for paralytic ileus** — auscultate bowel sounds, hold PO if absent.
- 8 **Fall precautions** — muscle weakness increases risk.

Hyperkalemia — Stabilize the Heart FIRST

Stabilize · Shift · Eliminate

- 1 **Obtain 12-lead ECG & place on continuous cardiac monitor.**
Peaked T waves = emergency.
- 2 **STABILIZE myocardium:** administer IV **calcium gluconate** (cardioprotective — does not lower K^+).
- 3 **SHIFT K^+ into cells:** IV regular insulin + D50W; sodium bicarb (if acidotic); nebulized albuterol.
- 4 **ELIMINATE K^+ :** Kayexalate (PO/rectal), patiromer, loop diuretics, or **hemodialysis** (definitive in renal failure).
- 5 **Stop all sources of K^+** — IV fluids with K^+ , salt substitutes, supplements, ACE/ARBs, K^+ -sparing diuretics.
- 6 **Recheck serum K^+** 1–2 hours after each intervention; trend carefully.
- 7 **Prepare for dialysis** if anuric or $K^+ > 6.5$ with ECG changes.
- 8 **Monitor for hypoglycemia** after insulin/D50 (check blood glucose hourly).

Drug / Class	Mechanism	Key Side Effects	Nursing Considerations
Potassium Chloride (KCl) — PO	Replaces lost K ⁺	GI upset, nausea	Give with food & full glass of water; do not crush extended-release
Potassium Chloride (KCl) — IV	Rapid K ⁺ replacement	Phlebitis, vein burning, cardiac arrest if pushed	NEVER IV push. Always dilute; use pump; max 10 mEq/hr peripheral, 20 mEq/hr central
Spirolactone (Aldactone) — K⁺-sparing diuretic	Blocks aldosterone → retains K ⁺ , excretes Na/H ₂ O	Hyperkalemia, gynecomastia	Give with meals; avoid salt substitutes; monitor K ⁺
Calcium Gluconate IV	Stabilizes cardiac membrane — does NOT lower K ⁺	Hypercalcemia, bradycardia, extravasation	#1 priority when ECG changes present; give slowly via central line preferred
Insulin (Regular) + D50W IV	Shifts K ⁺ from serum into cells	Hypoglycemia	Check blood glucose hourly × 6; onset 15–30 min, lasts 4–6 hrs
Sodium Polystyrene Sulfonate (Kayexalate)	Exchanges Na ⁺ for K ⁺ in the GI tract → K ⁺ excreted in stool	Diarrhea, sodium retention, constipation, bowel necrosis (rare)	Must produce BM for it to work; assess bowel sounds; not for post-op ileus
Patiromer (Veltassa)	Binds K ⁺ in GI tract	Constipation, hypomagnesemia	Separate from other meds by 3 hours; for chronic hyperkalemia
Sodium Bicarbonate IV	Corrects acidosis → K ⁺ shifts back into cells	Metabolic alkalosis, fluid overload	Use when metabolic acidosis is present
Albuterol (nebulized)	Beta-2 agonist → drives K ⁺ into cells	Tachycardia, tremor	Adjunct therapy; monitor HR

Drug / Class	Mechanism	Key Side Effects	Nursing Considerations
Loop Diuretics (Furosemide)	Enhance renal K ⁺ excretion	Hypokalemia, hypotension, dehydration	Only effective if kidneys functioning; monitor I&O

⚠️ IV Potassium Chloride — Safety Rules You MUST Know

- ✗ **NEVER** administer IV push / IV bolus — will cause **cardiac arrest**
- ✗ **NEVER** give without dilution — always diluted in IV fluid
- ✗ **NEVER** infuse without an **infusion pump**
- ✓ **VERIFY** urine output ≥ 30 mL/hr before infusing
- ✓ **MAX rate:** 10 mEq/hr peripheral • 20 mEq/hr central line
- ✓ **MONITOR** IV site for phlebitis / extravasation; patient reports burning → slow rate
- ✓ **RECHECK** serum K⁺ after infusion to confirm correction

07 Complications

WHAT CAN GO WRONG

Hypokalemia Complications

- ❗ **Digoxin toxicity** — low K^+ makes digoxin bind more → lethal dysrhythmias even at therapeutic dose
- ❗ **Ventricular dysrhythmias** — PVCs, VT, torsades de pointes
- ❗ **Respiratory failure** — diaphragmatic weakness
- ❗ **Paralytic ileus** — bowel standstill, abdominal distention
- ❗ **Rhabdomyolysis** in severe cases
- ❗ **Metabolic alkalosis** (H^+ shifts into cells in exchange for K^+)

Hyperkalemia Complications

- ❗ **Cardiac arrest** — V-fib or asystole (most feared; $K^+ > 7.0$ mEq/L)
- ❗ **Progressive conduction blocks** — prolonged PR → loss of P → sine wave pattern
- ❗ **Flaccid paralysis** of skeletal muscles
- ❗ **Respiratory failure** — respiratory muscle paralysis
- ❗ **Metabolic acidosis** ($K^+ - H^+$ exchange reversed)
- ❗ **Dialysis dependence** if underlying renal cause unresolved

08 NCLEX Test Traps ⚠️

AVOID THESE ERRORS

✗ COMMON TRAP

Giving calcium gluconate to "lower" K^+ in hyperkalemia.



✓ CORRECT THINKING

Calcium gluconate **stabilizes the cardiac membrane** — it does NOT remove K^+ . Insulin/D50, Kayexalate, or dialysis does that.

✗ COMMON TRAP

Giving IV KCl by rapid push to correct hypokalemia faster.



✓ CORRECT THINKING

NEVER IV push potassium — always dilute, pump-controlled, max 10 mEq/hr peripheral. IV push = cardiac arrest.

✗ COMMON TRAP

Recommending a salt substitute to a client on lisinopril (ACE inhibitor).



✓ CORRECT THINKING

Salt substitutes contain **KCl**. Combined with ACE inhibitors → dangerous **hyperkalemia**.

✗ COMMON TRAP

Administering digoxin when K^+ is 3.0 mEq/L.



✓ CORRECT THINKING

HOLD digoxin and notify HCP. Hypokalemia potentiates digoxin → toxicity, lethal dysrhythmias.

✗ COMMON TRAP

Starting IV KCl on an oliguric client without checking urine output.



✓ CORRECT THINKING

Verify urine output ≥ 30 mL/hr FIRST. No urine = no K^+ excretion → rebound hyperkalemia.

✗ COMMON TRAP

Kayexalate given to a post-op ileus client.



✓ CORRECT THINKING

Kayexalate requires **bowel function** — assess bowel sounds. Without a BM, K^+ is not excreted and bowel necrosis can occur.

✗ COMMON TRAP

Confusing peaked T waves with T-wave inversion.



✓ CORRECT THINKING

Peaked (tall, tented) T = Hyperkalemia. **Flat/inverted T + U wave = Hypokalemia**.

09 NCJMM Clinical Judgment Scenario

NGN CASE STUDY

Case: 68-year-old with CKD on ACE Inhibitor

Scenario: A 68-year-old client with Stage 4 CKD is admitted with weakness, nausea, and palpitations. Home meds include lisinopril, spironolactone, and daily salt substitute. VS: HR 52, BP 108/62, RR 18, SpO₂ 95%. Labs: **K⁺ 6.8 mEq/L**, BUN 58, Creatinine 3.9. ECG shows **tall, peaked T waves and widening QRS**.

STEP 1

Recognize Cues

K⁺ 6.8 (critical high), peaked T waves, wide QRS, bradycardia, weakness, CKD + ACE + spironolactone + salt substitute (K⁺ stacking).

STEP 2

Analyze Cues

Life-threatening hyperkalemia with cardiac membrane instability. Renal failure prevents K⁺ excretion; all home meds contribute.

STEP 3

Prioritize / Take Action

① IV calcium gluconate (stabilize heart) → ② Insulin + D5O (shift) → ③ Hold ACE/spironolactone/salt sub → ④ Prepare dialysis → ⑤ Continuous ECG.

STEP 4

Evaluate Outcomes

K⁺ trends down, T waves normalize, QRS narrows, HR 60–90, no dysrhythmia. Glucose stable after insulin. Client education begun on K⁺-restricted diet.

10 Patient & Family Education

NUTRITION & TEACHING

⬆️ INCREASE — High-Potassium Foods

Encourage in hypokalemia; restrict in hyperkalemia



Bananas



Avocado



Oranges / juice



Tomatoes



Potatoes



Sweet potato



Spinach



Beans / lentils



Cantaloupe



Nuts / seeds



Raisins / dried fruit



Salmon / cod

⬇️ DECREASE — Low-Potassium Choices

Encourage in hyperkalemia (CKD, K-sparing meds)



Apples / applesauce



Berries



Pears



White bread / pasta



White rice



Cucumbers



Carrots (cooked)



Onions



Eggs



Chicken (small portion)



Plain tea



NO salt substitute

Hypokalemia Teaching

- ☒ Take PO K⁺ supplement with **food and full glass of water** to reduce GI upset.
- ☒ Do NOT crush or chew extended-release potassium tablets.
- ☒ Eat potassium-rich foods daily if on a loop or thiazide diuretic.
- ☒ Report muscle weakness, cramps, palpitations, dizziness, constipation.
- ☒ If taking digoxin, learn to count pulse daily & report pulse < 60 or visual changes (yellow/green halos).
- ☒ Avoid laxative overuse; manage vomiting/diarrhea promptly.
- ☒ Keep follow-up labs for serum K⁺ as ordered.

Hyperkalemia Teaching

- ☒ **AVOID salt substitutes** — most contain potassium chloride.
- ☒ Follow a low-potassium diet; learn to read food labels for "K" content.
- ☒ Do not take OTC NSAIDs (ibuprofen, naproxen) without HCP approval if on ACE/ARB.
- ☒ Report muscle weakness, numbness, tingling, irregular or slow pulse, palpitations.
- ☒ Take K⁺-sparing diuretics (spironolactone) exactly as prescribed — never extra doses.
- ☒ Keep all lab appointments — routine K⁺ and renal function checks are essential.
- ☒ Never stop ACE inhibitors abruptly; report symptoms and call HCP first.

11 Memory Boosters & Mnemonics

LOCK IT IN

Hypokalemia — "6 L's"

EVERYTHING IS LOW & LIMP

- L** **Lethargy** — confusion, fatigue
- L** **Leg cramps** — weakness
- L** **Limp muscles** — hyporeflexia
- L** **Low / shallow respirations**
- L** **Lethal dysrhythmias** — U waves, PVCs
- L** **Lots of urine** — polyuria

Hyperkalemia — "MURDER"

BECAUSE IT CAN KILL

- M** **Muscle** cramps / weakness
- U** **Urine** — oliguria (renal failure)
- R** **Respiratory** failure (late)
- D** **Decreased** cardiac contractility
- E** **ECG changes** — peaked T, wide QRS
- R** **Reflexes** hyperactive → flaccid paralysis

Quick Pattern Associations

HYPO = Slow & Low

Slow bowels (ileus), slow reflexes, low BP, low respirations, **low & flat T waves**, U waves present.

HYPER = Tight Then Dead

Early cramps/twitching/diarrhea → late flaccid paralysis & asystole.
Peaked T waves are the alarm.

Treatment Formula — Hyperkalemia

C-S-E: Calcium (stabilize) → Shift (insulin, bicarb, albuterol) → Eliminate (Kayexalate, diuretics, dialysis).

Link the Lab — Dig + K

Low K^+ + Digoxin = Toxicity. See hypokalemia on labs → **always check the digoxin level and hold the dose.**

