CLINICALJUDGE™

ELECTROLYTE, RENAL & CKD PHARMACOLOGY

Daily Review Print Edition · 2026 NGN NCLEX-RN Test Plan

NCJMM Clinical Judgment Framework · 14 High-Yield Drugs · 16 Sections Each

14 High-Yield Drugs	**16** Sections Per Drug	**14** NGN Questions
100% Answers Revealed	**2026** Test Plan Aligned	**NCJMM** Framework

Critical / Danger · Pathophysiology · Safe / Expected · Complications / Hold · NCLEX Traps · Medications / Antidote · Patient Education

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DRUG 01 / 14 · 2026 NGN NCLEX-RN

⚡ POTASSIUM CHLORIDE

KCl · Electrolyte Replacement

HIGH RISK | SAFETY ALERT | EMERGENCY

Client Need: Pharmacological & Parenteral Therapies

MECHANISM

Replaces intracellular potassium essential for cardiac conduction, nerve impulse transmission, and cellular membrane potential maintenance. Primary intracellular cation; maintains resting membrane potential.

INDICATION

Hypokalemia (K^+ < `3.5 mEq/L`), prevention in patients on loop/thiazide diuretics, DKA management with insulin, metabolic alkalosis correction.

MUST-ASSESS BEFORE GIVING

- ⚠ Serum K^+ baseline before every dose
- ⚠ Renal function (BUN, Cr, GFR) — kidneys MUST excrete K^+
- ⚠ Urine output ≥ `30 mL/hr` — MANDATORY before IV
- ⚠ Cardiac rhythm / ECG baseline
- ⚠ Serum Mg^{2+} (hypoMg → refractory hypokalemia)
- ⚠ Digoxin use (hypoK^+ potentiates toxicity)
- ⚠ ACE inhibitors, ARBs, K^+-sparing diuretics (additive)
- ⚠ IV: NEVER give push or undiluted — FATAL

KEY LABS & MONITORING

- Serum K^+ — normal `3.5-5.0 mEq/L`
- Continuous cardiac telemetry during IV
- Serum Mg^{2+} (low Mg → refractory hypoK^+)
- Serum pH (alkalosis shifts K^+ intracellularly)
- Urine output every 1–2 hrs
- Repeat K^+ 1–2 hrs post-replacement

MOST DANGEROUS ADVERSE EFFECT

⚠ CARDIAC ARREST from hyperkalemia if given IV push or too rapidly. IV infiltration → severe tissue necrosis & compartment syndrome. Pattern: Peaked T-waves → Wide QRS → Sine wave → VFib → Asystole.

COMMON SIDE EFFECTS

GI irritation, nausea, vomiting, diarrhea (oral); burning/pain at IV site; phlebitis with peripheral infusion; metallic taste.

HOLD / NOTIFY PROVIDER

- ⚠ HOLD if K^+ ≥ `5.0 mEq/L`
- ⚠ HOLD if urine output < `30 mL/hr`
- ⚠ HOLD for any ECG change — STAT notify
- ⚠ Max peripheral rate: `10 mEq/hr` | Central: `20 mEq/hr`
- ⚠ Max peripheral concentration: 40 mEq/100 mL

ANTIDOTE / REVERSAL — FOR KCL-INDUCED HYPERKALEMIA

① Calcium gluconate (cardiac membrane stabilization) → ② Sodium bicarbonate / Insulin + D50W (shift K^+ intracellularly) → ③ Kayexalate / Patiromer / SZC (remove K^+) → ④ Hemodialysis (definitive)

PATIENT TEACHING

- Take oral KCl with full glass water and FOOD
- Do NOT crush extended-release tablets (Klor-Con)
- Report: muscle weakness, irregular heartbeat, tingling
- AVOID salt substitutes (contain KCl — doubles K^+)
- K^+-rich foods: bananas, oranges, potatoes, avocados

NURSING ACTION

- ✓ NEVER IV push — FATAL
- ✓ Always use infusion pump
- ✓ Verify urine output before every dose
- ✓ Monitor IV site hourly for infiltration/phlebitis
- ✓ Recheck K^+ 1–2 hrs post-replacement

NGN CLINICAL JUDGMENT CUE

Patient receiving IV KCl at 20 mEq/hr on peripheral IV develops peaked T-waves and widened QRS on telemetry. Urine output over the past 2 hours = 15 mL total.

NGN PRIORITY QUESTION

A nurse prepares to administer IV KCl 40 mEq in 1,000 mL NS at 10 mEq/hr. K^+ = 2.8 mEq/L. Which finding is MOST important to address BEFORE starting the infusion?

A) The patient reports nausea and does not want to eat

B) ✓ Urine output over the past 2 hours is 28 mL total

C) Patient is on telemetry showing normal sinus rhythm

D) Serum Mg^{2+} is 1.2 mEq/L

✓B **Urine output 28 mL/2 hrs = 14 mL/hr — well below the 30 mL/hr threshold.** K^+ is excreted renally; oliguria means K^+ will accumulate → hyperkalemia → cardiac arrest. This is a hard stop before any IV KCl. A: nausea doesn't contraindicate IV KCl. C: normal ECG expected, not a barrier. D: low Mg^{2+} worsens hypokalemia but is not a safety contraindication to starting IV KCl.

🧠 **TIP:** KCl = KIDNEY FIRST. Rule #1 on NCLEX: urine output ≥ 30 mL/hr before every IV potassium dose. NEVER IV push. NEVER undiluted. KCl + oliguria = stop sign. Max peripheral rate: 10 mEq/hr.

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HIGH RISK | SAFETY ALERT | EMERGENCY

CALCIUM GLUCONATE

Ca-Gluconate · Calcium Salt / Electrolyte / Antidote

MECHANISM

Replaces ionized calcium essential for neuromuscular excitability, cardiac action potential, and bone integrity. As antidote in hyperkalemia: directly stabilizes cardiac cell membrane by restoring threshold potential — does NOT lower K^+.

INDICATION

Hypocalcemia (symptomatic/severe), cardiac membrane stabilization in hyperkalemia (FIRST action), reversal of $MgSO_4$ toxicity, post-thyroidectomy/parathyroidectomy hypocalcemia, hypoparathyroidism.

MUST-ASSESS BEFORE GIVING

- Total Ca^{2+} normal `8.5-10.5 mg/dL` ; ionized `4.5-5.1 mg/dL`
- Correct for albumin: Ca + 0.8 × (4 - albumin)
- ECG: hypocalcemia prolongs QT → torsades risk
- DIGOXIN use — calcium potentiates toxicity (caution!)
- Serum Mg^{2+} (low Mg worsens hypocalcemia)
- IV site patency (extravasation = severe necrosis)
- Chvostek's and Trousseau's signs

KEY LABS & MONITORING

- Serum total + ionized Ca^{2+}
- Serum phosphorus (inverse relationship)
- Serum albumin (corrected Ca^{2+} calculation)
- Serum Mg^{2+} (must correct Mg to correct Ca)
- ECG — prolonged QT, Osborn waves (severe)
- PTH level (parathyroid function)

MOST DANGEROUS ADVERSE EFFECT

⚠ CARDIAC ARREST if infused too rapidly (bradycardia → asystole). Potentiates digoxin toxicity (AV block, VFib). Severe tissue necrosis with extravasation. Hypercalcemia with overdose.

COMMON SIDE EFFECTS

Hypotension, bradycardia, warmth/flushing during IV, metallic taste, constipation (oral), nausea. Slowing the infusion typically resolves warmth/flushing.

HOLD / NOTIFY PROVIDER

- HOLD if Ca^{2+} > `10.5 mg/dL`
- STOP if bradycardia or severe hypotension
- CAUTION with digoxin — notify provider
- Max rate: `0.5 mEq/min IV`
- Do NOT mix with $NaHCO_3$ or phosphate (precipitate)

ANTIDOTE FOR HYPERCALCEMIA

IV NS hydration + furosemide (renal Ca^{2+} excretion) → calcitonin (rapid) → bisphosphonates (pamidronate — sustained) → hemodialysis (severe refractory cases)

PATIENT TEACHING

- Report perioral tingling, muscle cramps, numbness
- Signs of hypercalcemia: constipation, fatigue, confusion, excessive thirst, kidney stones
- Oral Ca^{2+}: take WITH food; separate from iron by 2 hrs
- Post-thyroidectomy: muscle twitching expected if Ca drops

NURSING ACTION

- ✓ KEEP AT BEDSIDE for all $MgSO_4$ drips & post-thyroidectomy
- ✓ Max IV rate: 0.5 mEq/min — use infusion pump
- ✓ Warm solution to body temperature
- ✓ Monitor IV site continuously for extravasation
- ✓ Check Chvostek's and Trousseau's signs

NGN CLINICAL JUDGMENT CUE

Post-thyroidectomy patient day 1: facial twitching when cheek is tapped (Chvostek's+), bilateral hand cramping, perioral tingling. Ionized Ca^{2+} = 3.8 mg/dL.

NGN PRIORITY QUESTION

A patient with ESRD presents with K^+ = 7.1 mEq/L, peaked T-waves, and widening QRS. Multiple medications are ordered. Which does the nurse administer FIRST?

A) Sodium polystyrene sulfonate (Kayexalate) orally

B) Regular insulin 10 units IV with D50W

C) ✓ **Calcium gluconate 1 g IV over 10 minutes**

D) Sodium bicarbonate 50 mEq IV push

✓C **Calcium gluconate stabilizes the cardiac membrane within 1–3 minutes** — the immediate threat is fatal dysrhythmia. It does NOT lower K^+ but protects the heart while other measures work. A: Kayexalate removes K^+ but takes hours — never first in cardiac emergency. B: Insulin/D50W shifts K^+ intracellularly (20–30 min) — comes after cardiac stabilization. D: $NaHCO_3$ also shifts K^+ but is slower. Sequence: **Stabilize → Shift → Remove.**

🧠 **TIP:** Hyperkalemia emergency = C-S-R: Calcium first (Cardiac stabilization) → Shift K^+ (insulin/bicarb) → Remove K^+ (Kayexalate/dialysis). Peaked T-waves + high K^+ → Calcium gluconate is ALWAYS the first answer. "C for Cardiac, C for First."

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HIGH RISK SAFETY ALERT

SODIUM PHOSPHATE

$NaPO_4$ · Phosphate Electrolyte / Osmotic Laxative

MECHANISM

IV: replenishes phosphate — essential for ATP synthesis, 2,3-DPG (O_2 delivery), bone mineralization, and cellular metabolism. As enema/laxative: osmotic action draws water into colon. CONTRAINDICATED in CKD (cannot excrete phosphate).

INDICATION

Hypophosphatemia (PO_4 < `2.5 mg/dL`), bowel preparation for colonoscopy, constipation relief. Use with extreme caution in CKD — risk of acute phosphate nephropathy and severe hypocalcemia.

MUST-ASSESS BEFORE GIVING

- ⚠ Serum Ca^{2+} — INVERSE relationship; phosphate drops Ca^{2+}
- ⚠ Renal function (GFR) — AVOID if GFR < `30 mL/min`
- ⚠ Serum Na^+ (significant sodium load → edema)
- ⚠ Fluid and cardiac status
- ⚠ Do NOT run phosphate + calcium in same IV line (precipitate)

KEY LABS

- Serum PO_4 — normal `2.5-4.5 mg/dL`
- Serum Ca^{2+} — will drop as phosphate rises
- Serum Na^+ (hypernatremia risk)
- BUN, Cr, GFR (CKD contraindication)
- ECG if severe hypocalcemia suspected (↑ QT)

MOST DANGEROUS ADVERSE EFFECT

⚠ Severe HYPOCALCEMIA → tetany, laryngospasm, cardiac arrest. Acute phosphate nephropathy (enema overuse in CKD/elderly). Hyperphosphatemia in CKD → vascular calcification. Hypernatremia from sodium load.

COMMON SIDE EFFECTS

Nausea, vomiting, diarrhea, cramping (enema), abdominal bloating, electrolyte imbalances, dehydration with enema overuse.

HOLD / NOTIFY PROVIDER

- ⚠ HOLD if Ca^{2+} < `8.5 mg/dL` before IV phosphate
- ⚠ HOLD if GFR < `30 mL/min`
- ⚠ NEVER in same IV line as calcium (precipitate)
- ⚠ Notify if tetany: Chvostek's+, carpopedal spasm
- ⚠ Fleet enema: max ONE per 24 hrs; avoid in CKD/CHF/elderly

ANTIDOTE / MANAGEMENT

Calcium gluconate IV for phosphate-induced hypocalcemia. Dialysis for severe hyperphosphatemia. Supportive care for acute phosphate nephropathy (no direct antidote).

PATIENT TEACHING

- Never use more than one fleet enema in 24 hours
- Drink plenty of fluids with oral phosphate
- Report: muscle cramps, tingling, difficulty breathing
- CKD patients: AVOID sodium phosphate enemas — ask provider for alternative bowel prep

NURSING ACTION

- ✓ Infuse IV via pump — slow rate per protocol
- ✓ NEVER same line as calcium (fatal precipitate)
- ✓ Monitor Ca^{2+} during and after IV phosphate
- ✓ Question fleet enema orders in CKD/CHF/elderly
- ✓ Assess Trousseau's and Chvostek's signs

NGN CLINICAL JUDGMENT CUE

Elderly patient with unknown CKD stage 4 used two fleet phosphate enemas for colonoscopy prep. Post-procedure: carpopedal spasm, facial twitching, extreme anxiety, confusion. Ca^{2+} = 6.4 mg/dL.

NGN PRIORITY QUESTION

A nurse prepares to administer IV sodium phosphate to a patient with PO_4 = 1.4 mg/dL. Which finding is the PRIORITY CONCERN before administration?

A) Serum potassium 3.8 mEq/L

B) Serum sodium 142 mEq/L

C) ✓ Serum calcium 7.0 mg/dL

D) Serum phosphate 1.4 mg/dL

✓C **Ca^{2+} 7.0 mg/dL is already below normal (8.5–10.5 mg/dL).** Administering IV phosphate will drop calcium further (inverse relationship) → tetany, laryngospasm, cardiac arrest. Provider must be notified; calcium likely needs correction first. A & B: normal values, no concern. D: confirms need for replacement but is not a safety contraindication by itself.

🧠 **TIP:** PO_4 ↑ = Ca^{2+} ↓. Always check calcium BEFORE phosphate. NEVER run phosphate and calcium in the same IV line. Fleet enema = DANGEROUS in CKD/elderly. Think: "Phosphate UP = Calcium DOWN."

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HIGH RISK | SAFETY ALERT | EMERGENCY

MAGNESIUM SULFATE

$MgSO_4$ · Electrolyte Replacement / Anticonvulsant

MECHANISM

Replaces magnesium — cofactor for 300+ enzymatic reactions. As anticonvulsant: blocks neuromuscular transmission and decreases CNS excitability by competing with calcium at nerve terminals. Vasodilates and inhibits platelet aggregation.

INDICATION

Hypomagnesemia, eclampsia/preeclampsia seizure prevention (DRUG OF CHOICE), torsades de pointes, refractory hypokalemia/hypocalcemia (must correct Mg first), alcohol withdrawal, pre-term labor tocolysis.

MUST-ASSESS BEFORE EVERY DOSE

- Serum Mg^{2+} — normal `1.5-2.5 mEq/L`
- Respiratory rate — MUST be ≥ `12/min`
- Deep tendon reflexes (patellar) — absent = early toxicity
- Urine output ≥ `25-30 mL/hr` (renal excretion)
- Level of consciousness, blood pressure, heart rate
- OB: fetal heart rate, cervical dilation, uterine activity

KEY LABS — TOXICITY LEVELS

- Therapeutic eclampsia: `4-7 mEq/L`
- Loss of DTRs: `7-10 mEq/L`
- Respiratory paralysis: `10-13 mEq/L`
- Cardiac arrest: `≥15 mEq/L`
- Serum Ca^{2+} (hyperMg → hypocalcemia)
- BUN, Cr (impaired renal function → Mg accumulation)

MOST DANGEROUS ADVERSE EFFECT

⚠ RESPIRATORY ARREST (Mg >10–13 mEq/L). Cardiac arrest (Mg ≥15 mEq/L). Profound hypotension. Neonatal Mg toxicity — respiratory depression in neonate at delivery. Neuromuscular blockade.

COMMON SIDE EFFECTS

Flushing, diaphoresis, warmth/heat sensation, nausea, headache, drowsiness, decreased DTRs (expected at therapeutic levels for eclampsia), hypotension. Warmth/flushing during loading dose is normal.

HOLD / NOTIFY — NON-NEGOTIABLE

- HOLD if RR < `12/min` — respiratory arrest imminent
- HOLD if patellar DTRs ABSENT — early toxicity
- HOLD if urine output < `25-30 mL/hr`
- HOLD if Mg^{2+} > `7 mEq/L` — notify provider
- ALWAYS use infusion pump — NEVER gravity drip

ANTIDOTE — MUST BE AT BEDSIDE AT ALL TIMES

CALCIUM GLUCONATE 1 g IV push — direct antidote for $MgSO_4$ toxicity. Directly antagonizes magnesium at neuromuscular junction. Also: mechanical ventilation if respiratory arrest occurs. Dialysis for extreme toxicity with renal failure.

PATIENT TEACHING

- Flushing and drowsiness during infusion are expected
- REPORT: difficulty breathing, chest tightness immediately
- Baby will be closely monitored — Mg crosses placenta
- Reflexes checked frequently for your safety

NURSING ACTION

- ✓ ALWAYS use infusion pump
- ✓ Calcium gluconate 1 g at bedside — ALWAYS
- ✓ Patellar DTRs every 1–2 hrs (absent → STOP infusion)
- ✓ RR continuously — minimum every 15–30 min
- ✓ Keep airway, suction, crash cart accessible

NGN CLINICAL JUDGMENT CUE

Preeclamptic patient on $MgSO_4$: RR = 9/min, no patellar reflex elicited, patient difficult to rouse, confused. Serum Mg^{2+} = 10.2 mEq/L.

NGN PRIORITY QUESTION

A preeclamptic patient on $MgSO_4$ infusion: RR = 9/min, absent patellar reflexes, BP 150/96, Mg^{2+} = 10 mEq/L. What is the nurse's PRIORITY action?

A) Decrease infusion rate by 50% and recheck in 30 minutes

B) ✓ Stop infusion and administer calcium gluconate 1 g IV immediately

C) Place patient in Trendelenburg and increase IV fluid rate

D) Apply oxygen at 10 L/min via non-rebreather mask

✓B **STOP infusion + calcium gluconate IMMEDIATELY.** RR <12 + absent DTRs + Mg^{2+} = 10 = active toxicity requiring emergency reversal. A: insufficient — drug must be stopped, not slowed. C: Trendelenburg is for hypotension/shock, not appropriate here. D: oxygen is supportive only — does not reverse Mg toxicity. If breathing fails, mechanical ventilation will be needed.

🧠 **TIP:** $MgSO_4$ toxicity = 3 CHECKS before each dose: RR ≥ 12, DTRs present, urine output ≥ 30 mL/hr. Antidote = Calcium gluconate (the CALCIUM blocks magnesium). "DTRs disappear BEFORE breathing stops" — early warning system. Ca gluconate MUST be at bedside.

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COMMONLY TESTED | TEACHING-HEAVY | SAFETY ALERT

SEVELAMER

Renagel / Renvela · Non-Calcium Phosphate Binder (Polymer)

MECHANISM

Non-absorbed cationic polymer binds dietary phosphate in the GI tract via ionic/hydrogen bonding → prevents phosphate absorption. Excreted with phosphate in feces. Secondary benefit: reduces LDL. Does NOT add calcium load → preferred when Ca^{2+} is elevated.

INDICATION

Hyperphosphatemia in CKD stage 4–5 and ESRD (dialysis patients). Preferred over calcium-based binders when Ca^{2+} elevated or $Ca \times PO_4$ >55. Prevents secondary hyperparathyroidism, renal osteodystrophy, and vascular calcification.

MUST-ASSESS BEFORE GIVING

- Serum PO_4 — dialysis goal `3.5–5.5 mg/dL`
- Serum Ca^{2+} (non-calcium binder — less hypercalcemia risk)
- $Ca \times PO_4$ product — keep < `55 mg²/dL²`
- GI history — bowel obstruction is ABSOLUTE contraindication
- Swallowing ability — tablets are large
- Medication list — binds other drugs (thyroid, quinolones, cyclosporine)

KEY LABS

- Serum PO_4 (monthly in stable dialysis pts)
- Serum Ca^{2+} (less hypercalcemia risk vs. Ca^{2+}-based binders)
- $Ca \times PO_4$ product (goal <55)
- Intact PTH (secondary hyperparathyroidism marker)
- LDL (secondary benefit — sevelamer HCl lowers LDL)

MOST DANGEROUS ADVERSE EFFECT

⚠ Bowel obstruction/perforation (constipation → obstruction). Metabolic acidosis (Renagel = HCl form worsens pre-existing CKD acidosis; prefer Renvela carbonate form in acidotic patients).

COMMON SIDE EFFECTS

Constipation, nausea, vomiting, dyspepsia, flatulence, diarrhea. GI symptoms are the most common reason for non-adherence in dialysis patients.

HOLD / NOTIFY PROVIDER

- HOLD if absent bowel sounds or suspected bowel obstruction
- Notify if no BM for >3–4 days
- Notify if severe abdominal pain or distension
- Separate from other medications by 1–3 hours (binding interaction)

PATIENT TEACHING

- TAKE WITH OR IMMEDIATELY AFTER MEALS — must bind dietary PO_4
- Do NOT crush, chew, or open tablets — destroys polymer mechanism
- Strict phosphate-restricted diet: limit dairy, processed foods, cola, nuts
- Separate from all other medications by 1–3 hours
- Report: constipation, severe abdominal pain, no stool for days

NURSING ACTION

- ✓ Administer WITH meals — NOT between meals
- ✓ NEVER crush or chew tablets
- ✓ Monitor bowel function and GI symptoms regularly
- ✓ Educate on dietary phosphate sources
- ✓ Review medication interactions (levothyroxine, cyclosporine)

NGN CLINICAL JUDGMENT CUE

Dialysis patient on sevelamer 800 mg TID with meals. Reports no bowel movement in 6 days, abdominal distension, nausea/vomiting. Abdomen rigid on palpation. Last X-ray shows colonic dilation.

NGN PRIORITY QUESTION

A nurse educates a dialysis patient newly prescribed sevelamer (Renvela). Which patient statement indicates a need for FURTHER TEACHING?

A) "I will take this pill right after breakfast, lunch, and dinner."

B) "I should cut back on cheese, milk, and processed foods."

C) ✓ "I can crush this tablet and mix it with applesauce if it's hard to swallow."

D) "I need to wait a few hours before taking my other medications."

✓C **Sevelamer CANNOT be crushed.** Crushing destroys the cross-linked polymer structure that allows phosphate binding. The entire mechanism depends on the intact tablet. Renvela powder packets are available for patients with swallowing difficulty. A, B, D are all correct statements — no teaching needed.

🧠 **TIP:** Sevelamer = MEAL-TIME binder. Rules: WITH FOOD, NEVER CRUSH, SEPARATE from other meds 1–3 hours. Preferred in ESRD when Ca^{2+} is elevated. Renvela (carbonate) preferred in acidosis vs. Renagel (HCl). Bowel obstruction = serious risk with constipation.

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COMMONLY TESTED | SAFETY ALERT | TEACHING-HEAVY

💊 CALCIUM CARBONATE (AS PHOSPHATE BINDER)

Tums / Os-Cal · Calcium-Based Phosphate Binder / Antacid

⚙️ MECHANISM

$CaCO_3$ binds dietary phosphate in the GI tract (requires gastric acid) → forms insoluble Ca-phosphate complex excreted in feces. Also neutralizes gastric acid (antacid role). Provides supplemental calcium. Requires acid for dissolution — reduced efficacy with PPIs/H2 blockers.

🏥 INDICATION

Hyperphosphatemia in CKD, calcium supplementation, GI antacid use. Used with caution in dialysis — significant hypercalcemia risk, especially when combined with calcitriol or other active vitamin D analogs.

⚠️ MUST-ASSESS BEFORE GIVING

- ⚠ Serum Ca^{2+} — HYPERCALCEMIA risk (esp. with calcitriol)
- ⚠ Ca×PO_4 product — keep < `55 mg²/dL²`
- ⚠ Total calcium from ALL sources (diet + supplements + antacid use)
- ⚠ Vitamin D analog use (calcitriol dramatically increases Ca^{2+} absorption)
- ⚠ Gastric acid status (requires acid for dissolution — PPIs reduce efficacy)
- ⚠ Separate from iron by 2 hrs; levothyroxine by 4 hrs

🔬 KEY LABS

- Serum Ca^{2+} — normal `8.5-10.5 mg/dL` ; HOLD if >10.5
- Serum PO_4
- Ca×PO_4 product (goal <55)
- 25-OH Vitamin D
- Intact PTH
- BUN, Cr (CKD staging)

💀 MOST DANGEROUS ADVERSE EFFECT

⚠ HYPERCALCEMIA → vascular calcification, metastatic calcification, cardiac dysrhythmias, renal calculi. Milk-alkali syndrome (hypercalcemia + metabolic alkalosis + renal insufficiency). Ca^{2+} carbonate + calcitriol = HIGH-RISK combo for dangerous hypercalcemia.

😖 COMMON SIDE EFFECTS

Constipation, GI upset, belching (CO_2 release as antacid), flatulence, hypercalcemia with chronic use. "Bones, Stones, Groans, Moans" = hypercalcemia symptoms.

🛑 HOLD / NOTIFY PROVIDER

- ⚠ HOLD if Ca^{2+} > `10.5 mg/dL`
- ⚠ HOLD if Ca×PO_4 > 55
- ⚠ "Bones, Stones, Groans, Moans" = hypercalcemia → NOTIFY
- ⚠ NOTIFY if on calcitriol AND Ca^{2+} rising
- ⚠ SEPARATE from iron (2 hrs), levothyroxine (4 hrs)

💊 ANTIDOTE FOR HYPERCALCEMIA

IV NS hydration + furosemide (forces renal Ca^{2+} excretion) → calcitonin (rapid) → bisphosphonates (pamidronate — sustained) → corticosteroids (if granulomatous disease) → hemodialysis (severe)

📚 PATIENT TEACHING — KEY TIMING DIFFERENCE

- As PHOSPHATE BINDER: take WITH MEALS (to bind dietary PO_4)
- As ANTACID ONLY: take BETWEEN meals (different from phosphate binder use!)
- Report: constipation, extreme fatigue, confusion, excessive thirst, kidney stones
- Limit calcium from ALL sources — too much causes buildup
- Separate from iron by 2 hrs; avoid excessive antacid use

🩺 NURSING ACTION

- ✓ Monitor Ca^{2+} frequently in dialysis patients on calcitriol
- ✓ Know the DUAL role: PO_4 binder (WITH food) vs. antacid (BETWEEN food)
- ✓ Requires gastric acid — less effective with PPIs (consider sevelamer instead)
- ✓ Monitor for milk-alkali syndrome in heavy users

🔍 NGN CLINICAL JUDGMENT CUE

Dialysis patient on calcium carbonate 1,500 mg TID with meals + calcitriol 0.5 mcg daily. Presents with 3-day confusion, fatigue, constipation, muscle weakness. Ca^{2+} = 12.4 mg/dL.

📝 NGN PRIORITY QUESTION

A CKD stage 5 patient receives calcium carbonate and calcitriol. Which assessment finding requires the nurse's IMMEDIATE action?

A) Serum phosphate 5.8 mg/dL

B) ✓ **Serum calcium 12.1 mg/dL with confusion and muscle weakness**

C) Intact PTH of 290 pg/mL

D) Serum bicarbonate 20 mEq/L

✓B **Severe hypercalcemia (12.1 mg/dL) with neurological symptoms = emergency.** Calcium carbonate + calcitriol synergistically raises calcium → vascular calcification, dysrhythmias, neurological dysfunction. Hold both medications and notify provider STAT. A: elevated PO_4 needs adjustment, not emergency. C: elevated PTH = chronic CKD finding, not emergency. D: bicarb 20 = mild metabolic acidosis, not emergency.

🧠 **TIP:** $CaCO_3$ + calcitriol = HYPERCALCEMIA TRAP. "Bones, Stones, Groans, Moans" = hypercalcemia mnemonic. KEY NCLEX trick: PO_4 binder timing (WITH meals) vs. antacid timing (BETWEEN meals) — same drug, different timing, different role!

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HIGH RISK | SAFETY ALERT | COMMONLY TESTED

SODIUM POLYSTYRENE SULFONATE

SPS / Kayexalate · Cation Exchange Resin / Potassium-Lowering Agent

MECHANISM

Polystyrene resin exchanges Na^+ ions for K^+ ions in the colon. Each gram binds ~0.5–1.0 mEq K^+. Potassium-resin complex excreted in feces. Works in distal colon. SLOW onset (several hours to days). NOT for cardiac emergency use.

INDICATION

Chronic or subacute hyperkalemia (K^+ > 5.5 mEq/L) in CKD/ESRD patients. NOT for emergency cardiac hyperkalemia. Newer agents (patiromer, SZC) now preferred in many guidelines due to lower GI complication risk.

MUST-ASSESS BEFORE GIVING

- ⚠ Bowel function history — CONTRAINDICATED in reduced gut motility, bowel obstruction
- ⚠ Recent GI surgery — HIGH RISK colonic necrosis (avoid within 7 days post-op)
- ⚠ Serum Na^+ — SPS adds Na^+ → sodium overload → edema/HTN
- ⚠ Sorbitol content — avoid sorbitol mixture (↑ colonic necrosis risk)
- ⚠ Cardiac monitoring — confirm no active ECG changes first
- ⚠ NOT for patient with cardiac symptoms from hyperkalemia

KEY LABS

- Serum K^+ (repeat every 2–4 hrs)
- Serum Na^+ (hypernatremia risk)
- BMP (full panel)
- ECG (if symptomatic hyperkalemia → escalate first)
- Abdominal assessment: bowel sounds, distension

MOST DANGEROUS ADVERSE EFFECT

⚠ COLONIC NECROSIS AND PERFORATION — most feared complication (with sorbitol + post-op patients with reduced gut motility). Hypokalemia with overtreatment. Hypernatremia. Bowel obstruction from constipation.

COMMON SIDE EFFECTS

Nausea, vomiting, constipation, diarrhea (with sorbitol), hypokalemia with prolonged use, hypernatremia, anorexia.

HOLD / NOTIFY PROVIDER

- ⚠ HOLD if bowel sounds absent or reduced
- ⚠ HOLD within 7 days of abdominal surgery
- ⚠ HOLD in post-op ileus or bowel obstruction
- ⚠ NOTIFY if K^+ < `3.5 mEq/L` (overtreatment)
- ⚠ NOTIFY if severe abdominal pain, bloody stool (necrosis)

PATIENT TEACHING

- For retention enema: retain for 30–60 minutes
- Report: severe abdominal pain, bloody stool, no BM
- Takes several hours to work — NOT for emergency use
- Follow low-potassium diet

NURSING ACTION

- ✓ NOT appropriate for emergency cardiac hyperkalemia — too SLOW
- ✓ Assess bowel sounds before every dose
- ✓ AVOID in post-surgical or low-motility patients
- ✓ Maintain continuous cardiac monitoring
- ✓ Document stool output (K^+ eliminated via stool)

NGN CLINICAL JUDGMENT CUE

ESRD patient post colon resection day 4. Provider orders Kayexalate for K^+ = 6.0 mEq/L. Patient reports mild abdominal pain. Bowel sounds hypoactive. This is a contraindication to Kayexalate.

NGN PRIORITY QUESTION

A patient with ESRD has K^+ = 6.4 mEq/L with peaked T-waves on the cardiac monitor. The provider orders sodium polystyrene sulfonate. What is the nurse's PRIORITY action?

A) Administer Kayexalate immediately — K^+ is critically high

B) ✓ Hold Kayexalate; contact provider; request calcium gluconate order first

C) Administer via retention enema with sorbitol for fastest effect

D) Check urine output and administer if adequate

✓B **Peaked T-waves = cardiac emergency. Kayexalate is NOT an emergency drug.** The cardiac membrane must be stabilized first (calcium gluconate), then K^+ shifted (insulin + D50W), then removed (Kayexalate). C: sorbitol + SPS = increased colonic necrosis risk. D: urine output is relevant for KCl, not Kayexalate — and delays cardiac protection.

🧠 **TIP:** Kayexalate = SLOW (hours). NEVER first in cardiac emergency. Sequence: Calcium gluconate → Insulin/D50W → Kayexalate → Dialysis. Avoid post-op / low-motility patients — colonic necrosis risk. Newer agents (patiromer, SZC) now preferred.

DRUG 08 / 14 · 2026 NGN NCLEX-RN

NEW PATIROMER

HIGH RISK · SAFETY ALERT · COMMONLY TESTED

Veltassa · Novel Potassium-Lowering Agent / Cation Exchange Polymer

MECHANISM

Non-absorbed polymer selectively binds free K^+ in exchange for Ca^{2+} in the DISTAL COLON. K^+-bound patiromer excreted in feces. Does NOT bind sodium. Onset: 7–48 hours. NOT for emergency use. Works distally — not throughout GI tract (unlike SZC).

INDICATION

Chronic hyperkalemia in CKD, especially while maintaining RAAS inhibitor therapy (ACE inhibitors/ARBs cause hyperkalemia but must continue for CKD/cardiac protection). Allows continuation of life-saving RAAS therapy.

MUST-ASSESS BEFORE GIVING

- ⚠ Serum K^+ baseline — do NOT use for K^+ > 6.5 mEq/L with cardiac symptoms
- ⚠ Serum Ca^{2+} — exchanges K^+ for Ca^{2+} → hypercalcemia risk
- ⚠ Serum Mg^{2+} — lowers magnesium over time → hypomagnesemia
- ⚠ GI motility — contraindicated in severe constipation/obstruction
- ⚠ ALL other oral meds must be separated by ≥ `3 hours`

KEY LABS

- Serum K^+ — at baseline, 1 week, then monthly
- Serum Ca^{2+} (hypercalcemia — exchange ion is calcium)
- Serum Mg^{2+} (hypomagnesemia risk with prolonged use)
- BMP (full panel)

MOST DANGEROUS ADVERSE EFFECT

⚠ HYPOKALEMIA — excessive K^+ binding if not monitored. HYPOMAGNESEMIA (can be severe). Drug interactions from binding other oral medications (reduced bioavailability of critical drugs like levothyroxine, antibiotics). Bowel obstruction.

COMMON SIDE EFFECTS

Constipation, hypomagnesemia, hypokalemia (overuse), nausea, diarrhea, flatulence. Hypomagnesemia: muscle weakness, tremors, cardiac arrhythmias.

HOLD / NOTIFY PROVIDER

- ⚠ HOLD if K^+ < `3.5 mEq/L`
- ⚠ HOLD if suspected bowel obstruction
- ⚠ ALL other oral meds: minimum `3-hour separation`
- ⚠ NOTIFY if Mg^{2+} < `1.2 mEq/L`
- ⚠ NOT for emergency hyperkalemia

PATIENT TEACHING

- Mix powder in ~85 mL water — stir and drink immediately
- Take at SAME TIME daily WITH or after meals
- WAIT AT LEAST 3 HOURS after patiromer before any other oral medication
- Refrigerate; room temperature stable up to 3 months
- Report: muscle weakness, irregular heartbeat, cramping
- Does NOT work in emergencies — takes at least 7 hours

NURSING ACTION

- ✓ Mix with water only — do not heat
- ✓ Separate ALL other oral meds by 3 hours — most common error
- ✓ Monitor K^+ weekly × 1 month, then monthly
- ✓ Monitor Mg^{2+} and Ca^{2+} during treatment
- ✓ NOT for acute/emergency hyperkalemia

NGN CLINICAL JUDGMENT CUE

CKD patient on patiromer + lisinopril develops profound muscle weakness, cramping, ECG showing flattened T-waves and U-waves. K^+ = 2.8 mEq/L. Patient admits doubling patiromer dose because "my K^+ was really high last week."

NGN PRIORITY QUESTION

A nurse teaches a patient prescribed patiromer who also takes levothyroxine, metoprolol, and furosemide. Which instruction is MOST critical?

A) "Take patiromer on an empty stomach for maximum binding."

B) "Take all medications at the same time each morning for convenience."

C) ✓ "Wait at least 3 hours after patiromer before taking your other medications."

D) "You can take a double dose if your potassium feels high this week."

✓C **The 3-hour separation rule is the most critical safety teaching.** Patiromer binds other meds in the GI tract — taking levothyroxine with patiromer causes hypothyroidism relapse; metoprolol absorption is reduced. A: should be taken WITH food. B: taking all meds together with patiromer causes dangerous binding interactions. D: doubling the dose causes severe hypokalemia and cardiac dysrhythmias.

🧠 **TIP:** Patiromer = 3-HOUR RULE. Exchanges K^+ for Ca^{2+} (not sodium — unlike SZC). Monitor Mg^{2+} (unique risk). NOT for emergencies. "PATI-romer = PATIent needs to WAIT 3 hours." New drug = new NCLEX questions.

DRUG 09 / 14 · 2026 NGN NCLEX-RN

HIGH RISK | SAFETY ALERT | COMMONLY TESTED

NEW SODIUM ZIRCONIUM CYCLOSILICATE

SZC / Lokelma · Novel Potassium-Lowering Agent (Ion Trap)

MECHANISM

Crystalline zirconium silicate — highly selective potassium ION TRAP throughout the ENTIRE GI tract (not just colon). Captures K^+ in exchange for Na^+ and H^+ ions. FASTER onset than patiromer (effect within 1 hour). Excreted in feces. Each dose delivers a meaningful SODIUM LOAD.

INDICATION

Hyperkalemia — acute/subacute AND chronic management. Can be used in urgent non-emergency hospital settings. Approved for CKD and heart failure patients (with caution re: sodium load in CHF).

MUST-ASSESS BEFORE GIVING

- ⚠ Serum K^+ — if >6.5 with cardiac symptoms, escalate to emergency first
- ⚠ Serum Na^+ — SZC exchanges K^+ for SODIUM → sodium load
- ⚠ Fluid status and daily weight — CRITICAL in CHF/CKD patients
- ⚠ Cardiac history — CHF patients at high risk of fluid overload
- ⚠ All other oral meds — separate by 2 hours

KEY LABS

- Serum K^+ — check within 1–2 hrs of first hospital dose
- Serum Na^+ — hypernatremia risk from sodium exchange
- Daily weight — fluid retention indicator
- BMP (full panel)
- Lung sounds, leg edema (fluid overload signs)

MOST DANGEROUS ADVERSE EFFECT

⚠ FLUID OVERLOAD in CHF/CKD — sodium exchange adds sodium load → worsens heart failure → pulmonary edema. HYPOKALEMIA from excessive K^+ removal. HYPERNATREMIA. This is the critical difference from patiromer (which uses Ca^{2+} exchange, not Na^+).

COMMON SIDE EFFECTS

Edema (lower extremity swelling), hypokalemia, nausea, constipation, diarrhea. Edema is the most clinically significant in CHF patients.

HOLD / NOTIFY PROVIDER

- ⚠ HOLD if K^+ < `3.5 mEq/L`
- ⚠ HOLD if weight gain > `2 lbs/day` or worsening dyspnea
- ⚠ HOLD if severe edema or pulmonary congestion develops
- ⚠ Separate from other oral meds by 2 hours
- ⚠ NOTIFY if Na^+ rising (hypernatremia trend)

PATIENT TEACHING

- Mix powder in 45 mL water — drink IMMEDIATELY, do not store
- Take at SAME TIME daily; morning with meal recommended
- Weigh EVERY MORNING — report gain > 2–3 lbs/day
- Report leg swelling, shortness of breath, difficulty breathing
- Separate from other meds by 2 hours

NURSING ACTION

- ✓ Monitor daily weight and fluid status — especially CHF patients
- ✓ Check K^+ within 1–2 hrs of first hospital dose
- ✓ Assess for edema: leg swelling, lung sounds, dyspnea
- ✓ Separate other oral meds by 2 hours
- ✓ Watch serum Na^+ for hypernatremia trend

NGN CLINICAL JUDGMENT CUE

CHF + CKD patient started on SZC for K^+ = 5.8 mEq/L. Day 3: weight gain 6 lbs, bilateral pitting edema to knees, crackles bilaterally in lung bases, SpO_2 89%, BP 162/98 mmHg. Pulmonary edema from SZC sodium load.

NGN PRIORITY QUESTION

A CHF + CKD patient receives sodium zirconium cyclosilicate (Lokelma) for hyperkalemia. Which finding requires the MOST urgent nursing action?

A) K^+ dropped from 5.8 to 4.2 mEq/L after 2 doses

B) Serum Na^+ = 141 mEq/L after day 3 of therapy

C) ✓ **Weight gain of 6 lbs in 3 days with worsening dyspnea and bilateral crackles**

D) Patient reports mild nausea after taking the morning dose

✓C **Acute CHF decompensation from SZC sodium load — life-threatening emergency.** SZC exchanges K^+ for Na^+. In CHF + CKD patients who cannot excrete extra Na^+, fluid accumulates → pulmonary edema. 6 lb gain + dyspnea + crackles = emergency. A: good K^+ response — no concern. B: Na^+ 141 = normal. D: minor expected side effect.

🧠 **TIP:** SZC = SODIUM LOAD → FLUID RETENTION. SZC vs. patiromer: SZC exchanges K^+ for Na^+ (CHF risk), patiromer exchanges K^+ for Ca^{2+} (hypercalcemia risk). SZC works FASTER (1 hr). CHF + SZC + weight gain + dyspnea = EMERGENCY. "Lokelma LOCKS K^+ OUT but LOADS sodium IN."

DRUG 10 / 14 · 2026 NGN NCLEX-RN

HIGH RISK | SAFETY ALERT | COMMONLY TESTED

EPOETIN ALFA

Epogen / Procrit · Erythropoiesis-Stimulating Agent (ESA)

MECHANISM

Recombinant human erythropoietin. Binds EPO receptors on erythroid progenitor cells in bone marrow → stimulates proliferation and differentiation into mature RBCs. Requires adequate iron, folate, and B12. Also stimulates platelet production.

INDICATION

Anemia of CKD/ESRD, chemotherapy-induced anemia (non-curative cancer only), anemia in HIV/zidovudine therapy, reducing allogeneic transfusions pre-surgery. AVOID in active curative-intent cancer treatment.

MUST-ASSESS BEFORE GIVING

- Hgb — do NOT start if ≥ `10 g/dL` ; titrate to `10–12 g/dL`
- Iron stores: TSAT >20%, ferritin >100 ng/mL (iron = fuel for RBC)
- Blood pressure — HYPERTENSION is major risk; HOLD if uncontrolled
- Thrombotic history (DVT, PE, stroke, MI) — ESAs ↑ clot risk significantly
- Active malignancy with curative intent — AVOID (tumor promotion)
- Renal function — IV for dialysis; SQ for outpatient

KEY LABS

- Hgb — every 1–2 weeks during adjustment; monthly stable
- Hematocrit — dialysis target `30–36%`
- Ferritin (goal > `100 ng/mL`)
- TSAT: transferrin saturation (goal > `20%`)
- Reticulocyte count (rises 5–10 days post-initiation)
- BP before EVERY dose

MOST DANGEROUS ADVERSE EFFECT

⚠ THROMBOSIS: DVT, PE, stroke, MI, AV fistula clotting in dialysis patients. HYPERTENSIVE CRISIS (especially with rapid Hgb rise). Pure Red Cell Aplasia (PRCA) — rare antibody-mediated; ESA becomes ineffective. Tumor progression.

COMMON SIDE EFFECTS

Hypertension, headache, arthralgias, flu-like symptoms (fever, myalgia), nausea, fatigue, injection site reactions. Headache + HTN = warning signs of excessive Hgb rise.

HOLD / NOTIFY PROVIDER

- HOLD if Hgb ≥ `12 g/dL`
- HOLD if Hgb rises > `1 g/dL in 2-week period` (reduce dose 25%)
- HOLD if uncontrolled HTN (BP persistently >140/90)
- NOTIFY for any thrombosis signs (leg pain/swelling, chest pain, neuro changes)
- NEVER use in curative-intent cancer chemotherapy

PATIENT TEACHING

- Do NOT shake vial — destroys the protein; swirl gently
- Refrigerate; do NOT freeze
- Check BP at home regularly; report if >140/90
- REPORT IMMEDIATELY: chest pain, leg pain/swelling, vision changes, severe headache
- Take iron, folate, B12 as prescribed — epoetin cannot work without iron
- Takes 2–6 weeks to see improvement — do not stop

NURSING ACTION

- ✓ SQ (outpatient) or IV during dialysis (ESRD)
- ✓ NEVER shake vial — gentle swirl only
- ✓ Check Hgb BEFORE each dose — DO NOT give if ≥ 12 g/dL
- ✓ Ensure iron supplementation is ongoing (no iron = no response)
- ✓ Monitor BP before every injection

NGN CLINICAL JUDGMENT CUE

Dialysis patient on epoetin alfa × 8 weeks. Labs: Hgb 13.8 g/dL, BP 176/102 mmHg. Patient reports severe throbbing headache and visual disturbances. Scheduled for today's injection.

NGN PRIORITY QUESTION

A dialysis patient's labs: Hgb 13.6 g/dL, K^+ 4.9, BP 174/98, TSAT 24%, ferritin 180 ng/mL. Which is the nurse's PRIORITY action?

A) Administer the scheduled dose since iron stores are adequate

B) ✓ **Withhold the dose and notify the provider about Hgb and blood pressure**

C) Reduce the dose by half and monitor Hgb in 1 week

D) Administer ferrous sulfate before giving epoetin alfa to maximize effect

✓B **Hgb 13.6 > 12 g/dL = HOLD and notify.** Continuing epoetin when Hgb exceeds the cap significantly increases thrombotic risk (stroke, MI, DVT, AV fistula occlusion). Concurrent HTN (174/98) further elevates cardiovascular risk. A: adequate iron stores do not override the Hgb threshold. C: nurse cannot independently adjust the dose. D: iron stores already adequate — not the priority.

🧠 **TIP:** Epoetin = HGB CAP AT 12. If Hgb >12 or rises >1 g/dL in 2 weeks: HOLD → CALL. Clot risk is danger #1. Never shake vial. Check BP every dose. No iron = no response. "ESA = Every Serum [Hgb] Assessed before Administration."

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HIGH RISK | SAFETY ALERT | COMMONLY TESTED

DARBEPOETIN ALFA

Aranesp · Long-Acting Erythropoiesis-Stimulating Agent (ESA)

MECHANISM VS. EPOETIN

Same mechanism as epoetin alfa (stimulates EPO receptors → erythropoiesis), but additional carbohydrate chains (glycosylation) extend half-life to ~25 hrs (vs. ~8 hrs for epoetin). This allows weekly or biweekly dosing instead of 3×/week. Same risks, less frequent administration.

INDICATION

Anemia of CKD/ESRD, chemotherapy-induced anemia (non-curative only). Same indications as epoetin alfa. Preferred when less frequent dosing improves patient adherence. CRITICAL: doses are NOT equivalent to epoetin (different units/conversion).

MUST-ASSESS — SAME AS EPOETIN

- Hgb — do NOT start if ≥ `10 g/dL` ; titrate to `10–12 g/dL`
- Blood pressure — HOLD if uncontrolled HTN
- Iron stores: TSAT >20%, ferritin >100 ng/mL
- Thrombotic history (same clot risk as epoetin)
- IMPORTANT: Doses NOT interchangeable with epoetin 1:1 — different units
- Active malignancy with curative intent — AVOID

KEY LABS

- Hgb every 2–4 weeks during dose adjustment; monthly stable
- TSAT and ferritin (ongoing iron monitoring)
- BP at every dose
- Reticulocyte count (bone marrow response)
- Hematocrit

MOST DANGEROUS ADVERSE EFFECT

⚠ THROMBOSIS (DVT, stroke, MI, AV fistula clotting) — same risk as epoetin. HYPERTENSIVE CRISIS with rapid Hgb rise. Pure Red Cell Aplasia (PRCA). Tumor progression in active cancer. "Worst headache of life" + HTN + Hgb >12 = emergency.

HOLD / NOTIFY PROVIDER

- HOLD if Hgb ≥ `12 g/dL` — same threshold as epoetin
- HOLD if Hgb rises > `1 g/dL in 2 weeks` (reduce 25%)
- HOLD if uncontrolled HTN
- NOTIFY for any signs of thrombosis
- Do NOT switch from epoetin without dose conversion table

COMMON SIDE EFFECTS

Hypertension, headache, edema, arthralgias, fatigue, injection site pain, flu-like symptoms. Less frequent dosing may improve tolerance. Same side effect profile as epoetin.

PATIENT TEACHING

- Given LESS OFTEN than epoetin — weekly or biweekly (not 3×/week)
- Do NOT assume missed dose — less frequent is intentional
- Do NOT shake vial; refrigerate, do not freeze
- Monitor BP weekly; report if >140/90
- Continue iron, B12, folate supplements — darbepoetin does NOT supply iron
- Report: chest pain, leg swelling, sudden weakness, blurred vision (clot signs)

NURSING ACTION

- ✓ SQ or IV — do NOT shake vial
- ✓ Check Hgb BEFORE every dose — HOLD if ≥ 12 g/dL
- ✓ Monitor BP before each injection
- ✓ Educate patient about LONGER interval (may think they missed a dose)
- ✓ Coordinate dose conversion protocol if switching from epoetin

NGN CLINICAL JUDGMENT CUE

Home health patient on darbepoetin every 2 weeks. Latest visit: Hgb 14.2 g/dL, BP 178/104 mmHg. Patient reports "worst headache ever" and blurring of vision. Scheduled for darbepoetin injection today — do NOT administer.

NGN PRIORITY QUESTION

A nurse educates a patient transitioning from epoetin alfa 3×/week to darbepoetin alfa. Which nurse statement is MOST accurate?

A) "You'll receive the same number of units as your epoetin dose."

B) "Darbepoetin works faster — your blood count will improve sooner."

C) ✓ "Darbepoetin lasts longer, so you'll need fewer injections per week."

D) "You can stop iron supplements since darbepoetin supplies iron directly."

✓C **Darbepoetin has ~3× longer half-life due to increased glycosylation → weekly or biweekly dosing vs. 3×/week for epoetin.** A: Doses are NOT equivalent — darbepoetin uses mcg/kg vs. IU for epoetin; 1:1 substitution is a medication error. B: Onset is similar — darbepoetin does NOT work faster. D: Both drugs require iron; neither supplies iron. Stopping supplements causes treatment failure.

🧠 **TIP:** Darbepoetin = LONG-ACTING EPO. Same cap (Hgb <12), same clot risk, same monitoring. Key difference: less frequent dosing. NEVER assume dose = epoetin dose. "Darb = Durable (longer half-life)." All ESA safety rules apply: no shaking, monitor BP, check Hgb first.

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COMMONLY TESTED | TEACHING-HEAVY | SAFETY ALERT

FERROUS SULFATE

$FeSO_4$ · Iron Supplement / Hematologic Agent

MECHANISM

Provides elemental iron (Fe^{2+}) required for hemoglobin synthesis (heme ring), myoglobin, and cellular respiration enzymes. Absorbed in duodenum/proximal jejunum via DMT-1 transporters. Ascorbic acid (Vitamin C) converts $Fe^{3+} \rightarrow Fe^{2+}$ for enhanced absorption. Gastric acid enhances dissolution.

INDICATION

Iron-deficiency anemia, anemia of CKD (adjunct to ESA therapy — ESA cannot work without iron), iron deficiency in pregnancy (increased demand), dietary iron deficiency. First-line for non-acute iron deficiency.

MUST-ASSESS BEFORE GIVING

- Hgb, Hct, MCV (microcytic <80 fL), MCHC (hypochromic)
- Serum ferritin, TSAT, serum iron, TIBC (confirm iron deficiency)
- GI history — ulcer disease (gastric irritation)
- Medication list — critical interactions (antacids, Ca^{2+}, dairy, thyroid, quinolones)
- Pediatric access — FATAL in children; store locked away

KEY LABS

- Hgb/Hct — improvement expected in 3–4 weeks
- Serum ferritin — women `12–150 ng/mL` ; men `12–300 ng/mL`
- TSAT — goal > `20%`
- MCV — microcytic; normalizes as iron repletes
- Reticulocyte count — rises 5–10 days post-initiation (confirms response)

MOST DANGEROUS ADVERSE EFFECT

⚠ IRON TOXICITY/OVERDOSE — especially in CHILDREN: GI hemorrhage, metabolic acidosis, hepatic necrosis, cardiovascular collapse. #1 cause of pediatric poisoning death. Parenteral iron: anaphylaxis risk. Antidote: DEFEROXAMINE (chelating agent; urine turns vin rosé = drug working).

COMMON SIDE EFFECTS

Constipation (most common), nausea, vomiting, dark/black tarry stools (EXPECTED — not melena), abdominal cramping, metallic taste, tooth staining (liquid form), diarrhea.

HOLD / NOTIFY PROVIDER

- HOLD if suspected toxicity (severe vomiting, bloody diarrhea, lethargy in child)
- NOTIFY if true melena suspected (guaiac test to differentiate from iron staining)
- NOTIFY if Hgb not improving after 4–6 weeks (reassess diagnosis)
- SEPARATE: antacids (2 hrs), dairy/Ca^{2+} (2 hrs), quinolones (2 hrs), levothyroxine (4 hrs)

ANTIDOTE FOR IRON TOXICITY

DEFEROXAMINE (Desferal) — iron chelating agent. Binds free iron; excreted renally. Urine turns reddish-brown/pink ("vin rosé color") — confirms deferoxamine is working. IV or IM in severe poisoning. Supportive care: GI decontamination if early ingestion.

PATIENT TEACHING

- Take on EMPTY STOMACH for best absorption (if GI intolerant, take with small amount of food)
- Take with VITAMIN C / orange juice — enhances absorption
- AVOID: milk/dairy, coffee, tea, antacids, calcium — significantly reduce absorption
- Stools will be DARK GREEN TO BLACK — EXPECTED and NORMAL — do not be alarmed
- Constipation common — increase water and fiber intake
- KEEP OUT OF REACH OF CHILDREN — tablets FATAL even in small amounts

NURSING ACTION

- ✓ Administer between meals (empty stomach) when possible
- ✓ Coordinate timing with all other medications (separation rules)
- ✓ Educate on expected dark stools vs. concerning melena
- ✓ Monitor Hgb, ferritin, reticulocyte count
- ✓ For parenteral iron: monitor for anaphylaxis; resuscitation equipment available

- Liquid form: use straw, rinse mouth (prevents tooth staining)
- Continue for 3–6 months AFTER Hgb normalizes — do NOT stop early

🔍 NGN CLINICAL JUDGMENT CUE

2-year-old brought to ED — parent found child with open ferrous sulfate bottle; child may have ingested 15–20 tablets. Child is vomiting, lethargic, pale, with bloody emesis. Iron toxicity emergency — antidote: deferoxamine.

📝 NGN PRIORITY QUESTION

A nurse provides discharge teaching for a patient with iron-deficiency anemia newly prescribed ferrous sulfate. Which PRIORITY instruction must the nurse emphasize?

A) "Take your iron tablet with milk to coat your stomach and reduce nausea."

B) ✓ **"Your stools may become dark or black — this is expected and not a sign of bleeding."**

C) "Once your energy returns, it's safe to stop taking the iron supplement."

D) "Take your antacid at the same time as iron to prevent stomach upset."

✓B **Dark/black stools are EXPECTED with iron therapy — patients must understand this to avoid alarm and premature discontinuation.** A: Milk/dairy INHIBITS iron absorption — never with milk. C: Iron stores must be repleted for 3–6 months after Hgb normalizes — stopping early causes relapse. D: Antacids significantly REDUCE iron absorption — must be separated by 1–2 hours.

🧠 **TIP:** Iron = VITAMIN C enhances, DAIRY BLOCKS. "Dark stools = EXPECTED" is a classic correct NCLEX answer. Antidote = DEFEROXAMINE (vin rosé/pink urine = working). Pediatric iron overdose = #1 pediatric poisoning death. Continue 3–6 months after Hgb normalizes — do NOT stop early.

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COMMONLY TESTED | SAFETY ALERT | TEACHING-HEAVY

⚡ ALLOPURINOL

Zyloprim · Xanthine Oxidase Inhibitor / Antigout / Uricosuric

MECHANISM

Inhibits XANTHINE OXIDASE — enzyme converting hypoxanthine → xanthine → uric acid. Reduces uric acid PRODUCTION.
CRITICAL: Xanthine oxidase ALSO metabolizes azathioprine and 6-MP → allopurinol inhibits their breakdown → 4–5× drug level increase → FATAL bone marrow suppression.

INDICATION

Chronic GOUT PREVENTION (NOT acute attacks), hyperuricemia from CKD or chemotherapy, tumor lysis syndrome (TLS) prevention, uric acid kidney stones, Lesch-Nyhan syndrome.
Start BETWEEN gout attacks — never during.

MUST-ASSESS BEFORE GIVING

- Serum uric acid — normal `2.4-6.0 mg/dL` ; men `3.4-7.0 mg/dL`
- Renal function (GFR) — DOSE REDUCTION required in CKD
- Liver function (LFTs) — hepatotoxicity risk
- Azathioprine or 6-MP use — POTENTIALLY FATAL INTERACTION → notify provider BEFORE first dose
- HLA-B*58:01 genotype (Asian patients) — very high SJS/TEN risk
- Prior allopurinol rash — contraindication to re-challenge

KEY LABS

- Serum uric acid — treatment goal < `6.0 mg/dL`
- BUN, Cr, GFR — renal dose adjustment (standard 300 mg/day → reduce in CKD)
- LFTs (hepatotoxicity monitoring)
- CBC (bone marrow suppression risk with azathioprine/6-MP)
- CK (myopathy — especially with statin combination)

MOST DANGEROUS ADVERSE EFFECTS

⚠ STEVENS-JOHNSON SYNDROME (SJS) / TOXIC EPIDERMAL NECROLYSIS (TEN) — life-threatening mucocutaneous reaction. ANY rash = STOP the drug immediately. Bone marrow suppression (agranulocytosis, aplastic anemia) with azathioprine/6-MP. Allopurinol hypersensitivity syndrome (multi-organ). Asian patients at extreme risk.

COMMON SIDE EFFECTS

Skin rash (STOP DRUG — first sign of SJS), GI upset, headache, drowsiness. Paradoxical GOUT FLARES during initiation (uric acid crystal mobilization) — expected; do NOT stop the drug; colchicine often co-prescribed.

HOLD / NOTIFY PROVIDER

- STOP IMMEDIATELY at FIRST SIGN of any rash — may be SJS
- HOLD if taking azathioprine or 6-MP — NOTIFY provider BEFORE first dose
- DOSE REDUCE if GFR < `30 mL/min`
- NOTIFY if: oral ulcers, blistering, eye changes, target lesions (SJS progression)

PATIENT TEACHING

- Start BETWEEN gout attacks — NEVER during an acute attack
- More gout attacks in first weeks = NORMAL (crystal mobilization) — do NOT stop
- Drink 2–3 liters of water per day (dilutes uric acid)
- Avoid: alcohol, organ meats, anchovies, sardines, shellfish, red meat
- STOP IMMEDIATELY + GO TO ER if ANY rash develops with blistering, mouth sores, eye changes
- Take with food to reduce GI upset
- This PREVENTS gout — does NOT treat acute attacks

NURSING ACTION

- ✓ NEVER start during acute gout attack
- ✓ Medication reconciliation — identify azathioprine or 6-MP BEFORE ordering
- ✓ Teach rash recognition at every encounter (SJS can develop months into therapy)
- ✓ Ensure adequate hydration (2–3 L/day)
- ✓ Asian patients: discuss HLA-B*58:01 testing with provider

NGN CLINICAL JUDGMENT CUE

Patient on allopurinol × 3 weeks: painful oral ulcers, bilateral eye redness (conjunctivitis), spreading rash with central blisters and purpuric centers on trunk and extremities. Temperature 38.9°C. = Evolving Stevens-Johnson Syndrome — STOP DRUG IMMEDIATELY.

NGN PRIORITY QUESTION

A nurse reconciles medications for a gout patient prescribed allopurinol. The patient also takes azathioprine (Imuran) for Crohn's disease. What is the nurse's MOST appropriate action?

A) Administer both medications as prescribed at standard doses

B) Hold allopurinol only; administer azathioprine as usual and monitor CBC

C) ✓ **Contact the provider immediately BEFORE administering allopurinol**

D) Reduce allopurinol dose by 50% and monitor uric acid levels

✓C **DANGEROUS drug interaction — provider must re-evaluate the entire regimen BEFORE the first dose.** Allopurinol inhibits xanthine oxidase → cannot break down azathioprine/6-MP → 4–5× plasma level increase → potentially fatal bone marrow suppression. If both are needed, azathioprine must be reduced to 25–33% under close monitoring. A: potentially fatal. B: insufficient — regimen must be re-evaluated. D: reducing allopurinol alone does not eliminate the interaction.

🧠 **TIP:** ALLOPURINOL + AZATHIOPRINE / 6-MP = DEADLY INTERACTION. #1 tested drug interaction with allopurinol. ANY rash = STOP immediately (SJS). Never start during acute gout. "A + A = Avoid" (Allopurinol + Azathioprine). Paradoxical early flares are EXPECTED — do NOT stop the drug.

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HIGH RISK | SAFETY ALERT | COMMONLY TESTED

COLCHICINE

Colcrys · Antigout / Anti-inflammatory Agent

MECHANISM

Inhibits microtubule polymerization by binding tubulin → disrupts mitotic spindle → prevents neutrophil migration to site of urate crystal deposition → blocks inflammatory cascade. Does NOT lower uric acid levels. Purely anti-inflammatory. Metabolized by CYP3A4 — critical drug interaction pathway.

INDICATION

Acute gout attacks (FIRST-LINE with NSAIDs), prevention of gout flares during allopurinol initiation (prevents early-mobilization flares), familial Mediterranean fever (FMF), acute/recurrent pericarditis. Take at FIRST sign of an acute attack.

MUST-ASSESS BEFORE GIVING

- Renal function (GFR) — DOSE ADJUST; AVOID if GFR < `30 mL/min`
- Liver function — hepatically metabolized; reduce in hepatic impairment
- CYP3A4 inhibitors (clarithromycin, ketoconazole, ritonavir) — DRAMATICALLY increase colchicine levels → POTENTIALLY FATAL toxicity
- P-glycoprotein (P-gp) inhibitors — same increased toxicity risk
- Statin use — increased myopathy risk
- GI history — nausea, vomiting, diarrhea are dose-limiting

KEY LABS

- BUN, Cr, GFR — renal dose adjustment; accumulation risk
- LFTs — hepatic metabolism
- CBC — bone marrow suppression (toxicity or chronic use)
- CK — myopathy risk (especially with statins)
- Serum uric acid (monitoring — colchicine does NOT change uric acid levels)

MOST DANGEROUS ADVERSE EFFECT

⚠ MULTI-ORGAN FAILURE with overdose. BONE MARROW SUPPRESSION (agranulocytosis, aplastic anemia). FATAL TOXICITY when combined with CYP3A4 inhibitors (clarithromycin most significant — even at therapeutic doses). Neuromuscular toxicity (myopathy, neuropathy). Dialysis NOT effective (highly protein-bound).

COMMON SIDE EFFECTS — TOXICITY ALARM

GI toxicity: nausea, vomiting, DIARRHEA (most common, dose-limiting, and EARLY SIGN OF TOXICITY), abdominal cramping. Diarrhea = warning that dose is too high. Alopecia with chronic use.

HOLD / NOTIFY PROVIDER

- HOLD if diarrhea is severe — early toxicity sign
- HOLD if WBC < `3,000/mm³` (bone marrow suppression)
- AVOID in GFR < `30 mL/min`
- AVOID concurrent clarithromycin/ketoconazole/ritonavir without dose adjustment
- NOTIFY if muscle weakness or peripheral neuropathy develops

ANTIDOTE / MANAGEMENT OF TOXICITY

NO specific antidote. Supportive care: GI decontamination if early overdose, respiratory support, G-CSF for agranulocytosis, transfusions. Dialysis is NOT effective (highly protein-bound). Prevention = medication reconciliation before ANY new prescription.

PATIENT TEACHING

- Take at FIRST SIGN of a gout attack — earlier = more effective
- Follow EXACT prescribed dose — do NOT take extra doses (FATAL toxicity risk)
- DIARRHEA = sign you have taken too much — stop and call provider
- AVOID GRAPEFRUIT JUICE (CYP3A4 inhibition → increases colchicine levels)

NURSING ACTION

- ✓ Confirm renal AND hepatic function before administration
- ✓ Medication reconciliation — CYP3A4 inhibitor check is ESSENTIAL
- ✓ Educate: diarrhea = toxicity alarm, NOT just a minor side effect
- ✓ Do NOT combine with clarithromycin without explicit provider guidance and dose reduction

- INFORM ALL providers (dentists, specialists) you take colchicine — MANY antibiotics cause dangerous interactions
- Colchicine does NOT lower uric acid — allopurinol/febuxostat needed for long-term prevention
- Report: severe diarrhea, muscle weakness, tingling in hands/feet, unusual bruising

✓ Chronic use: monitor CBC and CK regularly
✓ Remind patient: treats INFLAMMATION, not uric acid

🔍 NGN CLINICAL JUDGMENT CUE

Chronic gout patient on colchicine is prescribed clarithromycin for pneumonia. 3 days later: profuse watery diarrhea, severe leg weakness, unable to stand. CBC: WBC 1,200/mm³, neutrophils 300/mm³. = Colchicine toxicity from CYP3A4 inhibition by clarithromycin.

📝 NGN PRIORITY QUESTION

A gout patient asks: "My doctor prescribed colchicine AND allopurinol. Why do I need both? Isn't one enough?" Which nurse response is MOST accurate?

A) "You're right — they treat gout the same way, so using both is redundant."

B) ✓ **"Colchicine treats the inflammation of gout attacks, while allopurinol lowers the uric acid that causes gout."**

C) "Both medications work together to lower your uric acid and prevent future attacks."

D) "Colchicine protects your kidneys from the side effects of allopurinol."

✓B **These drugs have completely different mechanisms — they are complementary, not redundant.** Colchicine = ANTI-INFLAMMATORY (blocks neutrophil migration; does NOT lower uric acid). Allopurinol = URIC ACID REDUCER (inhibits xanthine oxidase). During allopurinol initiation, mobilization of urate crystals can trigger flares — colchicine prevents these early attacks. A: incorrect mechanisms. C: colchicine does NOT lower uric acid. D: colchicine has no renal-protective role against allopurinol.

🧠 **TIP:** COLCHICINE = stops the FIGHT (inflammation) but does NOT lower uric acid. ALLOPURINOL = reduces uric acid PRODUCTION. Together = standard of care during allopurinol initiation. Diarrhea = toxicity alarm. CYP3A4 inhibitor (clarithromycin) + colchicine = potentially FATAL. "COLchi = COLic/diarrhea = STOP."