NCLEX-RN · 2026 TEST PLAN · DAY 01

Cardiovascular & *Anticoagulants*

An advanced, NCJMM-aligned pharmacology deck built on the 2026 NCLEX-RN Test Plan — Pharmacological & Parenteral Therapies (13–19%), Reduction of Risk Potential, Physiological Adaptation, and Safety/Infection Prevention & Control.

CARDS: 22 NGN ITEMS: 5 CALC QS: 5 SAFETY RULES: 5 CASE STUDY: 1 (FULL NCJMM)

DIFFICULTY: APPLICATION+

01 Advanced Flashcards

22 CARDS · CARDIO + ANTICOAG

01 / 22

Lisinopril

ACE INHIBITOR — PROTOTYPE "-PRIL"

HIGH RISK · TEACHING

MECHANISM (SIMPLE)

Blocks the enzyme that turns angiotensin I into angiotensin II → vessels relax, less aldosterone → less Na/water held → BP drops.

WHY GIVEN

HTN, HF (reduces remodeling), post-MI, diabetic nephropathy (protects kidneys).

MUST-ASSESS BEFORE

BP & HR · K+ · BUN/Cr · pregnancy status (TERATOGENIC — D/X) · history of angioedema · taking K-sparing diuretics/K supplements/NSAIDs/lithium.

KEY LABS/MONITORING

K+ (3.5–5.0), BUN/Cr (expect mild ↑ Cr; >30% from baseline = concern), BP. Recheck within 1–2 weeks.

MOST DANGEROUS AE

Angioedema (lips/tongue/airway) — life-threatening; permanent contraindication. Also hyperkalemia, acute kidney injury (esp. with NSAIDs + diuretic = "triple whammy").

COMMON SIDE EFFECTS

Dry nagging cough (bradykinin), orthostatic hypotension, dizziness, fatigue.

HOLD / NOTIFY

SBP < 90, K+ > 5.0, Cr ↑ > 30% baseline, any facial/oral swelling, pregnancy confirmed.

ANTIDOTE / REVERSAL

No specific antidote. Angioedema → STOP drug, secure airway, IM epinephrine, antihistamine, steroids.

PATIENT TEACHING

Rise slowly, no salt substitutes (contain KCl), no NSAIDs without provider, report swelling/cough immediately, do not stop abruptly, avoid pregnancy.

NURSING ACTION

Baseline BP/labs, monitor first dose for hypotension, daily weights for HF, teach lifelong adherence even when "feeling fine."

NGN CLINICAL CUE

Client started lisinopril 3 days ago now reports "lump in my throat" and lips look swollen → STOP & treat as airway emergency.

Priority Q: A nurse is reviewing the medication list of a 62-year-old with HFrEF newly prescribed lisinopril. Which combination requires the MOST urgent intervention?

A. Atorvastatin 40 mg daily
B. Spironolactone 25 mg daily + ibuprofen PRN for arthritis
C. Metoprolol succinate 25 mg daily
D. Aspirin 81 mg daily

ANSWER: B.

The "triple whammy" — ACE-I + K-sparing diuretic + NSAID — causes severe hyperkalemia and AKI. Spironolactone raises K+, NSAIDs reduce renal blood flow, and ACE-I impairs efferent arteriolar tone → kidneys cannot autoregulate. A is safe and indicated. C is guideline-directed HF therapy. D is appropriate for cardiovascular protection.

💡 **TIP** — "-pril" → think Potassium & Pregnancy & coughy Pulmonary. Always pair an ACE-I question with a K+ value in your scan.

02 / 22

Losartan

ARB — "-SARTAN"

HIGH RISK · TEACHING

MECHANISM (SIMPLE)

Blocks angiotensin II from binding its receptor — same downstream effect as ACE-I but bradykinin pathway is untouched.

WHY GIVEN

HTN, HF, diabetic nephropathy, alternative when ACE-I causes cough/angioedema.

MUST-ASSESS BEFORE

BP, K+, Cr, pregnancy (TERATOGENIC), prior angioedema (still possible, lower rate).

KEY LABS/MONITORING

K+, BUN/Cr, BP — same as ACE-I.

MOST DANGEROUS AE

Hyperkalemia, AKI, angioedema (rare), fetal harm.

COMMON SIDE EFFECTS

Dizziness, fatigue, hypotension. NO cough (key difference vs ACE-I).

HOLD / NOTIFY

SBP < 90, K+ > 5.0, pregnancy, swelling.

ANTIDOTE / REVERSAL

No antidote; supportive care.

PATIENT TEACHING

No salt substitutes, avoid pregnancy, expect BP-lowering effect over 3–6 wks, do not combine with ACE-I (no benefit, more harm).

NURSING ACTION

Same monitoring as ACE-I; document why ARB chosen (often: prior ACE cough).

NGN CLINICAL CUE

Client switched from lisinopril to losartan for cough — develops new lip swelling. ARBs still carry small angioedema risk → STOP & assess airway.

Priority Q: Which finding in a client taking losartan most warrants holding the next dose?

A. Serum K+ 4.6 mEq/L

B. BP 138/82, HR 76

C. Serum K+ 5.8 mEq/L with peaked T waves on ECG

D. Reports occasional dizziness on standing

ANSWER: C.

Hyperkalemia with ECG changes is an emergency — peaked T waves precede V-fib. Treatment: calcium gluconate (membrane stabilization), insulin + D50, kayexalate/patiromer. A is normal. B is acceptable BP. D is expected and managed by teaching.

💡 **TIP** — ACE-I ends in "-pril" (Pril → Productive cough); ARB ends in "-sartan" (Sartan → Silent — no cough).

03 / 22

Metoprolol

B1-SELECTIVE BLOCKER — "-OLOL"

HIGH RISK · COMMONLY TESTED

MECHANISM (SIMPLE)

Blocks β1 receptors on the heart → slower HR, weaker contraction, less O2 demand, lower BP, less renin.

WHY GIVEN

HTN, angina, post-MI, HFrEF (succinate form), SVT, A-fib rate control, migraine prophylaxis.

MUST-ASSESS BEFORE

Apical HR (full minute) · BP · history of asthma/COPD/heart block · diabetes (masks hypoglycemia signs) · current decompensated HF.

KEY LABS/MONITORING

HR, BP, glucose (in DM), ECG if titrating.

MOST DANGEROUS AE

Bradycardia, heart block, acute HF exacerbation, bronchospasm (less than nonselective), masked hypoglycemia.

COMMON SIDE EFFECTS

Fatigue, cold extremities, depression, decreased exercise tolerance, sexual dysfunction.

HOLD / NOTIFY

HR < 60, SBP < 90, new wheezing, new HF signs (crackles, weight gain, edema).

ANTIDOTE / REVERSAL

Glucagon (high-dose) for severe overdose; atropine for bradycardia.

PATIENT TEACHING

Take pulse daily, never stop abruptly (rebound HTN, MI, dysrhythmia), rise slowly, in DM watch for sweating/confusion instead of typical hypoglycemia signs (tachycardia is blocked).

NURSING ACTION

Hold for HR < 60 unless parameters say otherwise; document apical HR; assess for HF signs before each dose during initiation.

NGN CLINICAL CUE

Diabetic on metoprolol arrives confused and diaphoretic, HR 78 → suspect HYPOGLYCEMIA (tachycardia is masked). Check BG immediately.

Priority Q: A nurse is preparing to give metoprolol 50 mg PO to a 72-year-old with new HFrEF. Apical HR 54, BP 102/64, lung crackles in bases, weight ↑ 2 kg in 3 days. What is the priority action?

A. Give the dose; metoprolol improves HF outcomes

B. Hold the dose and notify the provider

C. Give half the dose and reassess in 1 hour

D. Give the dose with extra IV fluids to support BP

ANSWER: B.

Beta-blockers are started in STABLE HF only — crackles + weight gain = decompensation. Plus HR 54 is below safe parameter. Giving the dose would worsen pump failure. Splitting doses without an order is outside scope. IV fluids would worsen pulmonary edema.

TIP — Three reasons to hold a beta-blocker: HR <60 · SBP <90 · signs of acute decompensated HF. Memorize this triad.

04 / 22

Carvedilol

NONSELECTIVE B + A1 BLOCKER

HIGH RISK · HF

MECHANISM (SIMPLE)

Blocks β1, β2, AND α1 → slows HR, reduces afterload through vasodilation.

WHY GIVEN

HFrEF (one of the "core 4" GDMT drugs), HTN, post-MI.

MUST-ASSESS BEFORE

HR, BP (orthostatics), asthma/COPD history (β2 blockade can bronchoconstrict), HF status, glucose.

KEY LABS/MONITORING

HR, BP, daily weights, BNP trend, glucose in DM.

MOST DANGEROUS AE

Severe bradycardia, bronchospasm in reactive airway disease, orthostatic syncope, masked hypoglycemia.

COMMON SIDE EFFECTS

Dizziness, fatigue, weight gain (paradoxical, from initial fluid retention).

HOLD / NOTIFY

HR < 55, SBP < 85, new dyspnea/wheeze, syncope.

ANTIDOTE / REVERSAL

Glucagon, atropine, supportive.

PATIENT TEACHING

Take WITH food (slows absorption, reduces hypotension), rise slowly, expect "feel worse before better" in HF (titrate up over weeks), daily weight, never stop abruptly.

NURSING ACTION

Start LOW dose, titrate every 2 weeks if tolerated, hold for asthma exacerbation.

NGN CLINICAL CUE

Asthma client given carvedilol develops wheezing and SpO2 88%. Nonselective β-blocker → bronchospasm. STOP and treat.

Priority Q: Which client is the BEST candidate for carvedilol?

A. HFrEF (EF 28%), stable on lisinopril and furosemide

B. Asthma with frequent exacerbations

C. 2nd-degree heart block, type II

D. Cardiogenic shock on norepinephrine

ANSWER: A.

Stable HFrEF benefits enormously from carvedilol (mortality reduction). B is contraindicated (nonselective). C is contraindicated (worsens block). D is contraindicated (blunts inotropy in shock).

💡 **TIP** — "Selective" β1 = metoprolol, atenolol, bisoprolol (relatively safer in lung disease). "Nonselective" = propranolol, carvedilol, labetalol (block β2 → bronchoconstriction).

05 / 22

Amlodipine

DIHYDROPYRIDINE CCB — "-DIPINE"

TEACHING-HEAVY

MECHANISM (SIMPLE)

Blocks calcium entry into vascular smooth muscle → arteries dilate → BP drops. Minimal effect on heart at usual doses.

WHY GIVEN

HTN, chronic stable angina, Raynaud's.

MUST-ASSESS BEFORE

BP, peripheral edema baseline, liver function, grapefruit juice intake.

KEY LABS/MONITORING

BP, LFTs, edema.

MOST DANGEROUS AE

Reflex tachycardia, severe hypotension, hepatotoxicity (rare).

COMMON SIDE EFFECTS

Peripheral edema (DOSE-DEPENDENT, ankle swelling — distinguish from HF edema!), headache, flushing, gingival hyperplasia.

HOLD / NOTIFY

SBP < 90, new significant pitting edema, syncope.

ANTIDOTE / REVERSAL

Calcium gluconate, glucagon, IV lipid emulsion in massive overdose.

PATIENT TEACHING

Avoid grapefruit juice (↑ levels via CYP3A4 inhibition), good oral hygiene (gum overgrowth), expect ankle swelling — not a sign of HF if isolated.

NURSING ACTION

Distinguish CCB edema (bilateral, distal, not pitting deeply) from HF edema (with crackles, dyspnea, weight gain).

NGN CLINICAL CUE

New crackles + 3 kg weight gain in a client on amlodipine → not just CCB edema; suspect HF, escalate.

Priority Q: A client on amlodipine reports new ankle swelling. Which assessment finding rules OUT a worsening cardiac cause?

A. Bilateral 1+ pitting edema, lungs clear, weight stable
B. JVD elevated and bilateral crackles
C. Weight ↑ 4 lb in 3 days, dyspnea on exertion
D. S3 heart sound and orthopnea

ANSWER: A.

CCB-induced edema is typically symmetric, mild, and NOT accompanied by pulmonary findings or weight gain. B/C/D all indicate volume overload from cardiac decompensation — assess and escalate.

TIP — "-dipine" → think Distal (peripheral) edema, Dilator, avoid grapefruit Drinks.

06 / 22

Diltiazem & Verapamil

NON-DHP CCB

HIGH RISK · RHYTHM

MECHANISM (SIMPLE)

Block Ca in cardiac muscle AND nodes → slow HR, slow AV conduction, weaker contraction.

WHY GIVEN

A-fib/flutter rate control, SVT, angina, HTN.

MUST-ASSESS BEFORE

HR, BP, ECG (block? WPW?), HF status, current beta-blocker (avoid combo — bradycardia/block).

KEY LABS/MONITORING

HR, BP, ECG (PR interval), LFTs.

MOST DANGEROUS AE

Bradycardia, AV block, hypotension, HF exacerbation (negative inotrope).

COMMON SIDE EFFECTS

Constipation (esp. verapamil), peripheral edema, dizziness, headache.

HOLD / NOTIFY

HR < 60, SBP < 90, new heart block on ECG, HF signs.

ANTIDOTE / REVERSAL

Calcium gluconate, glucagon, atropine, lipid emulsion.

PATIENT TEACHING

Fluids/fiber for constipation, avoid grapefruit, monitor pulse, do not double doses, no driving until tolerance established.

NURSING ACTION

For IV diltiazem in A-fib: on telemetry, frequent BP checks, observe for bradycardia/block.

NGN CLINICAL CUE

Client on diltiazem started on metoprolol — HR drops to 38, BP 78/40 → DOUBLE node-blockade synergy. Hold both, call provider.

Priority Q: Which combination is most dangerous?

A. Amlodipine + lisinopril
B. Diltiazem + metoprolol
C. HCTZ + lisinopril
D. Spironolactone + furosemide

ANSWER: B.

Non-DHP CCB + beta-blocker = severe bradycardia and AV block (both suppress the AV node). Other combos are common and safe with monitoring.

💡 **TIP** — DHP (-dipine) → arteries only → think peripheral edema. Non-DHP (diltiazem, verapamil) → nodes → think rate control. Never combine non-DHP with a β-blocker without a strong reason.

07 / 22

Furosemide

LOOP DIURETIC

HIGH RISK · ELECTROLYTES

MECHANISM (SIMPLE)

Blocks Na-K-2Cl in loop of Henle → massive Na/H2O loss, also dumps K, Mg, Ca.

WHY GIVEN

HF, pulmonary edema, edema in liver/renal disease, HTN (especially with renal impairment).

MUST-ASSESS BEFORE

BP, weight, lung sounds, I/O, K+, Mg, Na, Cr, sulfa allergy, hearing baseline.

KEY LABS/MONITORING

K+ (3.5–5.0), Na, Mg, BUN/Cr, daily weight, I/O, BP, BNP trend.

MOST DANGEROUS AE

Severe hypokalemia → dysrhythmias · ototoxicity (push IV slowly, ≤4 mg/min, esp. with aminoglycosides) · profound dehydration · hyponatremia.

COMMON SIDE EFFECTS

Frequent urination, dizziness, muscle cramps, photosensitivity.

HOLD / NOTIFY

K+ <3.5, SBP <90, weight loss >2 lb/day in HF without orders, ringing in ears.

ANTIDOTE / REVERSAL

No specific antidote — replace fluid & electrolytes (esp. K+, Mg).

PATIENT TEACHING

Take in morning, daily weight (call if >2–3 lb/day or 5 lb/week), eat K-rich foods (banana, orange, potato, spinach, avocado), rise slowly, sun protection, report ringing in ears.

NURSING ACTION

IV push SLOW (over 1–2 min), monitor for K+ drop in first 24 hrs, replace K+ before/along with dosing if low, weight daily SAME time/clothes/scale.

NGN CLINICAL CUE

HF client on furosemide + digoxin develops nausea + yellow-green vision + irregular pulse → hypokalemia potentiates DIGOXIN TOXICITY. Hold both, get level & K+.

Priority Q: Which client receiving IV furosemide requires the most immediate intervention?

A. Reports urinary frequency 4 hours after dose

B. K+ 3.2 mEq/L with U waves and PVCs on ECG

C. BP 118/72 after 20 mg dose

D. Has lost 1.5 lb in 24 hours

ANSWER: B.

Hypokalemia with ECG changes (U waves, PVCs) → risk of lethal arrhythmia. Replace K+ promptly, hold further furosemide doses until corrected. A = expected. C = acceptable. D = appropriate diuresis goal.

💡 **TIP** — Loop "loses" everything ("Lasix loses K, Na, Ca, Mg, H2O, hearing"). Thiazides "save Ca" (only loops lose Ca). K-sparing "saves K."

08 / 22

Hydrochlorothiazide (HCTZ)

THIAZIDE DIURETIC

TEACHING · FIRST-LINE HTN

MECHANISM (SIMPLE)

Blocks Na/Cl in distal tubule → mild Na/H2O loss, loses K and Mg, SAVES Ca.

WHY GIVEN

HTN (first-line per most guidelines), mild edema, prevents Ca-based kidney stones, central diabetes insipidus.

MUST-ASSESS BEFORE

BP, K+, Na, glucose, uric acid (worsens gout), sulfa allergy.

KEY LABS/MONITORING

K+, Na, glucose, uric acid, lipid panel.

MOST DANGEROUS AE

Hypokalemia, hyponatremia (esp. older adults), hyperglycemia, hyperuricemia → gout flare, hypercalcemia.

COMMON SIDE EFFECTS

Dizziness, dry mouth, photosensitivity, ED.

HOLD / NOTIFY

K+ <3.5, Na <130, SBP <90, gout flare, glucose >250 in DM.

ANTIDOTE / REVERSAL

None — electrolyte replacement.

PATIENT TEACHING

Morning dosing, K-rich foods, sun protection, monitor glucose if diabetic, report joint pain (gout).

NURSING ACTION

Trend Na & K weekly during titration, watch for confusion in elderly (hyponatremia).

NGN CLINICAL CUE

78 y/o on HCTZ now confused, Na 124 → thiazide-induced hyponatremia, esp. with thirst/water intake. Hold drug, restrict water, replace cautiously.

Priority Q: HCTZ is least appropriate for which client?

A. 55 y/o with HTN and history of Ca-oxalate kidney stones
B. 70 y/o with HTN and osteoporosis
C. 60 y/o with HTN and severe gout (3 flares in last year)
D. 65 y/o with HTN and well-controlled DM2

ANSWER: C.

Thiazides raise uric acid → trigger gout. A: thiazide REDUCES Ca-stones (saves Ca, less in urine). B: ↑ Ca retention can benefit osteoporosis. D: monitor glucose but not a hard contraindication.

TIP — Thiazide pearls: "Hyper GLUC" — Glucose, Lipids, Uric acid, Calcium all go UP. K, Na, Mg go DOWN.

09 / 22

Spironolactone

K-SPARING / ALDOSTERONE ANTAGONIST

HIGH RISK · K+

MECHANISM (SIMPLE)

Blocks aldosterone in distal tubule → keeps K, dumps Na & water mildly.

WHY GIVEN

HFrEF (mortality benefit), resistant HTN, ascites/cirrhosis, primary aldosteronism, PCOS, hirsutism.

MUST-ASSESS BEFORE

K+, BUN/Cr, BP, current ACE-I/ARB/K supplements/salt substitutes, pregnancy.

KEY LABS/MONITORING

K+ (recheck within 1 week, then periodically), Na, Cr.

MOST DANGEROUS AE

HYPERKALEMIA → fatal arrhythmias; AKI; gynecomastia.

COMMON SIDE EFFECTS

Gynecomastia, breast tenderness, menstrual irregularities, hyperkalemia.

HOLD / NOTIFY

K+ >5.0, Cr ↑ significantly, breast pain/swelling, irregular pulse.

ANTIDOTE / REVERSAL

Stop drug; for severe hyperkalemia: calcium gluconate (membrane stab), insulin + D50, kayexalate/patiromer.

PATIENT TEACHING

NO K supplements or salt substitutes, no high-K diet without provider, report breast changes, take with food.

NURSING ACTION

Trend K+ closely especially in renal impairment, watch for hyperkalemia signs (paresthesias, weakness, bradycardia, peaked T waves).

NGN CLINICAL CUE

HF client on spironolactone + lisinopril + KCl reports weakness, ECG peaked T waves → K+ likely >6. EMERGENCY: calcium gluconate first.

Priority Q: Which order in a client newly started on spironolactone requires clarification?

A. Lisinopril 10 mg PO daily
B. Potassium chloride 20 mEq PO BID
C. Furosemide 40 mg PO daily
D. Atorvastatin 40 mg PO daily

ANSWER: B.

Spironolactone already retains K+; adding KCl risks lethal hyperkalemia. A is monitored but synergistic for HF. C balances K+ effects. D is unrelated to K+.

💡 **TIP** — If the drug ends in "-actone" or starts with "spiro-" → think SAVES K+. Pair with: NO salt substitutes, NO KCl, watch ACE-I combo.

10 / 22

Digoxin

CARDIAC GLYCOSIDE

NARROW THERAPEUTIC INDEX

MECHANISM (SIMPLE)

Inhibits Na/K ATPase → ↑ intracellular Ca → stronger contraction (positive inotrope); also slows AV conduction (vagal tone).

WHY GIVEN

HFrEF symptomatic despite GDMT, A-fib rate control (especially in HF).

MUST-ASSESS BEFORE

Apical HR full minute, K+, Mg, Cr, digoxin level (0.5–2.0 ng/mL; HF target 0.5–0.9).

KEY LABS/MONITORING

Dig level, K+, Mg, Cr, ECG.

MOST DANGEROUS AE

DIGOXIN TOXICITY: bradycardia, heart block, V-tach. Hypokalemia & hypomagnesemia ↑ risk. Renal impairment ↑ levels.

COMMON SIDE EFFECTS

Anorexia, nausea, fatigue, yellow-green halos around lights, confusion (especially elderly).

HOLD / NOTIFY

HR <60 (adult), <70 (child), <90–110 (infant); ANY sign of toxicity; level >2.

ANTIDOTE / REVERSAL

Digoxin immune Fab (Digibind).

PATIENT TEACHING

Take pulse daily, eat K-rich foods (low K = toxicity risk), report nausea/visual changes, no antacids within 2 hours (reduce absorption), do not double doses.

NURSING ACTION

Apical HR 1 full minute; check K+ and Mg before dosing; recognize toxicity signs early.

NGN CLINICAL CUE

"Everything looks yellow and the rim of the lamp has a halo" + nausea + HR 48 = classic dig toxicity. Hold dose, draw level & K+/Mg, anticipate Digibind.

Priority Q: A nurse receives the following 0800 results for a client on digoxin: K+ 3.0, Mg 1.4, dig level 2.6, apical HR 52. Which intervention is FIRST?

A. Administer the morning dose of digoxin

B. Hold digoxin, hold furosemide, notify provider; anticipate Digibind & K/Mg replacement

C. Give scheduled KCl 20 mEq PO and reassess in 4 hours

D. Place client on bedrest and recheck level in 24 hours

ANSWER: B.

Toxic level + low K+/Mg + bradycardia = imminent lethal arrhythmia. STOP digoxin, treat electrolyte deficits, expect Digibind. A worsens toxicity. C delays treatment. D is inadequate.

💡 **TIP —** "Dig + Low K = Big Trouble." Three risk factors for digoxin toxicity: low K, low Mg, renal impairment.

11 / 22

Nitroglycerin

NITRATE VASODILATOR

EMERGENCY · CHEST PAIN

MECHANISM (SIMPLE)

Releases nitric oxide → vasodilation (mostly veins) → preload drops → less O2 demand. At higher doses also dilates coronaries & arteries.

WHY GIVEN

Angina, ACS chest pain, acute decompensated HF/pulmonary edema, HTN emergency (IV).

MUST-ASSESS BEFORE

BP (CRITICAL), pain (0–10), recent PDE5 inhibitor (sildenafil/tadalafil/vardenafil — last 24–48 hr), right-sided MI, severe AS.

KEY LABS/MONITORING

BP q5 min during titration, pain, ECG, HA.

MOST DANGEROUS AE

SEVERE HYPOTENSION (esp. with PDE5 inhibitor → fatal), syncope, reflex tachycardia, headache.

COMMON SIDE EFFECTS

Throbbing headache (expected), flushing, dizziness, tolerance with continuous use.

HOLD / NOTIFY

SBP < 90, suspected R-sided MI, sildenafil use within 24 hr (48 for tadalafil).

ANTIDOTE / REVERSAL

None specific — IV fluids, Trendelenburg, dopamine/norepinephrine for severe drop.

PATIENT TEACHING

SL CHEST PAIN PROTOCOL:

sit down, 1 tab SL q5 min × 3 doses; call 911 if not relieved after FIRST tablet per current AHA guidance. Store in original dark bottle, replace q6 months, expect headache (use acetaminophen). NEVER with ED meds.

NURSING ACTION

Two large-bore IVs before starting IV nitro. Use non-PVC tubing for IV gtt. Wear gloves with topical/paste (you can absorb it). Remove old patch before new (overdose risk).

NGN CLINICAL CUE

Inferior MI on ECG (II, III, aVF), BP 90/50, given SL nitro → BP crashes to 60/30. Inferior MI often involves R ventricle — preload-dependent. Resuscitate with fluids.

Priority Q: A client in the ED reports chest pain. Which question must be answered before SL nitroglycerin is given?

A. Are you allergic to penicillin?

B. When did you last eat?

C. Have you taken sildenafil or tadalafil in the last 24–48 hours?

D. Have you had this pain before?

ANSWER: C.

Nitrates + PDE5 inhibitors = catastrophic hypotension; potentially fatal. A is unrelated. B/D are useful history but not safety-critical for first dose.

TIP — Always think "Nitrates + Erection meds = Fatal drop." Also: Inferior/RV MI + nitrate = preload collapse — pause and check ECG location first.

12 / 22

Norepinephrine

VASOPRESSOR (A1 ›› B1)

SHOCK · CRITICAL CARE

MECHANISM (SIMPLE)
Strong α1 → vasoconstriction → ↑ SVR/BP. Some β1 → mild ↑ contractility/HR.

WHY GIVEN
Septic shock (first-line), cardiogenic shock, severe hypotension not responsive to fluids.

MUST-ASSESS BEFORE
Central line in place? (vesicant — extravasation risk) MAP target (usually ≥65), fluid status, current arrhythmia, allergies.

KEY LABS/MONITORING
Continuous BP (arterial line preferred), HR, urine output (hourly), lactate, capillary refill, mental status, extremity perfusion.

MOST DANGEROUS AE
Extravasation → tissue necrosis (treat with PHENTOLAMINE infiltration). Reflex bradycardia, peripheral/digital ischemia, arrhythmias.

COMMON SIDE EFFECTS
Headache, anxiety, tremor.

HOLD / NOTIFY
Site blanching/pallor/pain, severe HTN, new arrhythmia.

ANTIDOTE / REVERSAL
Phentolamine for extravasation; stop infusion.

PATIENT TEACHING
(Family/caregiver) — explain need for central line and monitoring, frequent vitals, IV won't be stopped abruptly.

NURSING ACTION
Titrate per MAP order, taper down — never abrupt stop, check site q1h, dedicated line, double-pump check.

NGN CLINICAL CUE
Peripheral IV norepi infusing — site cool, pale, painful, blanched. EXTRAVASATION. Stop infusion (leave catheter), aspirate residual drug, give phentolamine SC around site.

Priority Q: A nurse is titrating norepinephrine for septic shock (goal MAP ≥65). BP 88/52, HR 118, MAP 64, lactate rising, U/O 15 mL/hr. Which action is BEST?

A. Stop the infusion to assess for fluid overload
B. Increase the infusion rate per protocol and reassess MAP in 5 minutes
C. Give a 1 L NS bolus instead
D. Switch to dopamine

ANSWER: B.
MAP below goal in septic shock — titrate up. A would worsen shock. C may be appropriate elsewhere in resuscitation but not the immediate response to MAP ‹ 65 already on norepi. D is not first-line per current sepsis guidelines.

💡 **TIP** — Vasopressors: "Levo-Lifts pressure." Always — central line preferred, MAP ≥65 is the magic number in septic shock, never stop abruptly.

13 / 22

Amiodarone

CLASS III ANTIARRHYTHMIC

HIGH TOXICITY · ACLS

MECHANISM (SIMPLE)

Prolongs action potential (K+ blocker) but also has Class I, II, IV effects — slows everything.

WHY GIVEN

Life-threatening V-tach/V-fib (ACLS), A-fib (rate & rhythm), stable wide-complex tachycardia.

MUST-ASSESS BEFORE

ECG (QT), BP, HR, pulmonary baseline (chest X-ray), TSH, LFTs, eye exam baseline, drug interactions (warfarin, digoxin — doubles their levels).

KEY LABS/MONITORING

ECG (QT prolongation), TSH (causes hypo OR hyper), LFTs, PFTs/CXR, eye exam yearly.

MOST DANGEROUS AE

Pulmonary fibrosis (often irreversible, life-threatening), hepatotoxicity, thyroid dysfunction, torsades de pointes (QT prolongation), blue-gray skin, corneal deposits, ↑ digoxin & warfarin levels.

COMMON SIDE EFFECTS

Photosensitivity, nausea, tremor, fatigue.

HOLD / NOTIFY

New cough/dyspnea (pulmonary toxicity), LFT >3× normal, QTc >500 ms, vision changes.

ANTIDOTE / REVERSAL

None — supportive; long half-life (~58 days) means effects linger.

PATIENT TEACHING

Wear sunscreen, report cough/SOB/weight changes, annual eye exam, lifelong monitoring even after stopping, do NOT take with grapefruit.

NURSING ACTION

Use in-line filter for IV (precipitates), central line preferred for long infusions (phlebitis), reduce warfarin and digoxin doses when initiating.

NGN CLINICAL CUE

Client 6 months on amiodarone reports new dry cough & exertional dyspnea — pulmonary toxicity. Stop drug, CXR, PFTs.

Priority Q: Client on amiodarone has INR rise from 2.4 to 5.8 while warfarin dose unchanged. What is the explanation?

A. Lab error — recheck

B. Amiodarone potentiates warfarin (CYP inhibition) — anticipate warfarin dose reduction

C. Patient is bleeding internally

D. Amiodarone has worn off

ANSWER: B.

Amiodarone inhibits warfarin metabolism — INR can spike. Standard practice: reduce warfarin by ~30–50%. A is unlikely. C is possible but not the cause. D is wrong.

💡 **TIP** — Amiodarone has the dirtiest side effect profile of any common drug — "from skin to lungs to liver to thyroid to eyes." Almost any new symptom = think amiodarone toxicity.

14 / 22

Adenosine

SVT-BREAKER

EMERGENCY · CODE DRUG

MECHANISM (SIMPLE)
Briefly blocks AV node conduction (~6 sec) — interrupts re-entry SVT.

WHY GIVEN
Stable SVT (PSVT) not responsive to vagal maneuvers.

MUST-ASSESS BEFORE
Continuous ECG, defib pads nearby, IV access (preferably AC vein), recent caffeine/theophylline (block effect), dipyridamole (potentiates).

KEY LABS/MONITORING
Continuous ECG (capture rhythm strip during conversion).

MOST DANGEROUS AE
Brief asystole (3–10 sec — EXPECTED), bronchospasm (avoid in asthma), profound bradycardia, sense of impending doom.

COMMON SIDE EFFECTS
Flushing, chest pressure, dyspnea, dizziness — all very brief.

HOLD / NOTIFY
Severe asthma, 2nd/3rd-degree heart block (unless paced), known severe sinus node dysfunction.

ANTIDOTE / REVERSAL
None needed — half-life <10 sec; effects self-resolve.

PATIENT TEACHING
"You will feel a heavy chest pressure, flushing, and like your heart pauses — it lasts less than 10 seconds."

NURSING ACTION
Give 6 mg rapid IV push over 1–3 sec, IMMEDIATELY follow with 20 mL NS flush, AC vein preferred, defib ready, capture rhythm strip. May repeat 12 mg ×2.

NGN CLINICAL CUE
After adenosine, monitor shows 7-second flatline then sinus rhythm at 80 — EXPECTED transient asystole. Document conversion.

Priority Q: Which technique is most critical for adenosine effectiveness?

A. Dilute in 50 mL NS over 30 minutes
B. Give over 5 minutes
C. Give as RAPID IV PUSH followed immediately by 20 mL saline flush, AC vein preferred
D. Give IM in deltoid

ANSWER: C.
Adenosine's half-life is <10 seconds — must reach the heart fast. Use 3-way stopcock or fast hands. A/B = inactivates the drug. D = no IM route.

💡 **TIP — "FAST & CLOSE": Fast push, Closer to heart (AC vein). If you don't push fast, the drug expires in the tubing.**

15 / 22

Atorvastatin / Rosuvastatin

HMG-COA REDUCTASE INHIBITOR

TEACHING · LONG-TERM

MECHANISM (SIMPLE)

Blocks cholesterol synthesis in liver → ↓ LDL, mild ↑ HDL, ↓ inflammation.

WHY GIVEN

Hyperlipidemia, ASCVD prevention, post-MI/stroke, diabetes risk reduction.

MUST-ASSESS BEFORE

Baseline LFTs, lipid panel, pregnancy (CONTRAINDICATED), muscle pain history, current CYP3A4 inhibitors (grapefruit, certain antifungals, amiodarone).

KEY LABS/MONITORING

Lipid panel 4–12 wk after start, LFTs (if symptoms), CK if muscle pain.

MOST DANGEROUS AE

RHABDOMYOLYSIS (muscle pain, dark "cola" urine, ↑ CK, AKI), hepatotoxicity (rare).

COMMON SIDE EFFECTS

Mild myalgia, GI upset, headache, new-onset DM (small risk).

HOLD / NOTIFY

Unexplained muscle pain/weakness, dark/tea-colored urine, jaundice, pregnancy.

ANTIDOTE / REVERSAL

Stop drug + IV fluids for rhabdo to protect kidneys.

PATIENT TEACHING

Take in evening (cholesterol made at night — most relevant for short-acting like simvastatin; atorva/rosuva can be any time), avoid grapefruit, report muscle pain or dark urine, lifelong therapy.

NURSING ACTION

Reinforce non-statin therapy is appropriate during pregnancy; review CYP3A4 interactions.

NGN CLINICAL CUE

Client on atorvastatin + new clarithromycin reports severe thigh pain & "cola" urine → RHABDO. Stop statin, IV fluids, check CK + Cr.

Priority Q: A client on atorvastatin reports diffuse muscle aching and dark urine. Which lab confirms suspicion of rhabdomyolysis?

A. LDL 130

B. CK 15,000 U/L with Cr 1.8 (baseline 0.9)

C. TSH 4.0

D. INR 1.2

ANSWER: B.

Massive CK elevation with AKI = rhabdomyolysis from statin myopathy. Other values are unrelated to muscle breakdown.

💡 **TIP** — "Statin + Muscle pain + Cola urine = Rhabdo until proven otherwise." Always pair statins with the question "any new muscle aches?"

16 / 22

HTN Emergency Drugs

NITROPRUSSIDE · LABETALOL · HYDRALAZINE · CLEVIDIPINE

CRITICAL CARE

MECHANISM (SIMPLE)

NITROPRUSSIDE:

arterial+venous dilator.

LABETALOL IV:

α1+β block (use in HTN emergency, pre-eclampsia).

HYDRALAZINE:

arterial dilator.

CLEVIDIPINE:

ultra-short DHP CCB.

WHY GIVEN

HTN emergency with end-organ damage (encephalopathy, MI, dissection, AKI, pulmonary edema, eclampsia).

MUST-ASSESS BEFORE

Arterial line (preferred), neuro baseline, target BP (lower by ≤25% in first hour to avoid hypoperfusion), pregnancy.

KEY LABS/MONITORING

Continuous BP, HR, neuro, urine output. Nitroprusside: thiocyanate level if >24 hr, lactate.

MOST DANGEROUS AE

Nitroprusside → cyanide/thiocyanate toxicity (esp. renal failure, prolonged infusion). Labetalol → bradycardia, bronchospasm. Hydralazine → reflex tachycardia, lupus-like syndrome.

COMMON SIDE EFFECTS

Hypotension, headache, nausea, flushing.

HOLD / NOTIFY

BP drop >25% in first hour, severe bradycardia, mental status change (cyanide).

ANTIDOTE / REVERSAL

Cyanide toxicity: hydroxocobalamin or sodium thiosulfate + sodium nitrite.

PATIENT TEACHING

Family — explain ICU monitoring, transition to oral antihypertensives, lifelong adherence to prevent recurrence.

NURSING ACTION

Nitroprusside: wrap bag in foil (light-sensitive — degrades to cyanide), max 10 mcg/kg/min, monitor for cyanide symptoms (confusion, lactic acidosis, almond breath).

NGN CLINICAL CUE

Day 3 on nitroprusside, client develops confusion, metabolic acidosis (lactate 6) — CYANIDE TOXICITY. Stop infusion, give antidote, switch agent.

Priority Q: A nurse caring for a 32-week pregnant client with severe pre-eclampsia (BP 200/118) and magnesium sulfate running. Which antihypertensive is FIRST-LINE?

A. IV nitroprusside

B. IV labetalol or hydralazine (or PO nifedipine IR)

C. IV enalaprilat

D. Sublingual nifedipine

ANSWER: B.

Labetalol, hydralazine, and IR nifedipine are first-line in pregnancy. Nitroprusside → cyanide risk to fetus. ACE-I are teratogenic. Sublingual nifedipine causes uncontrollable drops (never use).

💡 **TIP** — In pregnancy: "LAbor's HelpfuL Nurses" → Labetalol, Hydralazine, Nifedipine. ACE-I and ARBs are NEVER okay in pregnancy.

17 / 22

Heparin (Unfractionated)

ANTICOAGULANT — IV/SQ

HIGH-ALERT MEDICATION

MECHANISM (SIMPLE)

Activates antithrombin → inactivates thrombin (IIa) and factor Xa → blocks clot formation.

WHY GIVEN

DVT/PE treatment & prophylaxis, ACS, A-fib bridging, cardiac surgery, dialysis lines.

MUST-ASSESS BEFORE

Baseline aPTT, CBC (platelets!), bleeding history, recent surgery/trauma/stroke, current antiplatelets/NSAIDs, pregnancy (heparin is OK).

KEY LABS/MONITORING

APTT

(target 1.5–2.5× control; usually 60–80 sec) for therapeutic dosing.

ANTI-XA

if available.

PLATELETS BASELINE + DAY 5–14

(HIT screen). H/H.

MOST DANGEROUS AE

Bleeding (any site — GI, GU, intracranial), HIT (Heparin-Induced Thrombocytopenia, type II — platelet drop >50% by day 5–14, paradoxical clotting).

COMMON SIDE EFFECTS

Local bruising/injection-site reactions, mild thrombocytopenia, osteoporosis (long-term).

HOLD / NOTIFY

aPTT > therapeutic range, platelets <100k or >50% drop from baseline, any active bleeding, hematuria, melena.

ANTIDOTE / REVERSAL

PROTAMINE SULFATE

1 mg per 100 units heparin (max 50 mg).

PATIENT TEACHING

Bleeding precautions (electric razor, soft toothbrush), avoid IM injections, report bruising/black stools, no NSAIDs/aspirin unless ordered.

NURSING ACTION

SQ: rotate sites in abdomen ≥2" from umbilicus, DO NOT aspirate or massage, 90° angle, hold pressure. IV: independent double-check (high-alert), titrate per nomogram, recheck aPTT q6h initially.

NGN CLINICAL CUE

Day 7 of heparin, platelets fell from 220k → 75k AND new DVT in opposite leg → HIT TYPE II. Stop ALL heparin (including flushes!), start non-heparin anticoagulant (argatroban, bivalirudin).

Priority Q: A client on continuous heparin for PE has aPTT 110 sec (control 30), no overt bleeding. What is the priority?

A. Increase the infusion per protocol

B. Stop the infusion, notify provider, anticipate protamine if bleeding occurs

C. Give vitamin K 10 mg IV

D. Continue and recheck in 6 hours

ANSWER: B.

aPTT >100 = severely supratherapeutic, very high bleed risk. Stop infusion per protocol and notify. Protamine reserved for active bleeding. C is for warfarin, not heparin. D is unsafe.

💡 **TIP — Heparin = aPTT & Protamine. Warfarin = PT/INR & Vitamin K. Memorize the pair — it's high-yield every test.**

18 / 22

Enoxaparin

LMWH

HIGH RISK

MECHANISM (SIMPLE)

Preferentially inactivates factor Xa > IIa. More predictable than heparin → usually no routine monitoring.

WHY GIVEN

DVT/PE prophylaxis & treatment, ACS, bridging therapy, pregnancy-safe anticoagulation.

MUST-ASSESS BEFORE

Weight (dose is weight-based), CrCl (reduce if <30), platelets, bleeding risk, neuraxial anesthesia (epidural hematoma risk).

KEY LABS/MONITORING

Anti-Xa level (only if renal impaired, obese, pregnant, pediatric). Platelets baseline + periodically. No routine aPTT.

MOST DANGEROUS AE

Bleeding, HIT (lower risk than UFH), epidural/spinal hematoma in neuraxial procedures.

COMMON SIDE EFFECTS

Injection-site bruising, mild thrombocytopenia.

HOLD / NOTIFY

Active bleeding, platelets <100k, CrCl drop, planned spinal procedure.

ANTIDOTE / REVERSAL

Protamine — only partially effective (~60%).

PATIENT TEACHING

Self-injection technique, rotate abdominal sites, DO NOT expel air bubble in prefilled syringe (manufacturer-added), 90° angle, no rubbing, bleeding precautions.

NURSING ACTION

Hold per protocol before/after epidural placement; don't aspirate or massage; never give IM.

NGN CLINICAL CUE

Post-op client on enoxaparin develops new back pain + lower-extremity weakness after epidural removal → SPINAL HEMATOMA. STAT MRI, neurosurgery.

Priority Q: Teaching about self-administered enoxaparin, which statement requires correction?

A. "I'll inject in my abdomen, at least 2 inches from my belly button"

B. "I'll push out the air bubble before injecting"

C. "I won't rub the site after"

D. "I'll insert the needle at a 90° angle"

ANSWER: B.

The air bubble in pre-filled enoxaparin syringes is intentional — it clears the needle and prevents leakage/bruising. Do NOT expel.

💡 **TIP** — LMWH = "Lighter, More predictable Weight-based Heparin." No aPTT, do not expel air, do not aspirate, do not massage.

19 / 22

Warfarin

VITAMIN K ANTAGONIST

HIGH-ALERT · TEACHING-HEAVY

MECHANISM (SIMPLE)

Blocks vitamin K-dependent clotting factors (II, VII, IX, X) → takes 3–5 days to be effective.

WHY GIVEN

A-fib, mechanical heart valves (only oral option), recurrent DVT/PE, antiphospholipid syndrome.

MUST-ASSESS BEFORE

Baseline PT/INR, CBC, liver function, pregnancy (TERATOGENIC — category X), diet history, current meds (many interactions), fall risk.

KEY LABS/MONITORING
PT/INR

— target usually 2–3 (3–4 for mechanical mitral valve). Initially daily, then weekly, then monthly when stable.

MOST DANGEROUS AE

Bleeding (intracranial >> GI >> GU), warfarin-induced skin necrosis (rare, early), purple toe syndrome, teratogenic.

COMMON SIDE EFFECTS

Bruising, minor bleeding, GI upset.

HOLD / NOTIFY

INR > therapeutic range, any bleeding, planned procedure (hold 5 days before), pregnancy.

ANTIDOTE / REVERSAL
VITAMIN K (PHYTONADIONE)

— PO or IV (slow). For severe bleed: 4-factor PCC (Kcentra) or FFP.

PATIENT TEACHING

CONSISTENT vitamin K intake (don't avoid greens — keep them steady!), no NSAIDs/aspirin unless ordered, no alcohol binge, MedicAlert ID, bleeding precautions, ANY change in routine meds → call provider.

NURSING ACTION

Bridge with heparin for 3–5 days at start (warfarin is initially PRO-thrombotic due to early loss of protein C), patient education extensively.

NGN CLINICAL CUE

INR 8.5 with melena → severe over-anticoagulation w/ bleeding. STOP warfarin, give vitamin K + PCC, hold dose, find cause (new antibiotic? amiodarone? sudden diet change?).

Priority Q: Which is the BEST patient teaching point about diet on warfarin?

A. "Avoid all green leafy vegetables entirely"
B. "Keep your intake of vitamin K-containing foods consistent week to week"
C. "Double your vitamin K intake if your INR is low"
D. "Take a vitamin K supplement to be safe"

ANSWER: B.

CONSISTENCY is the goal. Drastic changes (binge or avoidance) destabilize the INR. Tell patients to keep their usual diet, not change it.

💡 **TIP** — "WEPT" — Warfarin = vitamin k antagonist, Extrinsic pathway, PT/INR monitored, Takes days to work. Pair with bleeding precautions.

20 / 22

Apixaban · Rivaroxaban · Dabigatran

DOACS

HIGH RISK · NEWER

MECHANISM (SIMPLE)

Apixaban/rivaroxaban → factor Xa inhibitors. Dabigatran → direct thrombin (IIa) inhibitor.

WHY GIVEN

Non-valvular A-fib stroke prevention, DVT/PE, post-orthopedic prophylaxis. NOT for mechanical valves.

MUST-ASSESS BEFORE

CrCl (renal-dosed), weight, bleeding risk, current P-gp/CYP3A4 inhibitors, planned surgery.

KEY LABS/MONITORING

No routine coag monitoring (advantage). CBC, Cr, liver enzymes periodically.

MOST DANGEROUS AE

Bleeding (GI > ICH compared to warfarin), especially with renal impairment.

COMMON SIDE EFFECTS

Mild bruising, GI upset (dabigatran more dyspepsia).

HOLD / NOTIFY

Active bleeding, CrCl drop, scheduled procedure (hold per timing protocol).

ANTIDOTE / REVERSAL

DABIGATRAN → IDARUCIZUMAB (PRAXBIND).
APIXABAN/RIVAROXABAN → ANDEXANET ALFA.

Activated charcoal if recent ingestion.

PATIENT TEACHING

Take exactly as prescribed (short half-life means missed doses = risk), no double-dosing, bleeding precautions, MedicAlert, no NSAIDs/herbal anticoagulants (ginkgo, garlic, ginger, ginseng).

NURSING ACTION

Verify indication (NOT mechanical valves!), confirm CrCl-based dosing, educate about strict adherence.

NGN CLINICAL CUE

Client on apixaban for A-fib comes for emergency surgery → check last dose & CrCl, anticipate andexanet alfa or 4-factor PCC.

Priority Q: A client with a mechanical mitral valve wants to switch from warfarin to apixaban. Best response?

A. "Apixaban is a great alternative; I'll let your provider know"
B. "DOACs are not approved for mechanical heart valves — warfarin remains the standard"
C. "You can switch but you'll need lifelong heparin too"
D. "Aspirin alone would be safer"

ANSWER: B.

RE-ALIGN trial showed worse outcomes with dabigatran in mechanical valves. ALL current DOACs are contraindicated; warfarin remains required.

TIP — "DOAC No-Go list": Mechanical valves, moderate-to-severe mitral stenosis, antiphospholipid syndrome (triple-positive), pregnancy/breastfeeding.

21 / 22

Aspirin & Clopidogrel

ANTIPLATELETS

HIGH RISK · ACS

MECHANISM (SIMPLE)

ASPIRIN:

irreversibly blocks COX-1 → ↓ thromboxane A2 → less platelet aggregation.

CLOPIDOGREL:

blocks P2Y12 ADP receptor on platelets.

WHY GIVEN

ACS (STEMI/NSTEMI dual therapy), post-stent (DAPT 6–12 mo), secondary stroke prevention, PAD.

MUST-ASSESS BEFORE

Bleeding history, recent surgery, GI ulcer, allergies (NSAID/aspirin sensitivity → asthma triad), planned surgery.

KEY LABS/MONITORING

CBC for bleeding, no routine functional platelet testing required.

MOST DANGEROUS AE

GI bleed, intracranial hemorrhage, aspirin sensitivity (asthma/nasal polyps/urticaria triad), Reye syndrome in children <19 with viral illness.

COMMON SIDE EFFECTS

Dyspepsia, bruising, tinnitus (aspirin toxicity sign), nausea.

HOLD / NOTIFY

Active bleeding, planned major surgery (hold per cardiologist with stents!), tinnitus.

ANTIDOTE / REVERSAL

No specific antidote — platelet transfusion if life-threatening bleeding (effects persist for life of platelet, ~7–10 days).

PATIENT TEACHING

Take with food, NEVER stop DAPT post-stent without cardiology approval (stent thrombosis = MI), report bleeding/black stools/severe headache, no aspirin in kids with fever.

NURSING ACTION

Verify stent timing before any procedure; coordinate with cardiology for elective surgery.

NGN CLINICAL CUE

Post-PCI client stopped clopidogrel "to save money" — develops crushing chest pain & ST elevation = STENT THROMBOSIS. Activate cath lab.

Priority Q: A 14-year-old with influenza has fever and headache. Which OTC analgesic should the nurse explicitly tell the parent to AVOID?

A. Acetaminophen
B. Aspirin
C. Ibuprofen
D. Saline gargle

ANSWER: B.

Aspirin in children/teens with viral illness → Reye syndrome (encephalopathy + liver failure). A is preferred. C is acceptable with hydration. D is supportive.

TIP — "ASA NO-NOs": kids w/ viral illness (Reye), asthma triad, peptic ulcers, 3rd trimester pregnancy, bleeding disorders.

22 / 22

Alteplase (tPA)

THROMBOLYTIC

EMERGENCY · CLOT-BUSTER

MECHANISM (SIMPLE)

Converts plasminogen → plasmin → dissolves existing clots.

WHY GIVEN

Acute ischemic stroke (within 3 h, up to 4.5 h in selected), massive PE, STEMI when PCI unavailable, occluded central line/dialysis catheter.

MUST-ASSESS BEFORE

Strict time-of-onset, NIH stroke scale, CT scan (rule out hemorrhage!), BP (must be <185/110), recent surgery/trauma/bleeding, anticoagulant use, INR <1.7, platelets >100k, glucose, weight (mg/kg).

KEY LABS/MONITORING

Neuro checks q15 min × 2 hr, then q30 min × 6 hr, then q1h × 16 hr. BP q15 min × 2 hr (keep <180/105 post-infusion).

MOST DANGEROUS AE

Intracranial hemorrhage (highest concern), systemic bleeding, angioedema, reperfusion arrhythmias.

COMMON SIDE EFFECTS

Bruising, minor bleeding from puncture sites.

HOLD / NOTIFY

ANY worsening neuro status, severe headache, vomiting, BP >185/110, new bleeding.

ANTIDOTE / REVERSAL

Cryoprecipitate, tranexamic acid (TXA), aminocaproic acid, platelet transfusion.

PATIENT TEACHING

(Family) Explain time-critical nature, ICU monitoring, frequent neuro checks, lifelong stroke prevention plan, MedicAlert.

NURSING ACTION

NO antiplatelets/anticoagulants × 24 hr post-tPA, NO NG tube/Foley/arterial sticks × 24 hr if possible, dedicated IV line, two RN dose verification.

NGN CLINICAL CUE

Post-tPA stroke client develops sudden severe headache, vomiting, drop in GCS — INTRACRANIAL BLEED. STOP infusion, head CT STAT, reverse with cryo/TXA.

Priority Q: A 68-year-old presents at 1430 with sudden left-sided weakness; symptoms started at 1300. CT shows no bleed. BP 178/100, INR 1.1, platelets 240k, glucose 130. Which is the priority?

A. Give tPA immediately — within 3-hour window
B. Lower BP to <185/110 first, then give tPA — within window and otherwise eligible
C. Wait 4 more hours to see if symptoms resolve
D. Give aspirin 325 mg and admit

ANSWER: B.

Onset 1.5 h ago = within 3-hour tPA window. BP must be <185/110 prior to administration (use labetalol or nicardipine). A skips a safety step. C wastes the window. D forgoes definitive therapy.

💡 **TIP —** "Time is Brain": 3-hour window (4.5 h with extra criteria). The biggest "no-go" is hemorrhage on CT. BP must be <185/110 before, <180/105 after.

02 5 "Must Not Miss" Safety Rules

HIGH-ALERT · DAILY REINFORCEMENT

01 **Hold the beta-blocker if HR <60 or SBP <90.**

Apical pulse for one full minute. Also stop and call for new HF signs (crackles, weight gain, edema) at initiation. Never stop beta-blockers abruptly — rebound HTN, MI, dysrhythmia.

02 **Digoxin + low K+ = lethal.**

Always check K+ and Mg before a digoxin dose. Triad of toxicity: nausea, yellow/green halos, bradydysrhythmia. Therapeutic level 0.5–2 (HF target 0.5–0.9). Antidote: Digoxin immune Fab.

03 **Nitrates and PDE5 inhibitors = fatal hypotension.**

Always ask about sildenafil/tadalafil/vardenafil in the last 24–48 hours before SL nitro, IV nitro, or isosorbide. Also pause if inferior MI suspected (preload-dependent).

04 **Heparin → aPTT & Protamine. Warfarin → PT/INR & Vitamin K.**

Never mix them up. HIT screen: platelets drop >50% by day 5–14 with new clot — STOP all heparin (including flushes) and switch to argatroban/bivalirudin.

05 **tPA: "Time is Brain" — and bleeding is the enemy.**

Confirm time of onset, CT rules out bleed, BP <185/110, INR <1.7, platelets >100k, glucose normal. NO antiplatelets/anticoagulants/sticks × 24 hr post-infusion. Neuro checks per protocol — sudden HA + vomiting = ICH.

03 5 Medication Calculation & Dosage Safety Questions

SHOW YOUR WORK

CALC · 01

Heparin titration. A 72 kg client has a continuous heparin order: bolus 80 units/kg IV, then start drip at 18 units/kg/hr. The bag is 25,000 units in 250 mL D5W. What is the (a) bolus dose in units and mL, and (b) drip rate in mL/hr?

▶ SHOW SOLUTION

CALC · 02

Norepinephrine drip. A 90 kg septic shock client is on norepinephrine 4 mg in 250 mL D5W at 8 mL/hr. What is the dose in mcg/kg/min?

▶ SHOW SOLUTION

CALC · 03

tPA dose for stroke. An 80 kg client meets all tPA criteria. Order: 0.9 mg/kg IV (max 90 mg), 10% as bolus over 1 min and remainder over 60 min. Calculate bolus and infusion.

▶ SHOW SOLUTION

CALC · 04

IV nitroglycerin titration. Order: titrate nitroglycerin from 5 mcg/min, increase by 5 mcg/min q5 min for pain. Bag: 50 mg/250 mL D5W. What is the starting infusion rate in mL/hr?

▶ SHOW SOLUTION

CALC · 05

Enoxaparin dose. Order: enoxaparin 1 mg/kg SQ q12h for DVT treatment in a 68 kg client. The prefilled syringe is 100 mg / 1 mL. What volume is given?

▶ SHOW SOLUTION

04 5 NGN-Style Items

MATRIX · BOW-TIE · SATA · PRIORITIZATION · CLOZE

NGN · MATRIX MULTIPLE RESPONSE

Case: 68 y/o post-MI day 2, new GDMT

> Newly prescribed: lisinopril 10 mg daily, metoprolol succinate 25 mg daily, atorvastatin 80 mg HS, aspirin 81 mg daily, clopidogrel 75 mg daily. Vitals: BP 112/68, HR 58, SpO2 96%. Labs: K+ 4.4, Cr 1.0. For each finding, indicate whether the nurse would **Administer**, **Hold & Reassess**, or **Hold & Notify Provider**.

FINDING / DRUG	ADMINISTER	HOLD & REASSESS	HOLD & NOTIFY
Lisinopril — BP 112/68	✓		
Metoprolol — HR 58, asymptomatic, BP 112/68		✓	
Atorvastatin — reports new bilateral thigh aching & "dark urine" since yesterday			✓
Aspirin 81 mg — patient on clopidogrel (DAPT post-stent)	✓		
Clopidogrel — patient asks to stop because "I bruise easily now"	✓		

Rationales: Lisinopril BP is fine. Metoprolol HR is at the borderline (60) — apical pulse 1 min, recheck; if stable and per parameters, may give (some protocols use HR ≥50). Atorvastatin: classic rhabdo signs — STOP & notify, send CK + Cr + UA myoglobin. DAPT must continue per cardiology — teach bleeding precautions, do not stop without cardiology.

NGN · BOW-TIE

Case: 75 y/o A-fib on warfarin presents with INR 7.2 and melena

Actions to take (choose 2)

- Stop warfarin immediately
- Give IV vitamin K + 4-factor PCC
- Resume warfarin at same dose
- Give protamine sulfate

CONDITION:
Major bleeding with supratherapeutic INR

Parameters to monitor (choose 2)

- INR q6h until < 1.5
- Hgb/Hct, BP, HR, mental status
- aPTT every 6 hours
- Serum potassium every 4 hours

Answers: Actions = Stop warfarin + IV vitamin K + 4-F PCC. Monitor = INR trend + Hgb/Hct + hemodynamics. Protamine is for heparin (distractor). aPTT and K+ are not the targets here.

NGN · SELECT ALL THAT APPLY

Q: A nurse is teaching a client newly prescribed apixaban for non-valvular A-fib. Which statements indicate correct understanding? *Select all that apply.*

1. "I should take my medication at the same times each day."
2. "I'll get my INR checked every week."
3. "I should tell any dentist or surgeon that I'm on a blood thinner."
4. "I can stop the medication for a few days if I run out — it has a long half-life."
5. "I shouldn't take ibuprofen without checking with my provider."
6. "If I have black stools or a bad headache I'll call my provider immediately."

Correct: 1, 3, 5, 6.
② INR is for warfarin — DOACs do not need routine coag monitoring.
④ DOACs have SHORT half-lives — missing doses quickly removes anticoagulation and ↑ stroke risk.

NGN · ORDERED RESPONSE

Q: A client on continuous IV heparin develops sudden severe headache and unilateral weakness. Order the nurse's actions from FIRST to LAST.

1. Stop the heparin infusion
2. Call a rapid response/stroke alert
3. Place the head of bed flat (or per protocol) and ensure airway
4. Obtain stat CT head and labs (aPTT, CBC, platelets)
5. Anticipate protamine sulfate administration if hemorrhage confirmed

Order: 1 → 3 → 2 → 4 → 5. Stop the offending drug FIRST (preventable harm), then airway/positioning, then activate team, then diagnostics, then reversal. ABC always wins ties — but stopping an active infusion that is harming the patient comes before everything else.

NGN · CLOZE / DROP-DOWN

Q: Complete the sentence using the most appropriate options.

The nurse caring for a client with HFrEF on digoxin and furosemide notes a serum potassium of 2.9 mEq/L and an apical heart rate of 50 with frequent PVCs. The nurse's **priority** intervention is to **[A]**, because hypokalemia **[B]** the risk of digoxin toxicity. The expected antidote, if confirmed, is **[C]**.

[A] options: hold digoxin and notify the provider · administer next dose · increase furosemide · give 500 mL normal saline bolus

[B] options: decreases · has no effect on · increases · masks

[C] options: protamine sulfate · flumazenil · digoxin immune Fab (Digibind) · vitamin K

Rationale: Hypokalemia sensitizes the myocardium to digoxin → ↑ toxicity risk. The drop in K+ & HR with PVCs is a dig toxicity pattern. Antidote is Digibind. Replace K+ before any restart.

05 **Mini Case Study · Full NCJMM**

6 CLINICAL-JUDGMENT STEPS

"The Brewing Storm" — Mr. R, 70 y/o male

Setting: Day 4 on a medical unit. **PMH:** HFrEF (EF 30%), A-fib, CKD stage 3 (baseline Cr 1.5), HTN, gout.
Home meds: Lisinopril 20 mg, carvedilol 12.5 mg BID, spironolactone 25 mg, furosemide 40 mg, digoxin 0.125 mg, warfarin 5 mg (INR 2.4 last week), atorvastatin 40 mg.
Today's new orders: Increase furosemide to 80 mg BID for "2 lb weight gain." Add KCl 20 mEq daily. Start clarithromycin for sinus infection.
Now 0700: Mr. R reports nausea, "yellow halos around the lights," weakness. Apical HR 46, BP 102/64, RR 18, SpO2 95%. Latest labs (last night): K+ 3.1, Mg 1.3, Cr 2.1, dig level 2.8 ng/mL, INR 6.9.

① RECOGNIZE CUES

Bradycardia (HR 46), GI & visual symptoms classic for digoxin toxicity, hypokalemia (3.1), hypomagnesemia (1.3), worsening renal function (Cr 2.1 vs 1.5), supratherapeutic INR (6.9), recent furosemide ↑, KCl just started, NEW clarithromycin.

② ANALYZE CUES

Multi-drug catastrophe brewing. Furosemide ↑ → ↓ K+ & Mg → sensitizes myocardium to digoxin. ↓ Renal clearance → digoxin accumulates → level 2.8 (toxic). Clarithromycin = CYP3A4 inhibitor → ↑ both warfarin AND digoxin levels. Lisinopril + spironolactone + recent K+ rise risk add electrolyte chaos. Visual halos + nausea + bradycardia = DIG TOXICITY TRIAD.

③ PRIORITIZE HYPOTHESES

① Digoxin toxicity with hypokalemia/hypomagnesemia → lethal arrhythmia risk (HIGHEST priority). ② Major bleeding risk from supratherapeutic INR. ③ Worsening AKI from drug accumulation. ④ Possible HF decompensation underneath all of this.

④ GENERATE SOLUTIONS

Hold digoxin, furosemide, KCl supplement (recheck K+ first), warfarin, and clarithromycin. Place on telemetry. Replace Mg first (Mg must be repleted for K+ to correct). Then replace K+. Anticipate digoxin immune Fab (Digibind). Anticipate vitamin K ± 4-F PCC for INR if bleeding. Consult cardiology & pharmacy. Strict I/O. Recheck labs in 6 hours.

⑤ TAKE ACTION

STOP all listed drugs. Call provider with SBAR. Place on continuous telemetry. Start IV access × 2. Send stat CBC, BMP, Mg, digoxin level repeat, INR, type & screen. Administer IV magnesium 2 g over 1 hr, then KCl per protocol (oral if no IV K+ ordered). Bedside fall precautions. Pharmacy notified to reverse interactions. Patient/family education in plain language about what is happening and why.

⑥ EVALUATE OUTCOMES

Within 6 hours: HR ≥60, no new ectopy on telemetry, K+ 3.8, Mg 2.0, dig level trending down, INR with active reversal plan, no overt bleeding, weight stable, mental status clear, vision normal, urine output ≥0.5 mL/kg/hr. If not improving → escalate to ICU.

06 Red-Flag Findings

STOP · HOLD · CALL · REVERSE · RESCUE

⚠ If you see these — act, do not wait.

ACE-I / ARB
Lip, tongue, or facial swelling = angioedema. STOP drug, secure airway, IM epinephrine, antihistamine, steroids. Never re-challenge.

Beta-blocker
HR <60 with symptoms, new HF signs (crackles, weight gain). Hold and notify. Atropine for symptomatic bradycardia; glucagon for massive OD.

Digoxin
HR <60, nausea, yellow-green halos, K+ <3.5. HOLD, draw level, replace K+/Mg, anticipate Digibind.

Nitrates
BP collapses after dose (especially with recent ED meds or inferior MI). STOP, fluids, Trendelenburg, vasopressor PRN.

Heparin (HIT)
Platelets drop >50% by day 5–14 + new clot. STOP all heparin (including flushes!), switch to argatroban or bivalirudin.

Warfarin / DOAC
Sudden severe headache, melena, hematuria, sudden focal neuro deficits. STOP drug, reverse (vit K + PCC for warfarin; idarucizumab/andexanet for DOACs).

tPA
Worsening neuro status, severe HA, vomiting during/after infusion = ICH. STOP infusion, CT STAT, cryoprecipitate + TXA.

Vasopressor
Site blanching/pain/coolness on a peripheral line = extravasation. STOP infusion (do not pull line), aspirate, give phentolamine SC around site.

Amiodarone
New dry cough + dyspnea = pulmonary toxicity (often irreversible). STOP, CXR, pulmonary consult.

Statin
Diffuse muscle pain + dark urine = rhabdomyolysis. STOP, IV fluids, CK + Cr + UA myoglobin.

07 "Easy to Confuse" Pairs

SIDE-BY-SIDE CLARITY

Heparin vs. Warfarin

Feature	Heparin (UFH)	Warfarin
Route	IV (or SQ)	PO only
Onset	Minutes	3–5 days
Monitor	**aPTT (1.5–2.5× control)**	**PT / INR (2–3)**
Antidote	**Protamine sulfate**	**Vitamin K + PCC/FFP**
Pregnancy	Safe (does not cross placenta)	Teratogenic (Category X)
Unique risk	HIT	Vitamin K diet interactions; warfarin skin necrosis (rare)

Furosemide vs. Spironolactone

Feature	Furosemide (loop)	Spironolactone (K-sparing)
Site of action	Loop of Henle	Distal tubule (blocks aldosterone)
Potency	Most powerful	Weak diuretic, strong K-saver
K+ effect	**↓ K+ (and Mg, Ca, Na)**	**↑ K+ (retains)**
Pair concerns	Watch K+ with digoxin	NO salt substitutes, NO KCl, caution with ACE-I/ARB
Key AE	Ototoxicity, dehydration, hypokalemia	Hyperkalemia, gynecomastia
Antidote	None — replace electrolytes	Calcium gluconate + insulin/D50 for hyperK

ACE inhibitor vs. ARB

Feature	ACE-I ("-pril")	ARB ("-sartan")
Mechanism	Blocks ACE enzyme	Blocks AT1 receptor
Cough	**YES (bradykinin)**	**NO**
Angioedema risk	Higher	Lower (still possible)
K+ effect	↑ K+	↑ K+
Pregnancy	Teratogenic	Teratogenic
Pick when	First-line in HFrEF/post-MI/CKD	ACE-I cough or angioedema

08

Day 1 — One-Page Infographic Summary

TAKE A SCREENSHOT

NCLEX-RN · 2026 · DAY 01 · CARDIO + ANTICOAG

The 60-Second Cardio & Clot Cheat-Sheet

"-PRIL" → ACE-I

- **Watch:** K+, Cr, cough, angioedema, pregnancy
- **Hold:** swelling · K>5 · SBP<90
- **Triple-whammy:** ACE-I + NSAID + diuretic = AKI

"-SARTAN" → ARB

- Like ACE-I, **no cough**
- Still teratogenic, still ↑K+
- Switch to if ACE-I cough

"-OLOL" → B-BLOCKER

- **Hold:** HR<60 · SBP<90 · new HF
- **Selective** (β1): metoprolol — relatively safer lungs
- **Nonselective** (carvedilol, propranolol): no asthma

CCBS

- **"-dipine"** → vessels → ankle edema, no grapefruit
- **Diltiazem/verapamil** → nodes → A-fib rate
- NEVER non-DHP + β-blocker without strong reason

DIURETICS

- **Loop** = loses everything (low K, Mg, Ca, Na, hearing)
- **Thiazide** = "HyperGLUC" up · K, Na, Mg down · saves Ca
- **Spiro** = saves K · gynecomastia · NO KCl/salt subs

DIGOXIN

- Level **0.5–2** (HF target 0.5–0.9)
- Triad: **N/V · yellow halos · bradycardia**
- Low K = high toxicity · Antidote = **Digibind**

NITRO

- SL: 1 q5min × 3 · 911 after 1st if not relieved
- **NO with PDE5 inhibitors** (24–48 h)
- Caution in inferior/RV MI · gloves for paste

PRESSORS

- Septic shock → **norepi**, MAP ≥65
- Central line preferred · taper, don't stop
- Extravasation → **phentolamine**

AMIODARONE

- "From skin to lungs to liver to thyroid to eyes"
- Doubles **warfarin & digoxin** levels
- New cough + dyspnea = **pulm fibrosis**

ADENOSINE

- **FAST PUSH + 20 mL flush**, AC vein
- Expect 3–10 sec asystole (normal)
- Avoid in severe asthma

STATINS

- Muscle pain + dark urine = **RHABDO**
- No grapefruit · pregnancy contraindicated
- Watch CK + Cr when symptoms

HTN EMERGENCIES

- Drop BP **≤25% in first hour**
- Pregnancy: **labetalol · hydralazine · nifedipine**
- Nitroprusside >24 h → cyanide watch

HEPARIN

- Monitor **aPTT** · antidote **protamine**
- HIT: platelets ↓50% day 5–14 = stop ALL heparin

WARFARIN

- Monitor **INR (2–3)** · antidote **vit K + PCC**
- Consistent vit K diet (don't avoid greens)

DOACS

- No routine labs · short half-life
- Dabi → **idarucizumab** · Xa → **andexanet**
- NOT for mechanical valves

- Pregnancy: safe

- Bridge with heparin · teratogenic

ANTIPLATELETS

- DAPT (ASA + clopidogrel) post-stent — **never stop without cardiology**
- Aspirin → Reye's in kids w/ viral illness

TPA

- **Time is brain**: 3 h (4.5 selected) · CT first
- BP < 185/110 before · < 180/105 after
- NO sticks/anticoag × 24 h post

TOP 3 LAB PAIRINGS

- Heparin → **aPTT**
- Warfarin → **PT/INR**
- Digoxin → **K+ & level**