PHARMACOLOGY · STUDY GUIDE

Exam 5 Study Guide

This guide covers chapters 9 and 10, the two cardiovascular chapters. Chapter 9 is blood and pressure: the drugs that thin, clot, rebuild and replace blood, and the antihypertensives. Chapter 10 is the heart itself: lipids, angina, heart failure, digoxin and rhythm. Nearly everything here is high-alert, so the numbers matter. Work one chapter at a time. Read the "what to know" paragraphs, copy the tables by hand, then try each worked example with the steps covered. Finish each chapter with its ten self-test items. Any dose or number in a worked example is a practice calculation only, not dosing advice.

WHAT THIS GUIDE COVERS

- Chapter 9 · the platelet plug and cascade, PT/INR and aPTT, heparin and HIT, enoxaparin, fondaparinux, warfarin and vitamin K, DOACs and their reversal, aspirin, P2Y12 inhibitors and DAPT, alteplase, ESAs, filgrastim, blood products and transfusion reactions, the blood pressure equation, every antihypertensive class, orthostatic hypotension and adherence.
- Chapter 10 · statins and myopathy, non-statin lipid agents, nitrates and the PDE5 rule, the ACS bundle, the four pillars of heart failure, diuretics, digoxin and potassium, the Vaughan Williams classes, amiodarone, adenosine, the apical pulse, levels and discharge teaching.

CHAPTER 9

Cardiovascular Drugs I

Read it in full: **Chapter 9 in the textbook**. Practice it in the lab: **The Clotting Cascade** and **Where Each Antihypertensive Lowers the Pressure**.

What to know

Hemostasis runs in three steps: vasoconstriction, a platelet plug, then a fibrin mesh from the cascade. Extrinsic (tissue factor, factor VII) and intrinsic pathways meet in the common pathway, where factor Xa turns prothrombin into **thrombin** and thrombin turns fibrinogen into fibrin.

PT/INR follows extrinsic and common and monitors warfarin; **aPTT** follows intrinsic and common and monitors heparin. Anticoagulants prevent clots from forming and growing; they do not dissolve an existing clot.

Unfractionated heparin boosts antithrombin, works in minutes IV, has a half-life of about 60 to 90 minutes, is monitored by aPTT (often 1.5 to 2.5 times control) or anti-Xa, and is reversed with **protamine**. **HIT**, usually 5 to 10 days in, drops platelets by 50% or more and causes **clotting**; stop all heparin including flushes and start a non-heparin agent such as argatroban.

Enoxaparin (LMWH) mostly blocks Xa, is weight-based SubQ without routine aPTT, is renally cleared and carries a spinal hematoma warning. Give it deep SubQ in the abdomen at least 2 inches from the umbilicus; do not expel the bubble, aspirate or massage. **Fondaparinux** is an indirect Xa inhibitor with no reliable reversal.

Warfarin blocks vitamin K recycling, so the liver cannot finish factors II, VII, IX and X (and proteins C and S). It takes 3 to 5 days, so acute clots are bridged with heparin until the INR is therapeutic on two consecutive days. Usual INR goal is 2.0 to 3.0 (2.5 to 3.5 for some valves). High INR without bleeding: hold and, if ordered, oral vitamin K. Major bleeding: IV phytonadione plus 4-factor PCC; IV vitamin K carries an anaphylactoid boxed warning and is infused no faster than 1 mg/min. Teach **consistent** vitamin K intake, not avoidance. Huge interaction list; teratogenic.

DOACs: dabigatran blocks thrombin (reversed by idarucizumab; original bottle, discard 4 months after opening, never open capsules); apixaban and rivaroxaban block Xa (for major bleeding, 4 factor PCC is now the usual reversal; andexanet alfa was withdrawn in December 2025; rivaroxaban treatment doses with food). No routine INR, but renal dosing, not for mechanical valves, and missed doses matter fast.

Arterial clots are platelet-rich, so they get **antiplatelets**; venous clots are fibrin-rich, so they get **anticoagulants**. **Aspirin** irreversibly blocks COX-1, disabling a platelet for its 7 to 10 day life; for suspected MI chew 162 to 325 mg non-enteric-coated; hold about 7 days before elective surgery. **Clopidogrel** blocks P2Y12, is a CYP2C19 prodrug (pair with pantoprazole, not

omeprazole); prasugrel is contraindicated after stroke or TIA; ticagrelor causes harmless dyspnea. Never stop DAPT after a stent without cardiology.

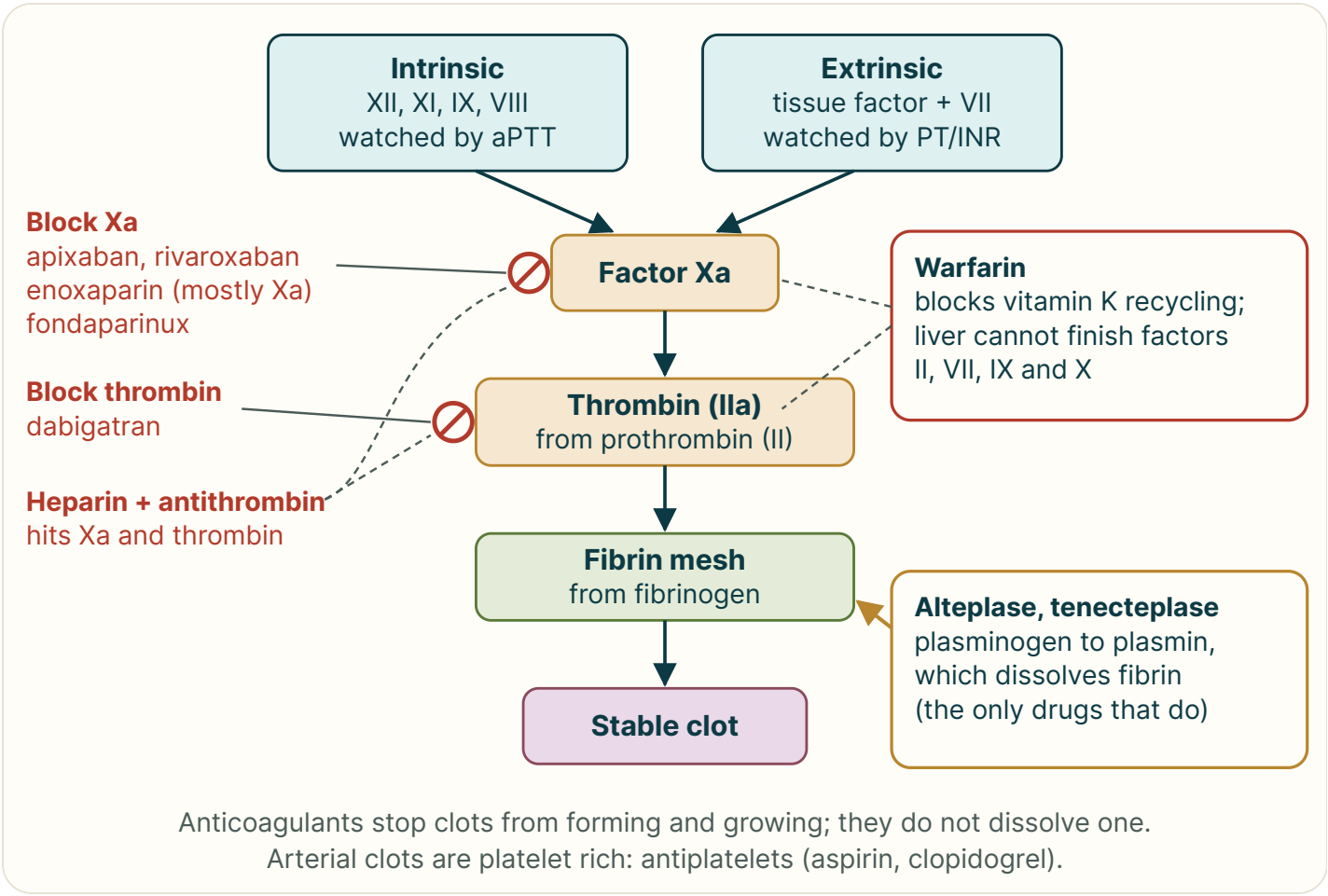
Thrombolytics (alteplase, tenecteplase) turn plasminogen into plasmin and dissolve fibrin. For stroke: noncontrast head CT first, within 3 hours of last known well (up to 4.5 in selected patients). Lines and labs before the infusion; no punctures or IM injections after; frequent neuro and BP checks; sudden headache or new deficit means stop and call.

ESAs (epoetin, darbepoetin) carry a boxed warning for death, MI, stroke, VTE and tumor growth if hemoglobin is pushed too high: hold above about 11 g/dL or a rise over about 1 g/dL in 2 weeks, check iron and BP, do not shake. **Filgrastim** goes at least 24 hours after chemotherapy; bone pain means it is working; follow the ANC; watch for splenic rupture.

Transfusion: NS only, 18 to 20 gauge for red cells, two-person bedside check, stay for the first 15 minutes, finish within 4 hours, never add drugs. For any reaction, **stop the transfusion first**, keep the line open with saline on new tubing, notify, return the unit. Acute hemolytic: fever, flank pain, dark urine, hypotension. **TACO** is hypertensive overload; **TRALI** is hypotensive lung injury.

BP = cardiac output × systemic vascular resistance. Drugs drain volume, slow the pump, widen the pipes or block the RAAS. **Thiazides**: hypokalemia, hyponatremia, high glucose and uric acid. **Loops**: potassium and calcium loss, ototoxicity with fast IV push. **Spironolactone**: hyperkalemia, gynecomastia. **ACE inhibitors** (-pril): cough, angioedema, hyperkalemia, first-dose hypotension, not in pregnancy. **ARBs** (-sartan): no cough. **Dihydropyridine CCBs** (-dipine): ankle edema. **Diltiazem, verapamil**: bradycardia, heart block, constipation. **Beta blockers**: never stop abruptly. **Alpha-1 blockers**: first dose at bedtime. **Clonidine**: rebound. Hydralazine causes reflex tachycardia; nitroprusside is the ICU drip with cyanide risk.

An accurate BP is an intervention: right cuff (bladder about 80% of the arm), seated 5 minutes, feet flat, arm at heart level, no talking, average two readings. **Orthostatic hypotension** is a drop of 20 systolic or 10 diastolic within 3 minutes of standing. Teach the slow three-stage rise. Non-adherence is rational when the disease is silent; predict side effects, teach "call, don't quit," use a home BP log, ask how many doses were missed, and avoid potassium salt substitutes, NSAIDs and decongestants.



Every anticoagulant has an address. Xa, thrombin, or the vitamin K factors; only the thrombolytics dissolve fibrin that is already there.

LOCK THESE IN

- aPTT for heparin (protamine); PT/INR for warfarin (vitamin K, plus 4-factor PCC for major bleeding).
- HIT: platelets fall 50% or more, clots form; stop all heparin.
- Warfarin: 3 to 5 days, bridge; INR 2 to 3; consistent vitamin K intake.
- DOAC reversal: idarucizumab (dabigatran); 4 factor PCC for apixaban and rivaroxaban (andexanet alfa was withdrawn in December 2025).
- Aspirin lasts the platelet's 7 to 10 days. Never stop DAPT after a stent without cardiology.
- Alteplase: CT first; 3 h (to 4.5 h); lines before; neuro and BP checks.
- Transfusion reaction: stop first. TACO hypertensive, TRALI hypotensive.
- Orthostatic: drop of 20 systolic or 10 diastolic within 3 minutes of standing.

Anticoagulants at a glance

Drug	Target	Monitor	Antidote
heparin	Antithrombin (thrombin, Xa)	aPTT or anti-Xa; platelets	Protamine sulfate
enoxaparin	Mostly Xa	None routine; renal function	Protamine (partial)
fondaparinux	Xa via antithrombin	None routine	None reliable
warfarin	Factors II, VII, IX, X	PT/INR 2 to 3	Vitamin K; 4-factor PCC
dabigatran	Thrombin	None routine	Idarucizumab
apixaban, rivaroxaban	Factor Xa	None routine; renal	4 factor PCC (andexanet withdrawn Dec 2025)
argatroban	Thrombin (for HIT)	aPTT	None

Key terms and comparisons: antihypertensives

Class (suffix)	Prototype	Signature effect	Teach
Thiazide	hydrochlorothiazide	Low K+, high glucose and uric acid	Morning dose
Loop	furosemide	K+ loss; ototoxic if pushed fast	Daily weight
Aldosterone antagonist	spironolactone	Hyperkalemia; gynecomastia	No salt substitutes
ACE inhibitor (-pril)	lisinopril	Cough, angioedema, high K+	Swelling = ED; not in pregnancy
ARB (-sartan)	losartan	High K+; no cough	Not in pregnancy
Dihydropyridine CCB (-dipine)	amlodipine	Ankle edema, flushing	Elevate legs
Non-DHP CCB	diltiazem, verapamil	Bradycardia, block, constipation	Pulse first
Beta blocker (-olol)	metoprolol	Bradycardia; masks hypoglycemia	Never stop abruptly
Alpha-1 blocker (-osin)	doxazosin	First-dose syncope	Bedtime; rise slowly
Central alpha-2	clonidine	Sedation; rebound	Never stop abruptly

Worked examples

WORKED EXAMPLE 1 · IS IT HIT?

Mr. Kovac is on day 7 of heparin. His platelets were 250,000 at baseline and are 110,000 today. Does this meet the book's threshold, and what do you do?

STEP 1 · FIND THE DROP

250,000 − 110,000 = 140,000.

STEP 2 · AS A PERCENT

140,000 ÷ 250,000 = 0.56, a **56% drop**.

STEP 3 · COMPARE

A fall of 50% or more on day 5 to 10 of heparin is HIT until proven otherwise.

STEP 4 · ACT

Stop all heparin, including flushes and coated catheters; notify the provider; expect argatroban. The danger is clotting, not bleeding.

WORKED EXAMPLE 2 · READING AN APTT AGAINST THE GOAL

The lab control aPTT is 30 seconds. The protocol goal is 1.5 to 2.5 times control. The patient's aPTT is 98 seconds. Where is he?

STEP 1 · FIND THE RANGE

30 × 1.5 = 45 s; 30 × 2.5 = 75 s. Goal **45 to 75 seconds**.

STEP 2 · COMPARE

98 s is above the range: more anticoagulated than intended.

STEP 3 · ACT

Follow the facility nomogram (often hold or reduce the rate), assess for bleeding and notify per protocol. Protamine is the reversal if bleeding is serious.

WORKED EXAMPLE 3 · HOW SLOWLY DOES IV VITAMIN K GO?

A patient with a major warfarin bleed is ordered 10 mg phytonadione IV with 4-factor PCC. The book limits IV vitamin K to no faster than 1 mg/min. What is the shortest infusion time?

STEP 1 · DIVIDE

10 mg ÷ 1 mg/min = **10 minutes** at the fastest.

STEP 2 · WHY BOTH DRUGS

Vitamin K restarts factor production over hours; PCC replaces factors now. Without vitamin K, the INR rebounds when PCC wears off.

STEP 3 · MONITOR

Cardiac monitor, emergency equipment ready, watch for flushing, hypotension, chest tightness or dyspnea.

WORKED EXAMPLE 4 · AN ORTHOSTATIC CHECK

Ms. Ndiaye, 78, on doxazosin and hydrochlorothiazide: supine 132/80 with HR 72; standing at 3 minutes 108/74 with HR 76. Is this orthostatic hypotension?

STEP 1 · SYSTOLIC CHANGE

132 − 108 = **24 mmHg** drop.

STEP 2 · DIASTOLIC CHANGE

80 − 74 = 6 mmHg drop.

STEP 3 · APPLY THE DEFINITION

A systolic drop of 20 or more within 3 minutes meets the definition, even though the diastolic drop is under 10.

STEP 4 · ACT

Report it, keep fall precautions, teach the three-stage rise, and ask whether the alpha blocker dose or timing can change.

TRAPS PEOPLE FALL INTO

- **Telling a warfarin patient to avoid all greens.** Consistency, not avoidance.
- **Watching a falling platelet count overnight on heparin.** HIT causes clots. Stop heparin now.
- **Calling an anticoagulant a clot dissolver.** Only thrombolytics dissolve fibrin.
- **Pairing clopidogrel with omeprazole.** Omeprazole blocks CYP2C19 activation; pantoprazole is preferred.
- **Giving filgrastim with chemotherapy.** At least 24 hours after.
- **Reaching for a diuretic before checking the BP in a transfusion reaction.** Stop first; TRALI is hypotensive and a diuretic worsens it.
- **Using a too-small cuff.** It reads falsely high.

Chapter 9 self-test · ten items

1. Which lab is used to monitor unfractionated heparin?

A. PT/INR
B. Ferritin
C. aPTT
D. CBC

C. aPTT aPTT tracks the intrinsic and common pathways. Platelets are also followed for HIT, but aPTT or anti-Xa titrates the dose.

2. A patient on heparin develops a new DVT while the platelet count falls by 60%. What is likely?

A. Warfarin-induced skin necrosis
B. A vitamin K deficiency state
C. An expected effect of heparin
D. Heparin-induced thrombocytopenia

D. Heparin-induced thrombocytopenia HIT paradoxically causes clotting. All heparin stops and a non-heparin anticoagulant starts.

3. Which enoxaparin technique is correct?

A. Leave the air bubble in the prefilled syringe
B. Inject it into the deltoid muscle instead
C. Massage the site well to speed absorption
D. Aspirate for blood before injecting it

A. Leave the air bubble in the prefilled syringe The bubble clears the drug from the needle track. Do not aspirate or massage; inject deep SubQ in the abdomen.

4. Why is heparin continued for the first days of warfarin therapy?

A. Heparin boosts warfarin absorption
B. Warfarin needs 3 to 5 days to work
C. Heparin lowers the INR target
D. Warfarin causes early bleeding alone

B. Warfarin needs 3 to 5 days to work Warfarin does not affect circulating factors. Heparin bridges until the INR is therapeutic on two consecutive days.

5. What reverses dabigatran?

A. Protamine sulfate
B. Andexanet alfa
C. Phytonadione
D. Idarucizumab

D. Idarucizumab Idarucizumab binds dabigatran. For the Xa inhibitors apixaban and rivaroxaban, 4 factor PCC is now the usual reversal; andexanet alfa was withdrawn in December 2025.

6. A patient with a coronary stent wants to stop clopidogrel before a dental extraction. What should the nurse say?

A. That decision belongs to the cardiologist
B. Switch to aspirin alone while at home
C. Stop it for one week before the visit
D. Stop both antiplatelets to be safe

A. That decision belongs to the cardiologist Stopping DAPT early can thrombose the stent. Most dental work proceeds on DAPT with local measures.

7. Before alteplase for stroke, what must be done first?

A. An IM antiemetic
B. A lumbar puncture
C. A noncontrast head CT
D. An arterial blood gas

C. A noncontrast head CT Giving a thrombolytic into a bleeding brain is fatal, so hemorrhage is ruled out first. Punctures and IM injections are avoided.

8. Ten minutes into a red cell transfusion, the patient has flank pain and falling BP. What is the first action?

A. Give a diuretic
B. Stop the transfusion
C. Slow the rate by half
D. Call the blood bank first

B. Stop the transfusion Stopping is always first. Every extra milliliter of an incompatible unit adds hemolysis.

9. Which antihypertensive is most associated with a dry, hacking cough?

A. Amlodipine
B. Losartan
C. Lisinopril
D. Metoprolol

C. Lisinopril ACE inhibitors let bradykinin build up. ARBs such as losartan do not, which is why patients are switched.

10. Which teaching is correct for a patient on spironolactone?

A. Eat extra bananas and oranges daily
B. Avoid potassium-based salt substitutes
C. Take potassium tablets every morning
D. Expect low potassium levels

B. Avoid potassium-based salt substitutes Spironolactone holds potassium. Salt substitutes are potassium chloride and can cause hyperkalemia.

CHAPTER 10

Cardiovascular Drugs II

Read it in full: **Chapter 10 in the textbook**. Practice it in the lab: **Before You Give Digoxin** and **Antiarrhythmics on the Action Potential**.

What to know

LDL carries cholesterol into the artery wall; HDL carries it out. Very high triglycerides (above about 500 mg/dL) are a pancreatitis issue. **Statins** block HMG-CoA reductase, so the liver adds LDL receptors and pulls LDL from blood; they also stabilize plaque and are started during ACS whatever the LDL. High-intensity regimens (atorvastatin 40 to 80 mg, rosuvastatin 20 to 40 mg) cut LDL by about half. Simvastatin, lovastatin and fluvastatin go in the evening; atorvastatin and rosuvastatin any consistent time. Grapefruit matters for simvastatin and lovastatin.

Statin muscle effects run from **myalgia** (normal CK) to **myopathy** (rising CK) to **rhabdomyolysis** (CK often over 10 times normal, cola-colored urine, kidney injury). Check lipids at baseline and 4 to 12 weeks, ALT at baseline only, and CK when muscles hurt. Stop and call for pregnancy. Add-ons: ezetimibe (gut absorption), PCSK9 antibodies and inclisiran (more LDL receptors), bempedoic acid (statin intolerance; gout), fibrates for triglycerides (never gemfibrozil with a statin), niacin (flushing; aspirin 30 minutes before), bile acid sequestrants (separate other drugs 1 h before or 4 h after), icosapent ethyl (bleeding, atrial fibrillation).

Every antianginal raises oxygen **supply** or lowers **demand**. Nitrates become nitric oxide and mainly dilate veins, dropping preload. Sublingual nitroglycerin: sit down, one 0.4 mg tablet or spray; if pain is not better in 5 minutes, call 911, then up to three doses 5 minutes apart. Original capped glass bottle away from heat and moisture; headache is expected. Patches need a 10 to 12 hour nitrate-free interval. IV nitroglycerin uses non-PVC tubing. Never combine with a **PDE5 inhibitor**: sildenafil or vardenafil within 24 hours, tadalafil within 48. Avoid in right ventricular infarction and SBP under 90.

Beta blockers are first-line for stable angina and cut mortality after MI, but are avoided in cocaine chest pain. CCBs are the choice for vasospastic angina; verapamil or diltiazem with a beta blocker risks heart block. Ranolazine prolongs QT. The **ACS bundle**: chewed aspirin first, a P2Y12 inhibitor, an anticoagulant, a beta blocker if stable, a high-intensity statin, an ACE inhibitor if stable. Oxygen only if SpO_2 is under about 90%; morphine is deprioritized. Door to needle 30 minutes or less; door to balloon 90 or less.

Heart failure with reduced EF is a neurohormonal siege. The **four pillars**, all mortality drugs: ARNI (sacubitril/valsartan) or ACE inhibitor or ARB (36 hour washout between an ACE inhibitor and an ARNI); an evidence-based beta blocker (carvedilol, metoprolol succinate, bisoprolol; start low, may feel worse for weeks); an MRA (spironolactone, eplerenone; check K+ and creatinine about 1 week and 1 month after starting); and an SGLT2 inhibitor. Extras: hydralazine with isosorbide dinitrate, ivabradine (luminous visual phenomena).

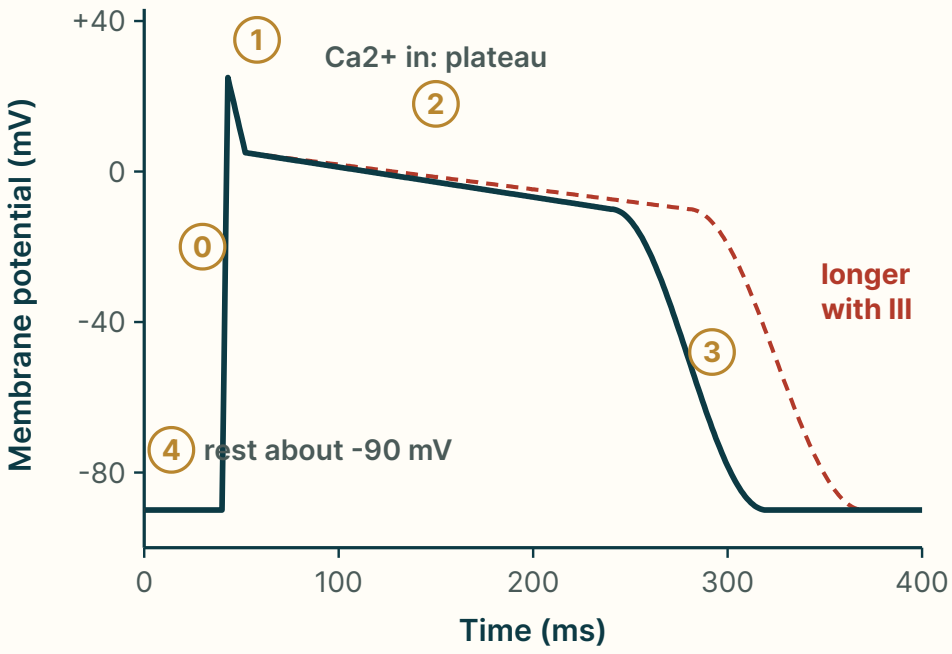
Diuretics relieve congestion but do not prolong life. Loops (furosemide) work at low GFR, waste potassium and magnesium, and are ototoxic if pushed fast (commonly no faster than about 20 mg/min). Thiazides fail below a GFR of about 30; metolazone 30 minutes before a loop strips potassium fast. Give doses in the morning and early afternoon.

Digoxin blocks the Na/K ATPase: stronger contraction and slower AV conduction. Range 0.5 to 2.0 ng/mL, but 0.5 to 0.9 in heart failure; draw a trough at least 6 to 8 hours after the dose. Toxicity: **anorexia first**, nausea, yellow-green vision and halos, confusion, any arrhythmia. **Hypokalemia**, hypomagnesemia and hypercalcemia make toxicity likely at a normal level. Amiodarone, verapamil, quinidine and clarithromycin raise levels (cut digoxin about in half with amiodarone). Antidote: **digoxin immune Fab**; afterward total levels are uninterpretable.

Antiarrhythmics are named for the channel they block. **Class I** sodium (phase 0): IA procainamide (lupus-like syndrome), quinidine (cinchonism); IB lidocaine (ventricular only; neurologic toxicity); IC flecainide (not with structural heart disease). **Class II** beta blockers. **Class III** potassium (phase 3, longer QT): amiodarone, sotalol, dofetilide, dronedarone. **Class IV** verapamil and diltiazem at the nodes. Magnesium treats torsades. Every antiarrhythmic is also proarrhythmic: a QTc over 500 ms or a rise over 60 ms is a call, with K+ and Mg^{2+} checked.

Amiodarone acts like all four classes, has a half-life of about 40 to 60 days, and injures lungs (fibrosis), thyroid (either way), liver, cornea and skin (blue-gray, photosensitivity). It raises digoxin and warfarin levels. **Adenosine** for stable SVT: warn the patient, 6 mg rapid IV push with an immediate 20 mL flush, expect a few seconds of asystole, keep a strip running.

Before digoxin, beta blockers, verapamil or diltiazem take an **apical pulse** for a full minute at the fifth intercostal space, midclavicular line. Hold digoxin for an adult rate under 60 (under 70 in a child, under 90 to 110 in an infant), over 100, or a new rhythm. Potassium 3.5 to 5.0 and magnesium 1.5 to 2.5 matter in both directions. Teach daily weights: call for 2 to 3 lb in a day or 5 lb in a week.



Class I: Na+ block
slows phase 0
wider QRS

Class III: K+ block
lengthens phase 3
longer QT

At the SA and AV nodes
II: beta blockers slow phase 4 and AV conduction
IV: verapamil, diltiazem block the nodal Ca2+ upstroke

Ventricular muscle cell. Every antiarrhythmic is also proarrhythmic: call for a QTc over 500 ms or a rise over 60 ms, and check K+ and Mg2+.

Antiarrhythmics are named for the channel they block. Sodium for phase 0, potassium for phase 3; beta blockers and calcium channel blockers work mainly at the nodes.

LOCK THESE IN

- Statin myopathy: muscle pain + dark urine = rhabdomyolysis; draw CK when symptomatic.
- Simvastatin and lovastatin in the evening; never gemfibrozil with a statin.
- SL nitroglycerin: sit, one dose, call 911 at 5 min, up to 3 doses 5 min apart.
- No nitrates within 24 h of sildenafil or 48 h of tadalafil.
- HFrEF pillars: ARNI/ACEi/ARB, evidence-based beta blocker, MRA, SGLT2 inhibitor.
- Digoxin: anorexia first; low K+ potentiates; HF target 0.5 to 0.9; antidote digoxin immune Fab.
- Vaughan Williams: I Na, II beta, III K (QT), IV Ca. Magnesium for torsades.
- Apical pulse 1 full minute; hold digoxin under 60 in adults.

Key terms and comparisons

Drug or class	Key effect	Nursing alert
Atorvastatin, rosuvastatin	LDL down 30 to 55%	Myopathy; CK if symptomatic
Ezetimibe	Blocks gut absorption	Separate from sequestrants
Fenofibrate	Triglycerides down	Safer statin partner than gemfibrozil
Niacin	HDL up, TG down	Flushing; aspirin before
Nitroglycerin SL	Preload down	PDE5 rule; SBP under 90 hold
Sacubitril/valsartan	ARNI	36 h after last ACE inhibitor
Spironolactone, eplerenone	MRA	K+ at 1 week and 1 month
Furosemide	Loop diuretic	No faster than about 20 mg/min IV
Digoxin	Inotrope, slows AV node	Apical pulse; K+; Fab antidote
Amiodarone	All four classes	Lungs, thyroid, liver, eyes, skin
Adenosine	Brief AV block	6 mg rapid push + 20 mL flush

Numbers to know

Check	Target	Act when
Apical pulse	60 to 100	Under 60 (adult): hold and call
Digoxin	0.5 to 2.0; HF 0.5 to 0.9 ng/mL	Toxic signs even at normal level
Potassium	3.5 to 5.0 mEq/L	Low: digoxin toxicity, torsades
Magnesium	1.5 to 2.5 mg/dL	Correct before potassium
QTc	Under 450 (men), 460 (women)	Over 500 or rise over 60 ms
Daily weight	Dry weight	2 to 3 lb/day or 5 lb/week
Nitrate hold	SBP under 90	Or RV infarction

Worked examples

WORKED EXAMPLE 1 · TIMING SUBLINGUAL NITROGLYCERIN

Mr. Abara's chest pain starts at 1900 while he is sitting. Walk through the book's sequence.

STEP 1 · FIRST DOSE

1900: sit, one tablet under the tongue.

STEP 2 · 5 MINUTE CHECK

1905: pain unimproved or worse, so **call 911** now.

STEP 3 · MORE DOSES WHILE WAITING

1905 second dose; 1910 third dose. **Three doses maximum**.

BEFORE ANY OF IT

Ask about erection drugs: sildenafil within 24 hours or tadalafil within 48 hours rules out nitrates.

WORKED EXAMPLE 2 · A PDE5 INHIBITOR CLOCK

A patient took tadalafil Monday at 2000. When is the earliest a nitrate could be considered? What if it had been sildenafil?

STEP 1 · TADALAFIL

48 hours: Monday 2000 + 48 h = **Wednesday 2000**.

STEP 2 · SILDENAFIL

24 hours: Monday 2000 + 24 h = **Tuesday 2000**.

THE RISK

Together they cause profound, refractory hypotension that can be fatal.

WORKED EXAMPLE 3 · HOW LONG AMIODARONE LINGERS

Using chapter 1's rule that a drug takes about 4 to 5 half-lives to clear, and amiodarone's half-life of about 40 to 60 days, how long can its effects outlast the last dose?

STEP 1 · LOW END

4 × 40 = **160 days**.

STEP 2 · HIGH END

5 × 60 = **300 days**.

STEP 3 · MEANING

Side effects can appear or persist many months after stopping, and digoxin and warfarin interactions persist too. Keep thyroid, liver, lung and eye follow-ups.

WORKED EXAMPLE 4 · A "NORMAL" DIGOXIN LEVEL

Mrs. Ito, 82, on digoxin and furosemide, has no appetite and says the TV looks yellowish. Digoxin 1.4 ng/mL; potassium 3.0 mEq/L; apical pulse 54. What do you do?

STEP 1 · READ THE LEVEL

1.4 is inside 0.5 to 2.0, and above the 0.5 to 0.9 heart failure target.

STEP 2 · READ THE POTASSIUM

3.0 is low. Potassium and digoxin compete for the same pump site, so low K+ lets digoxin bind more.

STEP 3 · READ THE PATIENT

Anorexia, yellow vision and a pulse of 54 point to toxicity despite a "normal" level.

STEP 4 · ACT

Hold the dose, notify the provider, check magnesium too, expect potassium replacement and, if life-threatening, digoxin immune Fab.

TRAPS PEOPLE FALL INTO

- **Giving simvastatin in the morning.** Short-acting statins go in the evening.

- **Stopping a statin for any ache.** Draw a CK, ask about urine color, and talk to the prescriber; most aching is benign.
- **Telling a patient to take three nitroglycerin then call.** Call 911 at 5 minutes after the first dose.
- **Giving routine oxygen in ACS with SpO2 96%.** Only if under about 90%.
- **Using metoprolol tartrate as the heart failure beta blocker.** Use metoprolol succinate, bisoprolol or carvedilol.
- **Trusting a normal digoxin level with low potassium.** Toxicity can occur at a normal level.
- **Giving adenosine slowly.** Its half-life is under 10 seconds; rapid push and immediate flush.

Chapter 10 self-test · ten items

1. A patient on rosuvastatin reports aching thighs and urine that looks like cola. What is the priority?

A. Add a second statin at a lower starting dose
B. Reassure him that aching is common and benign
C. Encourage more exercise to loosen the muscles
D. Hold the statin and check CK and kidney function

D. Hold the statin and check CK and kidney function Dark urine is myoglobin. This is rhabdomyolysis until proven otherwise.

2. Which lipid drug combination carries a high risk of severe myopathy?

A. Gemfibrozil with a statin
B. Icosapent ethyl with a statin
C. Ezetimibe with a statin
D. Fenofibrate with a statin

A. Gemfibrozil with a statin Gemfibrozil sharply raises statin levels. Fenofibrate is the safer fibrate partner.

3. A patient took sildenafil 10 hours ago and now has chest pain. Nitroglycerin is ordered. What should the nurse do?

A. Give it with an IV fluid bolus
B. Give it with the patient sitting
C. Hold it and notify the provider
D. Give half the usual dose

C. Hold it and notify the provider Nitrates within 24 hours of sildenafil can cause refractory, fatal hypotension.

4. Why is a nitroglycerin patch removed in the evening?

A. To reduce night headache only
B. To prevent nitrate tolerance
C. To allow the site to cool down
D. To avoid skin irritation only

B. To prevent nitrate tolerance A 10 to 12 hour nitrate-free interval keeps the vessels responsive.

5. Which drug is NOT one of the four pillars of HFrEF therapy?

A. Furosemide
B. Spironolactone
C. Carvedilol
D. Dapagliflozin

A. Furosemide Loop diuretics relieve congestion but do not lower mortality. The pillars are ARNI/ACEi/ARB, an evidence-based beta blocker, an MRA and an SGLT2 inhibitor.

6. How long must pass after the last ACE inhibitor dose before starting sacubitril/valsartan?

A. 12 hours
B. 7 days
C. 36 hours
D. 6 hours

C. 36 hours Overlap raises the risk of angioedema, so a 36 hour washout is required.

7. What is often the earliest sign of digoxin toxicity?

A. Slow heart rate
B. Loss of appetite
C. New seizures
D. Yellow-green vision

B. Loss of appetite Anorexia comes first. Yellow-green vision is nearly diagnostic but often later.

8. Which electrolyte problem most increases the risk of digoxin toxicity?

A. Hyperkalemia
B. Hypocalcemia
C. Hypernatremia
D. Hypokalemia

D. Hypokalemia Low potassium lets digoxin bind its pump site more avidly. Low magnesium and high calcium also increase risk.

9. Which drug is the treatment for torsades de pointes?

A. IV lidocaine
B. IV magnesium
C. IV adenosine
D. IV digoxin

B. IV magnesium IV magnesium treats torsades. Check potassium and magnesium with any QT-prolonging drug.

10. A patient on amiodarone develops a new dry cough and dyspnea. What is the concern?

A. Expected cold symptoms
B. ACE inhibitor cough
C. Pulmonary fibrosis
D. Digoxin toxicity

C. Pulmonary fibrosis Amiodarone can scar the lungs. New cough or dyspnea is reported promptly.

ONE PAGE

Cram sheet

Everything from chapters 9 and 10 in one place. Cover the right column and see if you can rebuild each row.

Blood and pressure

Item	Fact	Note
Heparin	aPTT 1.5 to 2.5 x control	Protamine; watch platelets
HIT	Day 5 to 10; drop of 50% or more	Clots; stop all heparin; argatroban
Enoxaparin	Deep SubQ abdomen	Keep bubble; no aspirate or massage
Warfarin	INR 2 to 3; 3 to 5 days	Vitamin K; PCC; IV vit K 1 mg/min max
DOACs	No routine INR	Idarucizumab; 4 factor PCC for Xa inhibitors
Aspirin (MI)	Chew 162 to 325 mg	Platelet 7 to 10 days
Clopidogrel	CYP2C19 prodrug	Pantoprazole; never stop DAPT alone
Alteplase (stroke)	CT first; 3 to 4.5 h	No punctures; neuro checks
ESAs	Hold if Hgb over about 11	Iron, BP
Filgrastim	At least 24 h after chemo	Bone pain; ANC
Transfusion	NS only; stay 15 min; 4 h max	Stop first for reactions
TACO vs TRALI	Hypertensive vs hypotensive	Diuretic only for TACO
Orthostatic	Drop 20 systolic or 10 diastolic in 3 min	Three-stage rise

The heart

Item	Fact	Note
Statins	Evening for simvastatin, lovastatin	CK if muscles hurt
SL nitroglycerin	Call 911 at 5 min; 3 doses max	Glass bottle; headache normal
PDE5 inhibitors	Sildenafil 24 h; tadalafil 48 h	No nitrates
ACS	Chewed aspirin; O2 only if SpO2 under 90%	Needle 30 min; balloon 90 min
HFrEF pillars	ARNI/ACEi/ARB, BB, MRA, SGLT2i	36 h ACEi to ARNI washout
Furosemide IV	About 20 mg/min max	Ototoxicity
Digoxin	0.5 to 0.9 in HF; trough 6 to 8 h+	Anorexia first; Fab antidote
Amiodarone	Half-life 40 to 60 days	Cut digoxin and warfarin
Adenosine	6 mg rapid + 20 mL flush	Brief asystole
QTc	Over 500 or rise over 60 ms	Check K+ and Mg
Apical pulse	Full minute, 5th ICS MCL	Hold digoxin under 60
Daily weight	2 to 3 lb/day or 5 lb/week	Call

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