Top 25 High Yield Drugs for Step 1

Question-tested pharmacology review

HOW IT'S TESTED → WHAT YOU NEED TO MEMORIZE → DON'T MISS

> **How to use this guide:** Start with **How It's Tested**. Those mini-stems are written to make you recognize common patient presentations. Then read **What You Need to Memorize** for the facts to get the question right, and **Don't Miss** for the common trap.

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1. ACE inhibitors

Drug Class: ACE inhibitor (lisinopril, enalapril, captopril)

How It's Tested

- *A 58-year-old with hypertension/diabetes starts a new medication and later develops a **dry cough**, **lip/tongue swelling** (angioedema), or hyperkalemia.*
- *A 70-year-old with abdominal or renal bruits has a **sharp creatinine rise** shortly after starting a BP medication.*

What You Need to Memorize

- MOA: inhibit ACE → decreased angiotensin II/aldosterone and increased bradykinin.
- **Increased bradykinin** → dry cough and angioedema.
- Decreased aldosterone → decreased K^+ secretion → hyperkalemia, worsened by ENaC blockers or aldosterone antagonists.
- Pregnancy exposure blocks fetal RAAS → oligohydramnios, **pulmonary hypoplasia**, **skull ossification defects**, and limb contractures.

Don't Miss

★ **In renal artery stenosis, creatinine rises because the kidney loses angiotensin II-mediated efferent arteriole constriction.**

2. Opioids

Drug Class: mu-opioid receptor (intoxication, withdrawal, neonatal abstinence)

How It's Tested

- *A college student is found by a roommate after a party: unresponsive, **respiratory rate 6/min**, and **pinpoint pupils**.*
- *A post-op patient returns restless with watery eyes, **yawning**, **diarrhea**, goosebumps, large pupils, and high BP/HR.*

What You Need to Memorize

- MOA: mu-opioid receptors are **G_i-coupled** → decreased cAMP, increased K^+ channel opening, and decreased presynaptic neurotransmitter release.
- Naloxone competitively antagonizes mu receptors; it can wear off before long-acting opioids and recurrent respiratory depression can occur.
- Buprenorphine is a high-affinity partial mu agonist that can displace full agonists and precipitate withdrawal.
- Neonatal opioid abstinence with irritability, high-pitched cry, hypertonia, or tremor is treated with an opioid agonist such as morphine.

Don't Miss

★ **Pinpoint pupils plus slow respirations is overdose; dilated pupils plus diarrhea/goosebumps is withdrawal.**

3. Benzodiazepines + alcohol withdrawal

Drug Class: GABA-A positive allosteric modulators (lorazepam, diazepam, chlordiazepoxide)

How It's Tested

- *A man admitted for trauma is stable at first; by hospital day 2-4/after surgery, he becomes tremulous, sweaty, tachycardic, hypertensive, confused, hallucinating, or seizes.*
- *A patient with chronic anxiety stops their medication and develops agitation, insomnia, autonomic instability, and seizures.*

What You Need to Memorize

- MOA: increase the **frequency** of GABA-A Cl^- channel opening.
- Alcohol withdrawal/delirium tremens are treated first-line with benzodiazepines.
- Status epilepticus: **IV lorazepam/diazepam** stops the seizure acutely; **phenytoin** is loaded afterward to prevent recurrence.
- Flumazenil is a competitive benzodiazepine-site antagonist for benzo intoxication.

Don't Miss

★ **Benzodiazepines increase GABA-A opening frequency; barbiturates increase duration.**

4. Amphetamines

Drug Class: monoamine release enhancers (amphetamine salts, methylphenidate)

How It's Tested

- *A 10-year-old boy leaves his seat, interrupts classmates, loses homework, forgets instructions, and finishes far fewer test questions than expected despite normal intelligence.*
- *A college student at a rave becomes euphoric, paranoid, **sweaty**, tremulous, **tachycardic**, hypertensive, and **mydriatic**.*

What You Need to Memorize

- MOA: **increase synaptic dopamine and norepinephrine**, especially by increasing presynaptic monoamine release.
- Stimulants are first-line for **ADHD**: atomoxetine is a selective NE reuptake inhibitor and clonidine/guanfacine are a_2-agonist alternatives.
- Amphetamine-induced psychosis is driven by increased presynaptic dopamine release.
- Hyperactivity is due more to dopamine dysregulation; inattention is due more to NE dysregulation.

Don't Miss

★ **Amphetamines increase monoamine release; cocaine blocks reuptake.**

5. Methotrexate

Drug Class: folate antimetabolite (antifolate, leucovorin rescue)

How It's Tested

- *A woman on a weekly immunomodulator for RA or psoriasis develops painful mouth ulcers, nausea/diarrhea, elevated liver enzymes, or **pancytopenia**.*
- *A stable ectopic pregnancy is treated medically with a drug that stops rapidly dividing trophoblast cells.*

What You Need to Memorize

- MOA: inhibits DHFR → dihydrofolate accumulates, THF falls → impaired thymidylate/purine synthesis and DNA replication.
- Rapidly dividing tissues are hit first: **GI mucosa** and bone marrow.
- Leucovorin/folinic acid is reduced folate that bypasses DHFR block for normal-cell rescue.

Don't Miss

★ **Leucovorin rescues MTX by donating one-carbon units for thymidylate synthesis; 5-FU blocks thymidylate synthase and is not rescued.**

6. Nitroglycerin / nitrates

Drug Class: nitrates (sublingual nitroglycerin, isosorbide)

How It's Tested

- *An older man gets exertional substernal pressure walking uphill, and a tiny sublingual tablet relieves the pain within minutes.*
- *A man treated for erectile dysfunction the night before chest pain becomes profoundly hypotensive after receiving another vasodilator.*

What You Need to Memorize

- MOA: donate NO → activate guanylyl cyclase → increased cGMP → myosin light-chain dephosphorylation → smooth muscle relaxation.
- Main angina effect = **venodilation → decreased preload/LVEDP → decreased myocardial O_2 demand**.
- PDE-5 inhibitors and nitrates both increase cGMP signaling → severe hypotension risk.
- Continuous exposure causes **tolerance**; a nitrate-free interval preserves response.

Don't Miss

★ **Acute angina relief is mainly from decreased preload/O_2 demand, not stronger contraction.**

7. Haloperidol / high-potency D2 blockers

Drug Class: high-potency typical antipsychotic (haloperidol; dopamine-blocking tic drugs)

How It's Tested

- *An agitated psychotic patient receives an ED antipsychotic and later develops fever, lead-pipe rigidity, confusion, unstable vitals, and very high CK.*
- *A child has repeated blinking or shoulder jerks plus grunting/throat-clearing sounds that improve when dopamine signaling is blocked.*

What You Need to Memorize

- MOA: block D_2 receptors; tested effects come from dopamine blockade in CNS pathways.
- **NMS** = **hyperthermia** + **lead-pipe rigidity** + altered mental status + autonomic instability + **high CK**; treat by stopping drug, cooling the patient, and **dantrolene**.
- High-potency typicals cause extrapyramidal symptoms/akathisia/parkinsonism; **diphenhydramine** or **benztropine** treats **dystonia/parkinsonism**.
- D2 blockade in the **tuberoinfundibular pathway** → increased prolactin → amenorrhea, galactorrhea, or breast tenderness.

Don't Miss

★ **Lead-pipe rigidity/high CK after a dopamine blocker = NMS; clonus/hyperreflexia after serotonergic drugs = serotonin syndrome.**

8. Thiazide diuretics

Drug Class: early DCT Na^+/Cl^- cotransporter inhibitors (hydrochlorothiazide, chlorthalidone)

How It's Tested

- *A hypertensive patient keeps forming calcium oxalate stones and needs a BP medication that also prevents recurrence.*
- *An older adult on a diuretic develops low Na^+, low K^+, **gout/hyperuricemia**, **hypercalcemia**, or metabolic alkalosis.*

What You Need to Memorize

- MOA: inhibit the Na^+/Cl^- cotransporter in the early distal convoluted tubule.
- Increase distal Ca^{2+} reabsorption → **decreased urinary Ca^{2+} excretion**.
- Thiazides increase proximal Na^+/lithium reabsorption → **lithium toxicity** with tremor, ataxia, or confusion.

Don't Miss

★ **For recurrent calcium stones, think thiazide; loops are the diuretics that increase urinary Ca^{2+} excretion.**

9. Leuprolide / GnRH agonists

Drug Class: continuous GnRH receptor agonists (leuprolide, goserelin)

How It's Tested

- *An older man with metastatic prostate cancer receives depot hormone therapy and briefly has worse bone pain or urinary symptoms before testosterone falls.*
- *A young child with central precocious puberty, or a patient with endometriosis, receives continuous hormone analog therapy that suppresses the reproductive axis over time.*

What You Need to Memorize

- MOA: continuous nonpulsatile GnRH receptor stimulation → pituitary receptor downregulation → decreased LH/FSH.
- Initial flare = transient rise in LH/FSH and sex hormones.
- Long-term effect = decreased testosterone/estrogen from pituitary desensitization; **chronic suppression can cause osteoporosis**.
- Prostate-cancer flare can be blocked with antiandrogen coverage such as bicalutamide or **flutamide**.

Don't Miss

★ **The early hormone rise is the expected flare before receptor downregulation, not treatment failure.**

10. Doxycycline / tetracyclines

Drug Class: 30S protein synthesis inhibitors (doxycycline, tetracycline, minocycline)

How It's Tested

- *A febrile 8-year-old hiker has severe headache, myalgias, and a rash that starts on wrists/ankles and spreads to palms/soles.*
- *A **teen on an acne antibiotic** gets a **severe sunburn-type rash**, **pill esophagitis**, or treatment failure after taking it with **milk**, **iron**, or **antacids**.*

What You Need to Memorize

- MOA: bind **30S** ribosomal subunit and **block aminoacyl-tRNA entry** at the A site.
- Divalent cations **chelate** tetracyclines → decreased absorption with **milk**, **iron**, calcium, magnesium, or **antacids**.
- Tested toxicities: photosensitivity, pill esophagitis/GI upset, **tooth discoloration**, and **impaired bone growth**.
- Gonorrhea is treated with ceftriaxone; **add doxycycline** when chlamydia has not been excluded.

Don't Miss

★ **RMSF is the classic exception where doxycycline is used despite the usual pediatric tetracycline concern.**

11. Aspirin

Drug Class: irreversible COX inhibitor (platelet COX inhibition, AERD)

How It's Tested

- *A patient with crushing substernal chest pain and ischemic ECG changes receives a chewable medication to limit platelet-driven thrombus growth.*
- *An asthmatic with chronic sinus disease/nasal polyps wheezes after taking an over-the-counter pain reliever.*

What You Need to Memorize

- MOA: **irreversibly inhibits COX**; in platelets this decreases thromboxane A_2 for the platelet lifespan.
- Acute MI benefit = **decreased platelet aggregation** and thrombus propagation.
- AERD mechanism: COX inhibition shifts arachidonic acid toward leukotrienes → bronchospasm.
- Salicylate toxicity = **tinnitus plus respiratory alkalosis (from hyperventilation)** and **anion-gap metabolic acidosis**; sodium bicarbonate traps ionized salicylate in urine.

Don't Miss

★ **Irreversible platelet COX inhibition is aspirin-specific; most NSAID platelet effects are reversible.**

12. NSAIDs

Drug Class: reversible COX inhibitors (ibuprofen, naproxen, indomethacin)

How It's Tested

- *A dehydrated athlete, rhabdomyolysis patient, or CKD-risk patient takes OTC pain relievers and **develops AKI**.*
- *A chronic pain patient using high-dose OTC analgesics develops **epigastric pain or ulcer bleeding**.*

What You Need to Memorize

- MOA: inhibit COX → **decreased prostaglandin synthesis**.
- Renal toxicity: **loss of afferent arteriole dilation** → decreased GFR.
- GI toxicity: **decreased mucus/bicarbonate protection** → ulcer/bleeding risk.
- **Indomethacin**/ibuprofen **close a PDA** by decreasing PGE_2.

Don't Miss

★ **NSAID AKI is an afferent-prostaglandin problem; ACE inhibitor creatinine rise is an efferent-angiotensin II problem.**

13. Warfarin

Drug Class: vitamin K epoxide reductase inhibitor (vitamin K antagonist, CYP interactions)

How It's Tested

- *A patient with atrial fibrillation starts an oral anticoagulant and **days** later develops **painful necrotic skin** on a breast, thigh, buttock, or other fatty area.*
- *A patient stable on **warfarin** starts **rifampin**, **phenytoin**, or **St John's wort** and the **INR falls**.*

What You Need to Memorize

- MOA: inhibits vitamin K epoxide reductase → decreased activation of factors II, VII, IX, X and proteins C/S.
- **Protein C has a short half-life → early hypercoagulable state** → skin necrosis.
- CYP induction increases warfarin metabolism → less warfarin → less anticoagulation.
- **Broad-spectrum antibiotics** increase INR by **killing gut bacteria** and **reducing vitamin K**.

Don't Miss

★ **Warfarin/rat poisoning bleeding requires PCC/FFP plus vitamin K when available.**

14. Beta-blockers

Drug Class: beta-adrenergic receptor antagonists (propranolol, metoprolol, atenolol, carvedilol)

How It's Tested

- *A patient with **tremor**, **palpitations**, anxiety, and tachycardia gets rapid relief from blocking adrenergic symptoms.*
- *An **asthmatic** treated for migraine/HTN starts **wheezing**, or a chronic user abruptly stops therapy and **rebounds with tachycardia or angina**.*

What You Need to Memorize

- MOA: blocks beta receptors; β_1 blockade lowers HR, contractility, and **renin/cAMP signaling in JG cells** and cardiomyocytes.
- **Nonselective** β-blockade can cause **bronchospasm** by blocking β_2 receptors and blunt adrenergic hypoglycemia signs.
- Propranolol treats **hyperthyroid adrenergic symptoms** and **essential tremor**; high doses **decrease peripheral T4→T3 conversion**.
- Overdose causes bradycardia, hypotension, and hypoglycemia; glucagon raises cardiac cAMP without needing β receptors.

Don't Miss

★ **In COPD/post-MI stems, choose β1-selective metoprolol over nonselective propranolol; abrupt chronic withdrawal can trigger rebound angina.**

15. SSRIs + serotonergic interactions

Drug Class: serotonin reuptake inhibitors (fluoxetine, sertraline, paroxetine; tramadol/linezolid interactions)

How It's Tested

- *A depressed patient receives tramadol, dextromethorphan, or linezolid, and then becomes febrile, sweaty, agitated, diarrheal, and **hyperreflexic with clonus**.*
- *A patient stopping phenelzine waits before starting sertraline because new monoamine oxidase must be synthesized.*

What You Need to Memorize

- MOA: SSRIs block serotonin reuptake; serotonergic combinations can overactivate serotonin receptors.
- Serotonin syndrome = mental status changes + autonomic instability + **neuromuscular hyperactivity**.
- C**yproheptadine** is a serotonin antagonist used for persistent serotonin syndrome.
- SSRIs commonly cause **sexual dysfunction**; **fluoxetine** is tested for **bulimia**.

Don't Miss

★ **Clonus/hyperreflexia favors serotonin syndrome; lead-pipe rigidity/high CK after antipsychotics favors NMS.**

16. Statins

Drug Class: HMG-CoA reductase inhibitors (atorvastatin, simvastatin, rosuvastatin)

How It's Tested

- *A post-MI patient starts a lipid drug that lowers LDL, CRP, and future vascular events.*
- *A patient on a cholesterol medication adds gemfibrozil or clarithromycin and develops proximal muscle pain, **dark urine**, or **very high CK**.*

What You Need to Memorize

- MOA: inhibit HMG-CoA reductase → decreased hepatic cholesterol synthesis → **increased LDL receptor expression**.
- Check baseline liver transaminases before starting statin therapy.
- **Ezetimibe blocks NPC1L1 cholesterol absorption** and can be used when statin myopathy limits therapy.

Don't Miss

★ **Dark urine or high CK with muscle soreness when taking a statin = rhabdomyolysis.**

17. Systemic glucocorticoids

Drug Class: intracellular steroid receptor agonists (prednisone, dexamethasone, hydrocortisone)

How It's Tested

- *A lupus patient diagnosed 10 years ago reports hyperglycemia, vertebral compression fracture/osteoporosis, or posterior subcapsular cataracts.*
- *An RA patient with swollen painful joints and prolonged morning stiffness receives prednisone for rapid symptom relief while slower drugs take effect.*

What You Need to Memorize

- **MOA: bind cytosolic glucocorticoid receptors → translocate to nucleus → alter gene transcription.**
- Chronic systemic therapy **causes osteoporosis** mainly by **decreasing osteoblast replication/differentiation**.
- Glucocorticoids suppress NF-kB, cause hyperglycemia, and impair leukocyte/neutrophil migration to inflammatory sites.

Don't Miss

★ **Steroid MOA is cytosolic/nuclear DNA-level transcription, not JAK, tyrosine kinase, or PLC signaling.**

18. Succinylcholine

Drug Class: depolarizing nicotinic ACh receptor agonist (depolarizing neuromuscular blocker)

How It's Tested

- *After intubation, a **patient remains unable to breathe for hours** despite the expected short duration of the paralytic.*
- *A patient with major burns or quadriplegia receives a paralytic and develops **hyperkalemia or ventricular fibrillation**.*

What You Need to Memorize

- MOA: nicotinic ACh receptor agonist at the NMJ → persistent depolarization → flaccid paralysis.
- Normal short duration is due to **plasma pseudocholinesterase metabolism**.
- Pseudocholinesterase deficiency → succinylcholine persists at NMJ and **causes prolonged apnea/paralysis**.

Don't Miss

★ **Prolonged apnea = pseudocholinesterase deficiency; sudden arrest after burns/crush/denervation = hyperkalemia.**

19. Digoxin

Drug Class: Na^+/K^+ ATPase inhibitor (cardiac glycoside)

How It's Tested

- *An HFrEF/AF patient with renal decline develops nausea, confusion, **yellow-green vision**, or bradycardia.*
- *A patient on digoxin develops toxicity after hypokalemia from a loop or thiazide diuretic.*

What You Need to Memorize

- MOA: inhibits Na^+/K^+ ATPase → increased intracellular Na^+ → decreased Na^+/Ca^{2+} exchange → increased intracellular Ca^{2+}.
- Increases contractility and increases vagal tone to slow AV nodal conduction.
- Renal clearance decline and hypokalemia increase toxicity risk.

Don't Miss

★ **Hypokalemia increases toxicity because K^+ and digoxin compete at the Na^+/K^+ ATPase.**

20. Spironolactone

Drug Class: mineralocorticoid receptor antagonist (aldosterone antagonist)

How It's Tested

- *A man treated for edema/HF develops hyperkalemia with breast tenderness, erectile dysfunction, and decreased libido.*
- *A patient with resistant hypertension, low K^+, low renin, and high aldosterone receives **potassium-sparing** therapy.*

What You Need to Memorize

- MOA: blocks mineralocorticoid receptors in late DCT/collecting duct → **decreased Na^+ reabsorption and decreased K^+/H^+ secretion**.
- Major tested toxicity: hyperkalemia, especially with ACEi or high K^+.
- Antiandrogen effect → **gynecomastia**, breast tenderness, erectile dysfunction, and decreased libido.
- Aldosterone antagonism improves survival in HFrEF and treats primary hyperaldosteronism or cirrhotic ascites.

Don't Miss

★ **Hyperkalemia plus gynecomastia/sexual dysfunction points to spironolactone.**

21. Desmopressin

Drug Class: V_2 receptor agonist (DDAVP, synthetic ADH analog)

How It's Tested

- *After head trauma or pituitary surgery, a patient has extreme thirst and liters of dilute urine; after a synthetic hormone, urine becomes concentrated.*
- *A patient with hemophilia A or von Willebrand disease has bleeding controlled by releasing stored vWF/factor VIII.*

What You Need to Memorize

- MOA: V_2 receptor activation in collecting duct → **cAMP/PKA** → aquaporin-2 insertion → increased free-water reabsorption.
- Normal V_2 response = decreased urine volume and increased urine osmolality; renal papillary necrosis can make urine unchanged despite ADH.
- **Central DI responds to desmopressin with ↑ urine osmolality; nephrogenic DI does not because the kidney cannot respond to ADH.**
- Also **releases vWF and factor VIII from endothelial storage sites** in vWD/hemophilia A.

Don't Miss

★ **Desmopressin helps central DI and vWD/hemophilia A; nephrogenic DI will not concentrate urine because the kidney cannot respond.**

22. Amiodarone

Drug Class: K^+, Na^+, Ca^{2+}, and beta blockade (class III antiarrhythmic with multichannel effects)

How It's Tested

- *A patient considered for long-term rhythm control needs baseline thyroid testing before starting an iodine-containing antiarrhythmic.*
- *An antiarrhythmic **prolongs QT but has low torsades risk** while also slowing sinus rate, prolonging PR, and widening QRS.*

What You Need to Memorize

- MOA: primarily **blocks K^+ channels; also blocks Na^+ channels, Ca^{2+} channels, and beta receptors.**
- Iodine content can cause hypo- or hyperthyroidism.
- Toxicities include **pulmonary fibrosis**, hepatotoxicity, **corneal deposits**, blue-gray skin, and photosensitivity.

Don't Miss

★ **New lung symptoms or thyroid labs in a patient on heart meds should make amiodarone toxicity jump out.**

23. TMP-SMX

Drug Class: sequential bacterial folate blockade (trimethoprim-sulfamethoxazole)

How It's Tested

- *An AIDS/immunocompromised patient has fever, dyspnea, dry cough, bilateral infiltrates, and BAL positive for **Pneumocystis jirovecii**.*
- *A patient treated with TMP-SMX for UTI develops fever, rash, eosinophilia, pyuria, and rising creatinine a few days later.*

What You Need to Memorize

- MOA: sulfamethoxazole inhibits dihydropteroate synthase; trimethoprim inhibits bacterial DHFR.
- TMP-SMX treats PCP and cystitis.
- AIN: fever + rash + eosinophilia + pyuria + rising creatinine after sulfonamide/antibiotic exposure.
- Trimethoprim blocks epithelial Na+ channels in the collecting duct → decreased K+ secretion → hyperkalemia.

Don't Miss

★ **PCP treatment of choice is TMP-SMX: two-step folate blockade, not ribosomal protein-synthesis inhibition.**

24. Fluconazole / azoles

Drug Class: fungal ergosterol synthesis inhibitors (fluconazole, voriconazole, clotrimazole)

How It's Tested

- *A patient stable on methadone or warfarin starts an antifungal and then becomes overly sedated or has an unexpectedly high INR.*
- *An immunocompromised patient has invasive mold disease with septate hyphae branching at acute angles.*

What You Need to Memorize

- MOA: **inhibit fungal lanosterol 14-α-demethylase → decreased ergosterol synthesis**.
- Azoles inhibit CYP enzymes → methadone, warfarin, and statin-type interaction risk.
- Topical azoles such as clotrimazole treat dermatophyte/tinea pedis by inhibiting ergosterol synthesis.
- Invasive Aspergillus points to **voriconazole**; azole resistance can occur by mutation of lanosterol 14-α-demethylase.

Don't Miss

★ **If the answer is 'binds ergosterol and forms pores,' that is amphotericin B, not fluconazole.**

25. Albuterol / β2 agonists

Drug Class: β_2 adrenergic agonists (albuterol, terbutaline)

How It's Tested

- *An asthma patient with wheezing gets rapid relief from an inhaled rescue medication but needs it more often as response fades.*
- *A patient with severe hyperkalemia receives a nebulized medication in the ED, and serum K^+ falls as potassium shifts into cells.*

What You Need to Memorize

- MOA: β_2 agonism → G_s → **increased cAMP** → smooth muscle relaxation.
- **Airway β_2 activation relaxes bronchial smooth muscle** → bronchodilation.
- β_2 activation **stimulates Na^+/K^+ ATPase** shifting K^+ intracellularly and can cause tremor/tachycardia.
- **Tachyphylaxis** comes from **β_2** receptor phosphorylation/**internalization**.

Don't Miss

★ **For airway smooth muscle relaxation, the receptor affected is β_2 not β_1.**