PHARMACOLOGY · STUDY GUIDE

Exam 4 Study Guide

This guide covers chapters 7 and 8. Chapter 7 is the receptor map that makes the rest of pharmacology predictable: learn which receptor a drug hits and you know its effects. Chapter 8 is endocrine drugs, where negative feedback and a decimal point drive most of the errors. 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 7 · sympathetic and parasympathetic divisions, the receptor map, epinephrine and the other sympathomimetics, clonidine, vasopressor extravasation, alpha and beta blockers, cholinergic agonists, AChE inhibitors, cholinergic and myasthenic crisis, anticholinergics and burden, and depolarizing versus non-depolarizing neuromuscular blockers.
- Chapter 8 · hormone chemistry and negative feedback, pituitary drugs and desmopressin, levothyroxine, methimazole and PTU, corticosteroid potency, adverse effects, tapering and stress dosing, insulin onset, peak and duration, mixing, hypoglycemia, metformin, the other oral classes and glucagon.

CHAPTER 7

Autonomic Drugs

Read it in full: **Chapter 7 in the textbook**. Practice it in the lab: **Pick a Receptor, See the Response** and **Paralyzed Two Ways**.

What to know

The **sympathetic** division is fight or flight: faster heart, higher pressure, open airways, dilated pupils, glucose released, blood away from gut and bladder. The **parasympathetic** division is rest and digest: slower heart, small pupils, active gut, bladder emptying. All autonomic ganglia use acetylcholine on **nicotinic** receptors. At the organ, parasympathetic nerves use acetylcholine on

muscarinic receptors and most sympathetic nerves use norepinephrine on adrenergic receptors. Two exceptions: the adrenal medulla releases epinephrine into the blood, and sweat glands are sympathetic but cholinergic, which is why anticholinergics make patients hot and dry.

The receptor map: **alpha-1** constricts vessels, dilates pupils and tightens the bladder neck. **Alpha-2** is a presynaptic brake that lowers norepinephrine release and blood pressure. **Beta-1** is the heart ("one heart"): rate, contractility, conduction, renin. **Beta-2** is the lungs ("two lungs"): bronchodilation, vasodilation, uterine relaxation. **Beta-3** relaxes the detrusor. **Muscarinic** gives SLUDGE. Blocking one division looks like stimulating the other.

Epinephrine is dose dependent: beta effects at low doses, alpha-1 vasoconstriction at high doses. In anaphylaxis it does three jobs: alpha-1 raises pressure and reduces edema, beta-1 supports output, beta-2 opens bronchi and stabilizes mast cells. The book gives the adult dose as 0.3 to 0.5 mg IM of the 1 mg/mL (1:1,000) concentration into the lateral thigh, repeated every 5 to 15 minutes, and 0.01 mg/kg for a child to a 0.3 to 0.5 mg maximum. The 0.1 mg/mL (1:10,000) syringe is the IV arrest concentration; swapping them is the classic tenfold error.

Norepinephrine is the first-line vasopressor in septic shock; extravasation causes necrosis, so stop, aspirate, leave the catheter and infiltrate **phentolamine**. Dobutamine is beta-1 selective for heart failure. Phenylephrine is alpha-1 selective. **Albuterol** is a short-acting beta-2 rescue drug that causes tremor, tachycardia and hypokalemia; long-acting beta-2 agonists are never rescue drugs and in asthma are always paired with an inhaled steroid. **Clonidine** is an alpha-2 agonist that lowers blood pressure and must never be stopped abruptly (rebound hypertensive crisis).

Alpha-1 blockers (-osin) relax vessels and the bladder outlet for hypertension and BPH. First-dose orthostatic syncope is the hazard: give at bedtime, rise slowly. Tamsulosin is uroselective; flag it before cataract surgery.

Beta blockers (-olol) slow rate, weaken contraction and cut renin. They lower BP, treat angina, control rate and improve survival after MI. In heart failure with reduced ejection fraction, only **metoprolol succinate, bisoprolol and carvedilol** have a mortality benefit; atenolol and metoprolol tartrate do not. Cardioselective agents (metoprolol, atenolol, bisoprolol, esmolol) mostly spare beta-2; nonselective agents (propranolol, nadolol, timolol) risk bronchospasm.

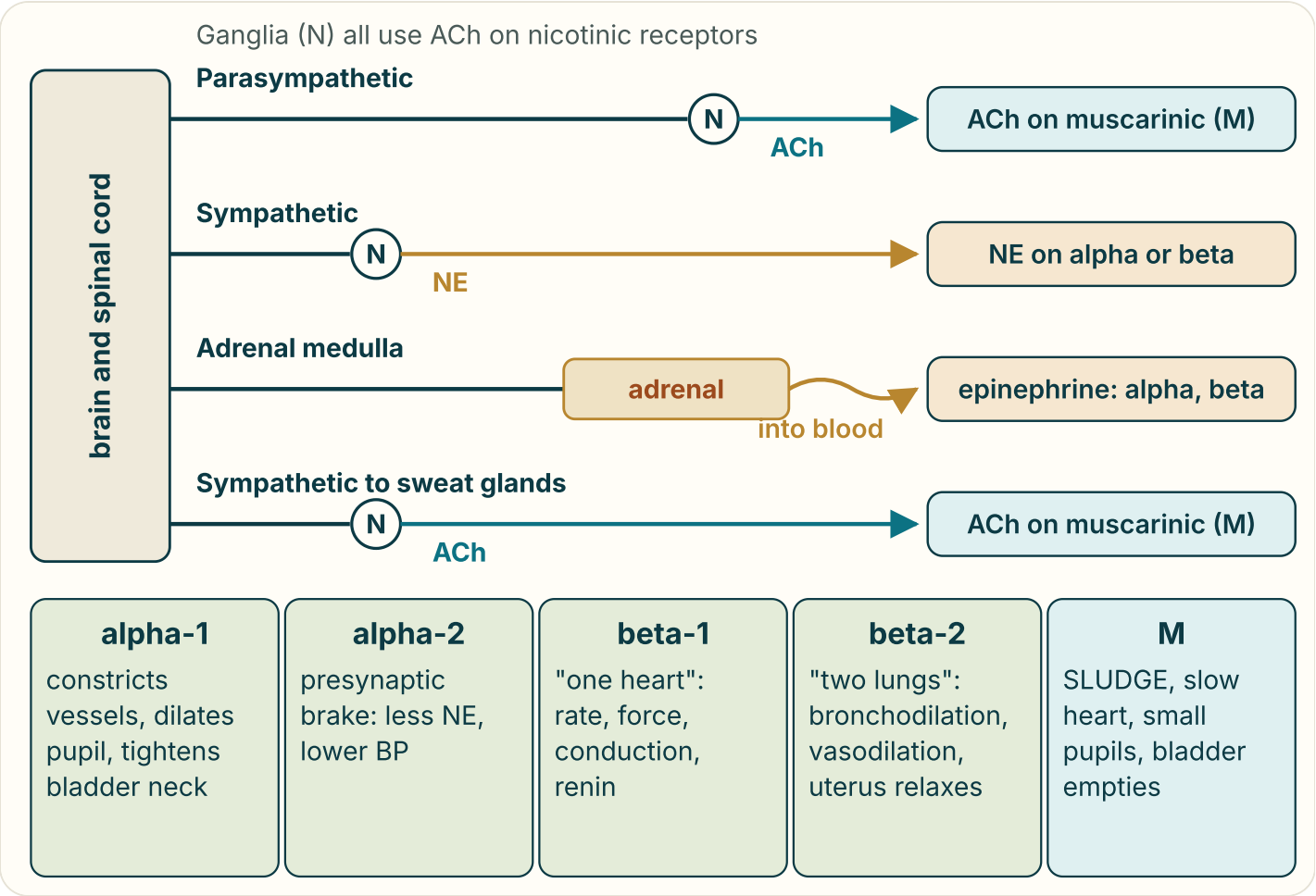
Carvedilol and labetalol also block alpha-1. Never stop abruptly; they **mask hypoglycemia** except sweating; take an apical pulse for a full minute and hold for HR under 60 or low SBP. Overdose antidote: **glucagon**. Timolol eye drops act systemically; teach punctal occlusion.

Cholinergic agonists make SLUDGE plus bradycardia, bronchoconstriction and miosis. Bethanechol treats non-obstructive urinary retention (never with obstruction). Pilocarpine treats glaucoma and dry mouth. **AChE inhibitors** make acetylcholine last: pyridostigmine for myasthenia gravis (30 to 60 minutes before meals), donepezil for Alzheimer disease (symptomatic help, not a cure), neostigmine for reversing non-depolarizing blockade (always with glycopyrrolate or atropine).

Cholinergic crisis is SLUDGE plus weakness and respiratory failure. **Myasthenic crisis** is weakness without SLUDGE. Airway first in both. Organophosphate poisoning needs **atropine** (muscarinic effects) plus **pralidoxime** (reactivates the enzyme); atropine alone cannot fix muscle weakness.

Anticholinergics are the mirror image: dry as a bone, red as a beet, blind as a bat, mad as a hatter, hot as a hare. Atropine for bradycardia is 1 mg IV pushed rapidly, every 3 to 5 minutes to 3 mg. Ipratropium and tiotropium bronchodilate in COPD; oxybutynin quiets the bladder; scopolamine prevents motion sickness; benztropine treats EPS. **Anticholinergic burden** stacks across antihistamines, TCAs and antipsychotics and causes confusion, retention and falls in older adults, plus heat stroke risk. Physostigmine is only for pure antimuscarinic toxicity with a normal QRS; it is contraindicated with a TCA or a QRS over 100 ms.

Neuromuscular blockers act at the nicotinic end plate and give no sedation, analgesia or amnesia. Sedation and analgesia run first, the airway is secured, eyes are protected, and you keep talking to the patient. **Succinylcholine** depolarizes: fasciculations, onset under a minute, 5 to 10 minute duration, no reversal, hyperkalemia risk (burns, crush, immobility), a boxed warning for children with undiagnosed myopathy, and malignant hyperthermia (dantrolene). **Rocuronium, vecuronium, cisatracurium** compete: no fasciculations, longer action, reversed by neostigmine plus glycopyrrolate or by sugammadex. Monitor with **train-of-four**. Aminoglycosides, magnesium, hypothermia and acidosis prolong blockade.



The receptor map. Know the transmitter at each junction and what each receptor does, and every autonomic drug follows: blocking one division looks like stimulating the other.

LOCK THESE IN

- One heart (beta-1), two lungs (beta-2); alpha-1 constricts; alpha-2 lowers BP.
- Anaphylaxis: epinephrine 0.3 to 0.5 mg IM of 1 mg/mL, lateral thigh; 0.1 mg/mL is the IV arrest syringe.
- Vasopressor extravasation: phentolamine. Beta blocker overdose: glucagon.
- Alpha blockers: first dose at bedtime. Beta blockers: never stop abruptly; mask hypoglycemia.
- HFrEF beta blockers: metoprolol succinate, bisoprolol, carvedilol only.
- Organophosphates: atropine plus pralidoxime.
- Anticholinergic toxidrome: dry, red, blind, mad, hot. No physostigmine with a TCA.
- Succinylcholine: no reversal, hyperkalemia, malignant hyperthermia. Non-depolarizers: neostigmine plus glycopyrrolate or sugammadex.

Key terms and comparisons: the receptor map

Receptor	Location	Stimulation causes	Drug example
Alpha-1	Vessels, pupil, bladder neck	Vasoconstriction, mydriasis, retention	Phenylephrine (agonist); prazosin (blocker)
Alpha-2	Presynaptic, brainstem	Less NE release, lower BP	Clonidine
Beta-1	Heart, kidney	Faster, stronger beat; renin	Dobutamine; metoprolol blocks
Beta-2	Lungs, vessels, uterus, liver	Bronchodilation, vasodilation	Albuterol; propranolol blocks
Beta-3	Detrusor, fat	Bladder relaxation	(bladder drugs)
Muscarinic	Heart, gut, bladder, glands, eye	SLUDGE, bradycardia, miosis	Bethanechol; atropine blocks
Nicotinic NN	Ganglia, adrenal medulla	Ganglionic transmission	(ganglia)
Nicotinic NM	Skeletal muscle end plate	Contraction	Rocuronium blocks

Crises and antidotes

Problem	Clue	Treatment
Anaphylaxis	Hypotension, wheeze, swelling	Epinephrine IM first
Vasopressor extravasation	Pale, cool, painful site	Phentolamine infiltration
Beta blocker overdose	Bradycardia, hypotension not helped by atropine	Glucagon
Cholinergic crisis	Weakness plus SLUDGE	Airway; atropine (+ pralidoxime for organophosphates)
Anticholinergic toxicity	Dry, hot, red, confused	Supportive; physostigmine only if pure and QRS normal
Malignant hyperthermia	Rising end-tidal CO_2, rigidity	Dantrolene
Non-depolarizing block	Train-of-four	Neostigmine + glycopyrrolate, or sugammadex

Worked examples

WORKED EXAMPLE 1 · AN EPINEPHRINE CONCENTRATION CHECK

A 20 kg child needs IM epinephrine for anaphylaxis. Using the book's 0.01 mg/kg dose, find the dose and the volume of the 1 mg/mL concentration. Then show what goes wrong if the 0.1 mg/mL syringe is grabbed. (Practice calculation; real doses are ordered and double-checked.)

STEP 1 · DOSE

20 kg × 0.01 mg/kg = **0.2 mg**. That is under the 0.3 to 0.5 mg maximum.

STEP 2 · VOLUME AT 1 MG/ML

0.2 mg ÷ 1 mg/mL = **0.2 mL**.

STEP 3 · THE WRONG SYRINGE

At 0.1 mg/mL, 0.2 mL holds only 0.02 mg, a tenth of the dose. To get 0.2 mg you would need 2 mL, which is the wrong product for an IM thigh injection.

THE LESSON

Concentration is part of the order. Verify it with an independent double-check every time.

WORKED EXAMPLE 2 · HOLDING A BETA BLOCKER

Metoprolol is due at 0900. Mr. Osei's apical pulse, counted for a full minute, is 52; his BP is 118/70. He also has type 1 diabetes. What do you do and teach?

STEP 1 · APPLY THE PARAMETER

Heart rate under 60 means **hold** the dose and notify the prescriber. Do not give it and chart the vitals.

STEP 2 · DO NOT STOP IT AT HOME

Teach that he must never stop it abruptly; rebound tachycardia, hypertension, angina or MI can follow. Any stop is a taper.

STEP 3 · TEACH HYPOGLYCEMIA

Beta blockade hides tremor and palpitations. Sweating still occurs. Check glucose on a schedule rather than trusting how he feels.

WORKED EXAMPLE 3 · ATROPINE FOR BRADYCARDIA

The book gives atropine for symptomatic bradycardia as 1 mg IV pushed rapidly, repeated every 3 to 5 minutes to a 3 mg maximum. How many doses fit, and over about how long?

STEP 1 · COUNT DOSES

3 mg ÷ 1 mg = **3 doses**.

STEP 2 · TIME

The two gaps between three doses take 2 × 3 = 6 to 2 × 5 = 10 minutes, so about **6 to 10 minutes** from first to last dose.

WATCH THE TECHNIQUE

A slow push or underdose can paradoxically worsen bradycardia.

WORKED EXAMPLE 4 · WHICH CRISIS IS IT?

Ms. Adeyemi has myasthenia gravis and takes pyridostigmine. She arrives weak and short of breath, drooling, tearing, with cramping and pinpoint pupils. What is happening?

STEP 1 · AIRWAY FIRST

Both crises can cause respiratory failure. Support ventilation before anything else.

STEP 2 · LOOK FOR SLUDGE

Drooling, tearing, cramping and miosis are cholinergic signs.

STEP 3 · NAME IT

This is **cholinergic crisis** from too much drug. Myasthenic crisis would show weakness without SLUDGE.

STEP 4 · EXPECT

Hold the pyridostigmine and expect atropine for the muscarinic effects.

TRAPS PEOPLE FALL INTO

- **Giving an antihistamine first in anaphylaxis.** Epinephrine IM is first; antihistamines and steroids are adjuncts.
- **Using the 0.1 mg/mL syringe for an IM dose.** That is the IV arrest concentration. Use 1 mg/mL for IM.
- **Thinking clonidine is a stimulant because it is an agonist.** Alpha-2 is a presynaptic brake; it lowers BP.
- **Assuming any beta blocker helps heart failure.** Only metoprolol succinate, bisoprolol and carvedilol.
- **Giving bethanechol for retention from an obstruction.** Squeezing against a blocked outlet risks rupture.
- **Giving physostigmine when a TCA is on board.** It can cause seizures and arrest. Treat supportively.
- **Trying to reverse succinylcholine with neostigmine.** It prolongs the block. Ventilate and wait.

Chapter 7 self-test · ten items

1. Why do anticholinergic drugs cause hot, dry skin?

A. Anticholinergics raise the body's thermostat directly

B. Anticholinergics stimulate alpha-1 receptors in skin

C. Sweat glands use norepinephrine on beta-2

D. Sweat glands are sympathetic but use acetylcholine

D. Sweat glands are sympathetic but use acetylcholine Sweat glands are the cholinergic exception in the sympathetic division. Block muscarinic receptors and sweating stops.

2. Which receptor, when stimulated, mainly causes bronchodilation?

A. Muscarinic
B. Beta-2
C. Beta-1
D. Alpha-1

B. Beta-2 "Two lungs": beta-2 relaxes bronchial smooth muscle. Muscarinic stimulation constricts the airways.

3. What is the first-line route and site for epinephrine in anaphylaxis?

A. IM into the lateral thigh
B. IV push of the 1 mg/mL
C. SubQ into the upper arm
D. IM into the deltoid area

A. IM into the lateral thigh The vastus lateralis absorbs fastest and most reliably. Concentrated 1 mg/mL is never pushed IV.

4. A norepinephrine site turns pale, cool and painful. What drug is infiltrated?

A. Atropine
B. Protamine
C. Phentolamine
D. Glucagon

C. Phentolamine Phentolamine is an alpha blocker that reopens the constricted vessels after vasopressor extravasation.

5. Why is clonidine never stopped abruptly?

A. Acute bronchospasm attack
B. Serotonin syndrome onset
C. Rebound hypertensive crisis
D. Sudden severe hypoglycemia

C. Rebound hypertensive crisis Clonidine suppresses norepinephrine release. Stopping it suddenly releases the brake and pressure surges.

6. A patient with asthma is prescribed a beta blocker. Which is the safer choice?

A. Propranolol
B. Timolol
C. Nadolol
D. Metoprolol

D. Metoprolol Metoprolol is beta-1 selective at usual doses. The other three are nonselective and can cause bronchospasm.

7. Which sign of hypoglycemia usually remains on a beta blocker?

A. Sweating
B. Palpitations
C. Anxiety
D. Tremor

A. Sweating Sweat glands are cholinergic, so diaphoresis survives beta blockade. Tremor, palpitations and anxiety are beta mediated.

8. A farmworker exposed to pesticide has secretions, bradycardia and weakness. Which pair treats it?

A. Physostigmine and atropine
B. Atropine and pralidoxime
C. Naloxone and flumazenil
D. Neostigmine and glycopyrrolate

B. Atropine and pralidoxime Atropine blocks the muscarinic effects; pralidoxime reactivates acetylcholinesterase before the bond ages.

9. Which reverses rocuronium by encapsulating it?

A. Pralidoxime
B. Neostigmine
C. Dantrolene
D. Sugammadex

D. Sugammadex Sugammadex binds rocuronium or vecuronium directly. Neostigmine works by raising acetylcholine.

10. Before succinylcholine, which history raises the greatest concern?

A. A large burn ten days ago
B. A mild cold last month
C. Seasonal pollen allergy
D. Allergy to penicillin

A. A large burn ten days ago Burns, crush injury and immobility upregulate receptors, so succinylcholine can cause a potassium surge and cardiac arrest.

Endocrine Drugs

Read it in full: **Chapter 8 in the textbook**. Practice it in the lab: **Long-Term Steroids & Why You Taper** and **Insulin Curves**.

What to know

Hormones act at tiny concentrations: levothyroxine is dosed in micrograms, so a decimal error is tenfold and a mcg to mg mix-up is a thousandfold error. **Lipid-soluble** hormones (thyroid, steroids) enter the cell, change gene transcription, act over hours to days and can be swallowed. **Peptide** hormones (insulin, growth hormone, glucagon, vasopressin) act on the surface in minutes and are digested, which is why insulin cannot be a pill.

Negative feedback runs the chapter. The loop cannot tell your hormone from the patient's own. Daily prednisone suppresses CRH and ACTH, and the adrenal cortex atrophies; stop abruptly and adrenal crisis follows. A TSH is the pituitary's opinion of whether the levothyroxine dose is right.

Pituitary drugs replace or suppress. Somatropin replaces growth hormone (raises glucose; report hip or knee pain). Octreotide suppresses GH and treats variceal bleeding (glucose swings, gallstones). Cabergoline lowers prolactin (first dose at bedtime). **Desmopressin** is synthetic ADH for diabetes insipidus; its danger is water intoxication and **hyponatremia**, so monitor sodium, daily weight (1 kg = 1 L), I&O and neuro status. Oxytocin is high-alert, always on a pump. DI and SIADH are mirror images.

Levothyroxine (T4) is converted to T3 in tissues, has a half-life of about 7 days and a narrow index. Take it on an empty stomach with water, 30 to 60 minutes before breakfast, same time daily; separate calcium, iron, multivitamins and antacids by at least 4 hours; stay on one product. Recheck TSH 6 to 8 weeks after a change. Over-replacement looks like hyperthyroidism and in older adults risks atrial fibrillation and osteoporosis, so older and cardiac patients start low. It is lifelong, and needs rise in pregnancy.

Methimazole is first-line for hyperthyroidism. **PTU** is for the first trimester, thyroid storm (it also blocks T4 to T3 conversion) and methimazole intolerance, and carries a boxed warning for hepatotoxicity. Both take weeks and both can cause **agranulocytosis**: sore throat, fever or mouth

ulcers means hold the drug and get a CBC. Iodine solution is given 1 hour after the thionamide, diluted, through a straw. Radioactive iodine is not used in pregnancy or lactation. Propranolol treats symptoms only.

Corticosteroids blend **glucocorticoid** (glucose up, inflammation and immunity down) and **mineralocorticoid** (sodium and water kept, potassium lost) activity. Equivalent doses: hydrocortisone 20 mg = prednisone 5 mg = methylprednisolone 4 mg = dexamethasone 0.75 mg. Hydrocortisone replaces adrenal function; dexamethasone is nearly pure glucocorticoid for cerebral edema; fludrocortisone is chosen for mineralocorticoid effect. Prednisone is a prodrug activated by the liver.

Steroid adverse effects are cortisol overdone: hyperglycemia (diabetics need more insulin), infection risk with masked signs, muscle wasting, thin skin, poor healing, osteoporosis, cushingoid changes, mood changes, GI ulcer (worse with NSAIDs), cataracts, glaucoma, growth suppression, fluid retention, hypertension and hypokalemia. Check glucose, weight, BP and potassium; give with food in the morning. After more than about 2 to 3 weeks, **taper**; stopping abruptly causes adrenal crisis (hypotension, shock, vomiting, hypoglycemia, hyponatremia; potassium usually normal). Increase the dose during surgery, trauma or illness (stress dosing). Treat crisis with IV hydrocortisone and fluids.

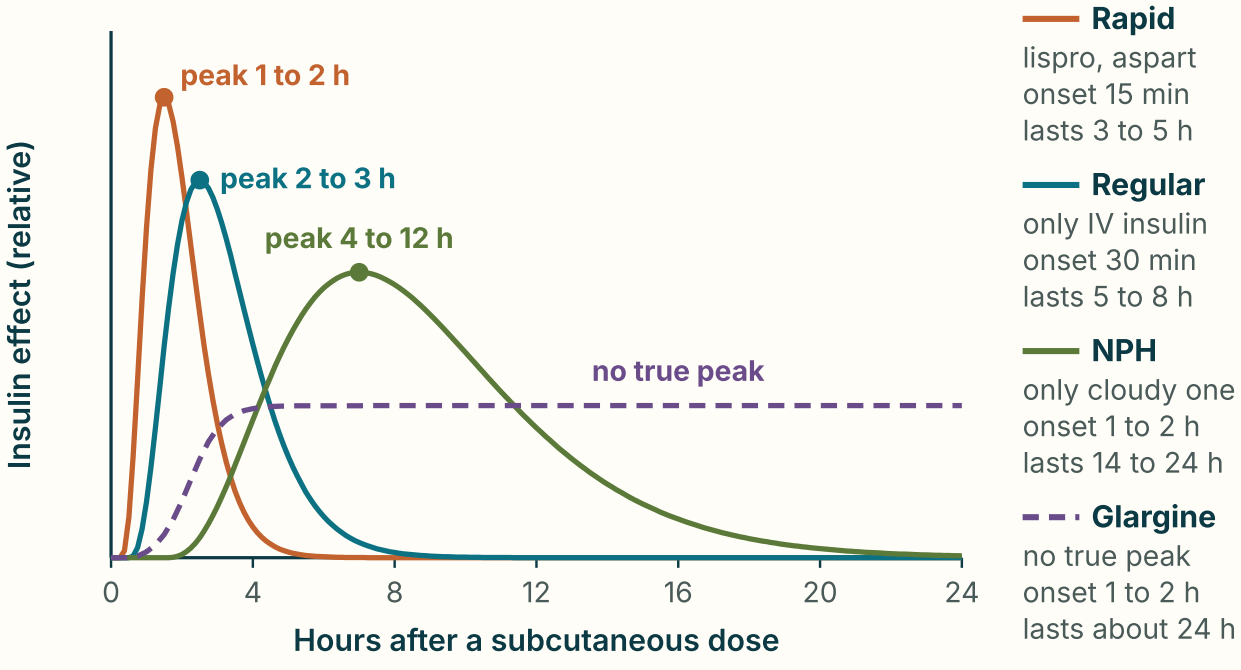
Insulin is high-alert: insulin syringe or pen only, never "U", independent double-check, read the label. Inject subcutaneously, abdomen fastest; rotate within one area; do not massage. In-use vials are generally discarded after 28 days. Know the **peaks**, because the peak is when the patient goes low: rapid (lispro, aspart, glulisine) onset about 15 min, peak 1 to 2 h; regular onset about 30 min, peak 2 to 3 h, the only IV insulin; NPH onset 1 to 2 h, peak 4 to 12 h, the only cloudy insulin (roll, never shake); glargine and detemir have no true peak; degludec lasts over 42 h. Never mix long-acting insulins. Draw **clear before cloudy**.

Hypoglycemia is under 70 mg/dL. Early signs are adrenergic (shaky, sweaty, fast heart, hungry); later signs are neuroglycopenic (confusion, slurred speech, seizure). Beta blockers hide the early signs. If awake and able to swallow: **rule of 15**, 15 g of fast carbohydrate, wait 15 minutes, recheck, repeat, then a snack if the meal is over an hour away. If not: IV D50 or glucagon. The

most common inpatient cause is rapid-acting insulin with no meal; match insulin to the tray, not the clock.

Metformin is first-line: it lowers liver glucose output, reduces absorption and improves sensitivity. It does not squeeze the pancreas, so alone it does not cause hypoglycemia. Take with food; check B12. Lactic acidosis is the boxed warning; avoid at eGFR under 30. With IV contrast, it continues at eGFR 30 or more without AKI, and is held at the study and for 48 hours only if eGFR is under 30, AKI is present or suspected, or the study is intra-arterial with renal exposure.

One alert per class: **sulfonylureas** (glipizide, glyburide) cause hypoglycemia; **meglitinides**: skip the meal, skip the dose; **pioglitazone**: heart failure boxed warning; **DPP-4 inhibitors**: pancreatitis; **GLP-1 agonists**: nausea, weight loss, thyroid C-cell boxed warning; **SGLT2 inhibitors**: genital infections and euglycemic DKA, with heart and kidney benefit; **acarbose**: treat a low with glucose, not sucrose. **Glucagon** (IM, SC or intranasal, works in 10 to 15 min) empties liver glycogen, fails without stores, causes vomiting (turn the patient), and is temporary, so follow with oral carbohydrate.



Insulin peaks predict hypoglycemia. Match rapid insulin to the meal tray, and expect lows at each product's peak; glargine is flat basal coverage.

LOCK THESE IN

- Negative feedback: exogenous hormone suppresses the gland. Taper steroids after more than 2 to 3 weeks.
- Desmopressin danger: hyponatremia. 1 kg weight = 1 L water.
- Levothyroxine: empty stomach 30 to 60 min before breakfast; binders 4 h apart; TSH in 6 to 8 weeks.
- Methimazole and PTU: sore throat and fever = hold and CBC. PTU: first trimester, storm, liver warning.
- Hydrocortisone 20 = prednisone 5 = methylprednisolone 4 = dexamethasone 0.75 mg.
- Peaks: rapid 1 to 2 h, regular 2 to 3 h, NPH 4 to 12 h; glargine no peak. Regular is the only IV insulin.
- Hypoglycemia under 70: rule of 15 if awake; D50 or glucagon if not.
- Metformin: no hypoglycemia alone; eGFR under 30 avoid; lactic acidosis warning.

Insulin profiles

Class	Examples	Onset / peak / duration	Nursing alert
Rapid	lispro, aspart, glulisine	About 15 min / 1 to 2 h / 3 to 5 h	Give with the meal in front of the patient
Short	regular	About 30 min / 2 to 3 h / 5 to 8 h	30 min before meal; only IV insulin
Intermediate	NPH	1 to 2 h / 4 to 12 h / 14 to 24 h	Cloudy; roll; nocturnal lows
Long	glargine, detemir	1 to 2 h / no true peak / about 24 h	Never mix; never IV
Ultra-long	degludec	1 h / no peak / over 42 h	Steady state about 3 days
Premixed	70/30, 75/25	Both profiles	Meals on time

Key terms and comparisons

Drug	Key fact	Nursing alert
Desmopressin	Synthetic ADH	Sodium, weight, neuro checks
Levothyroxine	T4, half-life about 7 days	Empty stomach; TSH 6 to 8 weeks
Methimazole	First-line thionamide	Agranulocytosis; not first trimester
PTU	Blocks T4 to T3 too	Hepatotoxicity boxed warning
Prednisone	Prodrug; 5 mg reference	Taper; morning with food
Dexamethasone	0.75 mg; little salt retention	Cerebral edema; glucose spikes
Fludrocortisone	Mineralocorticoid	BP, edema, potassium
Metformin	Biguanide	eGFR, B12, lactic acidosis
Glipizide	Sulfonylurea	Hypoglycemia
Empagliflozin	SGLT2 inhibitor	Genital infections; euglycemic DKA
Semaglutide	GLP-1 agonist	Nausea; C-cell warning
Glucagon	Mobilizes glycogen	Turn patient; follow with carbs

Worked examples

WORKED EXAMPLE 1 · STEROID EQUIVALENCE

A patient on prednisone 40 mg daily is switched to another steroid. Using the book's equivalents, what is the matching dose of hydrocortisone, methylprednisolone and dexamethasone? (Practice calculation.)

STEP 1 · FIND THE MULTIPLE

40 ÷ 5 = 8 prednisone "units".

STEP 2 · MULTIPLY EACH EQUIVALENT

Hydrocortisone 8 × 20 = **160 mg**. Methylprednisolone 8 × 4 = **32 mg**. Dexamethasone 8 × 0.75 = **6 mg**.

REMEMBER

The doses match glucocorticoid effect, not salt retention. Dexamethasone carries almost no mineralocorticoid effect; hydrocortisone carries the most.

WORKED EXAMPLE 2 · PREDICTING THE LOW

Lispro is given at 0730 with breakfast. NPH is given at 2200. Using the book's peaks, when is each patient most likely to go low?

STEP 1 · LISPRO

Peak 1 to 2 h: 0730 + 1 h = 0830; + 2 h = 0930. Highest risk **0830 to 0930**.

STEP 2 · NPH

Peak 4 to 12 h: 2200 + 4 h = 0200; + 12 h = 1000. Highest risk **0200 to 1000**, overnight into morning.

STEP 3 · PLAN

Check that the breakfast tray is eaten before giving lispro; plan an overnight glucose check and a bedtime snack per orders for the NPH patient.

WORKED EXAMPLE 3 · THE RULE OF 15

Mr. Lindgren, alert and able to swallow, has a glucose of 58 mg/dL at 1100. Lunch arrives at 1300. Walk through the response.

STEP 1 · TREAT

Give 15 g of fast carbohydrate: 4 oz of juice or regular soda, or 3 to 4 glucose tablets.

STEP 2 · WAIT AND RECHECK

Wait 15 minutes: recheck at 1115. If still under 70, repeat.

STEP 3 · PREVENT A SECOND LOW

Once above 70, lunch is more than an hour away, so give a snack with protein and complex carbohydrate.

IF HE COULD NOT SWALLOW

Nothing by mouth. IV D50 if there is access, glucagon IM, SC or intranasal if not.

WORKED EXAMPLE 4 · WEIGHT GAIN ON DESMOPRESSIN

A patient on desmopressin weighs 70.0 kg on Monday and 72.0 kg on Tuesday and reports a headache. What does the weight tell you?

STEP 1 · CONVERT

72.0 − 70.0 = 2.0 kg. One kilogram is one liter, so about **2 L of retained water**.

STEP 2 · CONNECT

Retained water dilutes sodium. Headache is an early sign of hyponatremia.

STEP 3 · ACT

Check serum sodium and neuro status, notify the provider and expect the dose to be held or reduced.

TRAPS PEOPLE FALL INTO

- **Stopping prednisone because the patient feels better.** The taper is for the gland, not the disease.
- **Skipping steroid doses on a sick day.** Illness is when the dose matters most; stress dosing may be needed.
- **Calling a sore throat on methimazole a cold.** Treat it as agranulocytosis until a CBC proves otherwise.
- **Giving levothyroxine with breakfast and calcium.** Empty stomach; calcium and iron at least 4 hours apart.
- **Shaking NPH.** Roll it gently. It is the only cloudy insulin.
- **Drawing cloudy before clear.** Clear (regular) first, so protamine never enters the regular vial.
- **Ruling out DKA on an SGLT2 inhibitor by a normal glucose.** Euglycemic DKA happens with near-normal glucose.

Chapter 8 self-test · ten items

1. Why can't insulin be swallowed as a pill?

A. It is fat soluble and stays in the gut wall
B. It works only after liver activation
C. It is too large to fit inside a capsule
D. It is a peptide that digestive enzymes destroy

D. It is a peptide that digestive enzymes destroy Peptide hormones are broken down in the gut. Lipid-soluble hormones such as thyroid hormone survive and can be swallowed.

2. A patient on desmopressin gains 2 kg overnight and reports headache. Which lab is most urgent?

A. Serum sodium
B. Serum TSH
C. Serum glucose
D. Serum calcium

A. Serum sodium Too much ADH effect retains water and dilutes sodium. Headache, confusion and seizure follow.

3. Which levothyroxine teaching is correct?

A. Take it with calcium to protect your bones
B. Take it 30 to 60 minutes before breakfast
C. Recheck TSH one week after any change
D. Stop it once your symptoms have resolved

B. Take it 30 to 60 minutes before breakfast Absorption is best on an empty stomach. Binders are separated by 4 hours, therapy is lifelong, and TSH is rechecked after 6 to 8 weeks.

4. A patient on methimazole calls with a sore throat and fever. What is the priority?

A. Start an antihistamine
B. Push fluids and rest at home
C. Hold the drug and get a CBC
D. Double the dose for two days

C. Hold the drug and get a CBC Thionamides can cause agranulocytosis. Sore throat and fever are the classic signs.

5. Which drug is preferred for hyperthyroidism in the first trimester?

A. Levothyroxine
B. Methimazole
C. Radioactive iodine
D. Propylthiouracil

D. Propylthiouracil Methimazole carries teratogenic risk early in pregnancy and radioactive iodine is contraindicated. PTU is used, with liver monitoring.

6. Which corticosteroid is chosen for cerebral edema?

A. Fludrocortisone
B. Hydrocortisone
C. Dexamethasone
D. Cortisone

C. Dexamethasone Dexamethasone is potent and nearly pure glucocorticoid, so it reduces swelling without retaining salt and water.

7. Which finding is usually normal in adrenal crisis after stopping chronic prednisone?

A. Serum potassium
B. Blood glucose
C. Serum sodium
D. Blood pressure

A. Serum potassium Steroid withdrawal suppresses ACTH but not aldosterone, so potassium is usually normal. Hyperkalemia points to primary adrenal failure.

8. Which insulin can be given IV?

A. NPH
B. Regular
C. Glargine
D. Detemir

B. Regular Regular insulin is the standard IV insulin for DKA drips and hyperkalemia. Long-acting and NPH insulins are not given IV.

9. Which oral agent is most likely to cause hypoglycemia on its own?

A. Pioglitazone
B. Glipizide
C. Metformin
D. Sitagliptin

B. Glipizide Sulfonylureas stimulate insulin release. Metformin, DPP-4 inhibitors and pioglitazone rarely cause lows alone.

10. A patient on acarbose plus insulin is hypoglycemic and alert. What should be given?

A. Table sugar
B. Orange juice
C. Regular soda
D. Glucose tablets

D. Glucose tablets Acarbose blocks breakdown of sucrose and other carbohydrates. Pure glucose is absorbed directly.

Cram sheet

Everything from chapters 7 and 8 in one place. Cover the right column and see if you can rebuild each row.

Autonomic drugs

Item	Fact	Note
Receptors	Beta-1 heart, beta-2 lungs, alpha-1 vessels, alpha-2 brake	Muscarinic = SLUDGE
Epinephrine anaphylaxis	0.3 to 0.5 mg IM of 1 mg/mL; child 0.01 mg/kg	Lateral thigh; repeat q5 to 15 min
Norepinephrine	First-line in septic shock	Extravasation: phentolamine
Albuterol	Rescue beta-2	Tremor, tachycardia, low K+
Clonidine	Alpha-2 agonist	No abrupt stop
Alpha blockers	-osin; BPH, BP	First dose at bedtime
Beta blockers	-olol; hold HR under 60	Mask hypoglycemia; glucagon for overdose
HFrEF beta blockers	Metoprolol succinate, bisoprolol, carvedilol	Start low, never in acute decompensation
Pyridostigmine	Myasthenia	30 to 60 min before meals
Organophosphates	Atropine + pralidoxime	Atropine alone misses weakness
Atropine (bradycardia)	1 mg rapid IV, q3 to 5 min, max 3 mg	Slow push can worsen it
Anticholinergic toxidrome	Dry, red, blind, mad, hot	No physostigmine with TCA or QRS over 100 ms
Succinylcholine	No reversal	Hyperkalemia, malignant hyperthermia
Non-depolarizers	Train-of-four	Neostigmine + glycopyrrolate or sugammadex

Endocrine drugs

Item	Fact	Note
Desmopressin	ADH replacement	Sodium, weight
Levothyroxine	Empty stomach; binders 4 h apart	TSH every 6 to 8 weeks
Thionamides	Methimazole first; PTU first trimester and storm	Agranulocytosis; PTU liver
Steroid equivalents	Hydrocortisone 20, prednisone 5, methylpred 4, dex 0.75 mg	Dex for cerebral edema
Steroid taper	After more than 2 to 3 weeks	Adrenal crisis: IV hydrocortisone
Rapid insulin	Peak 1 to 2 h	With the meal
Regular insulin	Peak 2 to 3 h	Only IV insulin
NPH	Peak 4 to 12 h; cloudy	Clear before cloudy
Glargine, detemir, degludec	No true peak	Never mix
Hypoglycemia	Under 70 mg/dL	Rule of 15; D50 or glucagon
Metformin	No lows alone	eGFR under 30 avoid; B12
Class alerts	SU lows, TZD heart failure, GLP-1 C-cell, SGLT2 euglycemic DKA	Acarbose: glucose only

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